THE IMMEDIATE EFFECT OF LOW BACK MANIPULATION ON SERUM CORTISOL LEVELS IN ADULT MALES WITH MECHANICAL LOW BACK PAIN

by

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A dissertation submitted to the Faculty of Health Sciences, in partial compliance with the requirements for a Master's Degree in Technology: Chiropractic at the Durban Institute of Technology.

I, Keseri Padayachy,
do hereby declare that this dissertation represents my own work in both conception and execution, except where specific assistance is sought and duly acknowledged

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DEDICATION

It is with immense pleasure that I dedicate this dissertation to:

The Supreme Lord of the Universe: thank you for your wisdom and guidance, especially through the difficult times.

My parents: thank you for your continued sacrifice, support and encouragement. You have given me the greatest gifts of a wonderful upbringing and an excellent education. I am eternally grateful.

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ABSTRACT

Objectives:
To determine if serum cortisol levels are increased following Spinal Manipulation Therapy (SMT) to the low back region and to determine the effect of a short rest interval on the cortisol levels.

Project Design:
The research project was in the form of a randomised, clinical trial using human subjects.

Setting:
Patients presenting with low back pain to the Chiropractic Day Clinic at the Durban Institute of Technology and the Community Health and Indigent Programme Services clinic.

Subjects:
Adult, male patients, aged between 18 and 35 years of age, diagnosed with mechanical low back pain.

Outcome measure:
Daytime, serum cortisol levels.

Results:
A decrease in serum cortisol levels following SMT. Serum cortisol levels decreased significantly following a short rest interval.

Conclusions:
The results of this study support the previous finding that a neuroendocrine effect can be stimulated by SMT, albeit, a decrease in serum cortisol levels. A short-term rest period also influenced the serum cortisol levels. However, the mechanism of these effects is not established and requires further investigation as this was not within the scope of the present study.
# TABLE OF CONTENTS

<table>
<thead>
<tr>
<th>CHAPTER</th>
<th>PAGE</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>CHAPTER 1: INTRODUCTION</strong></td>
<td></td>
</tr>
<tr>
<td>1.1 Introduction</td>
<td>1</td>
</tr>
<tr>
<td>1.2 Aim and Objectives</td>
<td>2</td>
</tr>
<tr>
<td>1.2.1 Aim</td>
<td>2</td>
</tr>
<tr>
<td>1.2.2 Objectives</td>
<td>2</td>
</tr>
<tr>
<td>1.3 Hypotheses</td>
<td>2</td>
</tr>
<tr>
<td><strong>CHAPTER 2: LITERATURE REVIEW</strong></td>
<td></td>
</tr>
<tr>
<td>2.1 Introduction</td>
<td>4</td>
</tr>
<tr>
<td>2.2 Mechanical Low Back Pain</td>
<td>4</td>
</tr>
<tr>
<td>2.3 Cortisol</td>
<td>6</td>
</tr>
<tr>
<td>2.3.1 Diurnal nature of serum cortisol secretion</td>
<td>6</td>
</tr>
<tr>
<td>2.3.2 Anti-inflammatory effects of cortisol</td>
<td>7</td>
</tr>
<tr>
<td>2.4 Spinal Manipulation Therapy (SMT)</td>
<td>8</td>
</tr>
<tr>
<td>2.5 Studies concerning salivary cortisol and SMT</td>
<td>9</td>
</tr>
<tr>
<td>2.6 Possible explanation of effects of SMT on cortisol</td>
<td>11</td>
</tr>
<tr>
<td>2.7 Summary</td>
<td>14</td>
</tr>
<tr>
<td><strong>CHAPTER 3: MATERIALS AND METHODS</strong></td>
<td></td>
</tr>
<tr>
<td>3.1 Introduction</td>
<td>16</td>
</tr>
<tr>
<td>3.2 Research design</td>
<td>16</td>
</tr>
<tr>
<td>3.3 Study Design protocol</td>
<td>16</td>
</tr>
<tr>
<td>3.3.1 Objectives</td>
<td>16</td>
</tr>
<tr>
<td>3.3.2 The Subject Demographics</td>
<td>16</td>
</tr>
<tr>
<td>3.3.2.1 Subject Recruitment</td>
<td>16</td>
</tr>
<tr>
<td>3.3.2.2 Sampling and Group Allocation</td>
<td>17</td>
</tr>
<tr>
<td>3.3.3 Inclusion and Exclusion Criteria</td>
<td>17</td>
</tr>
<tr>
<td>3.3.3.1 Inclusion Criteria</td>
<td>17</td>
</tr>
<tr>
<td>3.3.3.2 Exclusion Criteria</td>
<td>18</td>
</tr>
<tr>
<td>3.3.4 Patient procedure</td>
<td>18</td>
</tr>
</tbody>
</table>
3.3.5 Research procedure
3.4 Measurements and Observations
3.4.1 The Research Data
3.4.1.1 The Primary Data
3.4.1.2 The Secondary Data
3.5 Statistical Analysis
3.5.1 Methods of Data Analysis

CHAPTER 4: STATISTICAL ANALYSIS AND RESULTS
4.1 Results
4.1.1 Demographics
4.1.2 Intra-group Analysis of serum cortisol concentrations (nmol/l)
4.1.2.1 Group 1
4.1.2.2 Group 2
4.1.3 Inter-group analysis
4.2 Summary

CHAPTER 5: DISCUSSION
5.1 Introduction
5.2 Interpretation of Data
5.2.1 Demographical Data
5.2.2 Serum Cortisol Analysis
5.3 Proposed Explanations
5.4 Study Limitations

CHAPTER 6: CONCLUSIONS AND RECOMMENDATIONS
6.1 Conclusions
6.2 Recommendations

REFERENCES
LIST OF TABLES

<table>
<thead>
<tr>
<th>TABLE</th>
<th>Description</th>
<th>PAGE</th>
</tr>
</thead>
<tbody>
<tr>
<td>TABLE 1</td>
<td>Comparison of weight and height between participants from Groups A and B (n=30).</td>
<td>32</td>
</tr>
<tr>
<td>TABLE 2</td>
<td>Wilcoxon signed rank tests for paired intra-group comparison in Group A.</td>
<td>34</td>
</tr>
<tr>
<td>TABLE 3</td>
<td>Wilcoxon signed rank test for paired intra-group comparison in Group B between pre-treatment A and pre-treatment B.</td>
<td>34</td>
</tr>
<tr>
<td>TABLE 4</td>
<td>Wilcoxon signed ranks test for paired inter-group comparison in Group B between pre-treatment B and post-treatment C.</td>
<td>35</td>
</tr>
<tr>
<td>TABLE 5</td>
<td>Wilcoxon signed ranks test for paired intra-group comparison in Group B between pre-treatment A and post-treatment C.</td>
<td>35</td>
</tr>
<tr>
<td>TABLE 6</td>
<td>Mann-Whitney test for baseline (pre-treatment) cortisol level comparison between Groups A and B.</td>
<td>36</td>
</tr>
<tr>
<td>TABLE 7</td>
<td>Mann-Whitney test for post-treatment serum cortisol level comparison between Group A and B.</td>
<td>37</td>
</tr>
<tr>
<td>TABLE 8</td>
<td>Descriptives for change in serum cortisol levels between baseline (pre-treatment) and post-treatment reading by groups.</td>
<td>38</td>
</tr>
<tr>
<td>TABLE 9</td>
<td>Mann-Whitney test for comparison of change in serum cortisol by groups.</td>
<td>39</td>
</tr>
</tbody>
</table>
LIST OF FIGURES

FIGURE | PAGE
-------|-----
FIGURE 3.1 Subject being motion palpated. | 20
FIGURE 3.2 Subject resting supine. | 21
FIGURE 3.3 Instruments used for phlebotomy. | 21
FIGURE 3.4 Tourniquet placed about 4cm proximal to the wrist | 22
FIGURE 3.5 Dorsal surface of hand being cleansed. | 22
FIGURE 3.6 Butterfly catheter attached to sharp gauge. | 23
FIGURE 3.7 Butterfly catheter being inserted into a prominent vein. | 23
FIGURE 3.8 Plastic tube being pushed into sharp gauge. | 24
FIGURE 3.9 Cotton wool being placed over catheter before being removed from the vein. | 24
FIGURE 3.10 Subject's forearms being crossed over to opposite shoulders. | 25
FIGURE 3.11 Subject's upper leg being flexed at the hip and knee joints | 26
FIGURE 3.12 Foot of upper limb being placed in the region of the popliteal fossa of the lower limb. | 26
FIGURE 3.13 Doctor takes up the "Fencer" stance. | 27
FIGURE 3.14 Subject being manipulated. | 28
FIGURE 3.15 Subject was brought back to supine position. | 28
FIGURE 4.1 Median serum cortisol levels (nmol/l) at pre- and post- treatment in Group A. | 33
FIGURE 4.2 Median serum cortisol levels (nmol/l) at pre- and post-treatment in Group B. | 36
FIGURE 4.3 Distribution of pre-treatment serum cortisol levels (nmol/l) of Groups A and B. | 37
FIGURE 4.4 Distribution of post-treatment serum cortisol levels (nmol/l) of Groups A and B. | 38
### LIST OF FIGURES, CONTINUED

<table>
<thead>
<tr>
<th>FIGURE</th>
<th>PAGE</th>
</tr>
</thead>
<tbody>
<tr>
<td>FIGURE 4.5 Distribution of the change in serum cortisol levels (nmol/l) of Groups A and B.</td>
<td>39</td>
</tr>
<tr>
<td>FIGURE 4.6 Profile plot of median serum cortisol levels (nmol/l) over time by groups.</td>
<td>40</td>
</tr>
<tr>
<td>FIGURE 5.1 Schematic diagram illustrating cortisol levels, pre- and post-intervention.</td>
<td>42</td>
</tr>
</tbody>
</table>
# LIST OF APPENDICES

<table>
<thead>
<tr>
<th>Appendix</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Appendix 1</td>
<td>Patient Information Sheet (English)</td>
</tr>
<tr>
<td>Appendix 2</td>
<td>Patient Information Sheet (isiZulu)</td>
</tr>
<tr>
<td>Appendix 3</td>
<td>Informed Consent (English)</td>
</tr>
<tr>
<td>Appendix 4</td>
<td>Informed Consent (isiZulu)</td>
</tr>
<tr>
<td>Appendix 5</td>
<td>Case History</td>
</tr>
<tr>
<td>Appendix 6</td>
<td>Physical Examination</td>
</tr>
<tr>
<td>Appendix 7</td>
<td>Regional Examination: Lumbar Spine and Pelvis</td>
</tr>
<tr>
<td>Appendix 8</td>
<td>Numerical Pain Rating Scale (NRS)</td>
</tr>
<tr>
<td>Appendix 9</td>
<td>Advert for Research Patient Recruitment</td>
</tr>
<tr>
<td>Appendix 10</td>
<td>Research Ethics Committee</td>
</tr>
</tbody>
</table>
CHAPTER 1
INTRODUCTION

1.1. INTRODUCTION

Mechanical low back pain (LBP) is one of the most common clinical disorders that more people are seeking help for (Browning, 2003). The prevalence of LBP increases with age and the magnitude depends on the population surveyed (Docrat, 1999).

The options for management of LBP are vast, as there is no consensus amongst health care professionals as to the best possible form of intervention (Browning, 2003). With respect to spinal manipulation, literature reveals that it is a common form of intervention that is widely used in the treatment of LBP (Giles and Muller, 1999; Kirkaldy-Willis and Bernard, 1999). The outcome of most of the studies on LBP and spinal manipulative therapy (SMT) support the view that SMT results in a greater improvement in mechanical LBP.

Kirkaldy-Willis and Bernard (1999) suggest that mechanical LBP is possibly caused by a repetitive rotational and compressive motion to the lumbar facet joint. This will in turn cause trauma to the facet joint resulting in a synovitis of the facet joint. There are various chiropractic theories on the alleviation of mechanical LBP, such as Korr's theory, Sandoz's theory, somatovisceral theory, Dvorak's theory and the neuroendocrine physiological mechanism (Leach, 1994). One theory, which is under the ambit of neuroendocrine physiological mechanism that has been inadequately explained, is the stress effect of SMT on serum cortisol levels (Will, 1978). The researcher extrapolated this theory further, proposing that SMT causes an increase in serum cortisol levels by an unconfirmed yet hypothetically possible neuroendocrine mechanism and since cortisol is an anti-inflammatory hormone, the increased cortisol levels will have an anti-inflammatory effect on the inflamed lumbar facet and sacroiliac joints, thus reducing LBP.
One of the major hormones secreted by the adrenal gland is a glucocorticoid, called cortisol. Cortisol follows a circadian rhythm of activity. The rhythm consists of the highest cortisol levels shortly after awakening (9am-10am) and progressively falling until they are lowest during the first several hours of sleeping (Guyton and Hall, 2000). Acute physical and psychological stress activates the hypothalamic-pituitary-adrenal axis, resulting in increased plasma adrenocortico-trophic and cortisol levels (Wilson and Foster, 1992; Greenspan and Gardner, 2001).

There is little documented literature stating that SMT can, in fact, cause a stress effect on the body hence increasing cortisol levels and thus resulting in an anti-inflammatory effect. To date, there are only two studies that have attempted to determine, the effect of SMT on salivary cortisol levels. The findings of these studies are discussed in detail in Chapter 2, however, both these studies have methodological flaws in their design.

1.2. AIM AND OBJECTIVES

1.2.1 AIM
The aim of this study is to determine if serum cortisol increases immediately after SMT.

1.2.2. OBJECTIVES
- To determine if serum cortisol levels increase immediately post-SMT to the low back region
- To determine the effect of a short rest interval on serum cortisol levels

1.3. HYPOTHESES
- Serum cortisol levels should significantly increase after SMT.
- Short-term rest does not influence serum cortisol levels.
Data was analysed using the Wilcoxon signed rank test for intra group analysis and Mann-Whitney-U test for inter group analysis using the SPSS version 11.5 package.
CHAPTER 2
LITERATURE REVIEW

2.1. INTRODUCTION
LBP is one of the most common and costly medical conditions confronting health care providers and medical insurers today (Browning, 2003; Perina, 2005). Despite the magnitude of the problem, no general consensus exists concerning an appropriate treatment for this condition (Browning, 2003). The traditional allopathic methods of treating this condition undoubtedly have demonstrated inadequacies, therefore alternative treatment methods are often considered. Manipulative therapy has been shown to be an efficacious method of treating mechanical LBP (Pustaver, 1994; Hendler et al., 1995; Giles and Muller, 1999; Cooperstein et al., 2001). LBP has a lifetime prevalence of between 60% to 90% for any general population (Kirkaldy-Willis and Bernard, 1999; Hills, 2004).

2.2. MECHANICAL LOW BACK PAIN
The majority of LBP is mechanical i.e.: it exists without overt structural pathologies (Kirkaldy-Willis and Burton, 1992; Hills, 2004). Mechanical LBP is caused by:
1) Joint dysfunction i.e. joint mechanics showing area disturbances of function without structural change (Bergmann et al., 1993) and
2) “Soft tissue” syndromes (e.g. myofascial pain dysfunction syndromes) which involve nociceptive processes (pain sensitive processes) rather than nerve injury (Waddel, 1995).

Mechanical LBP conditions include lumbar facet syndrome, sacroiliac syndrome, Maignes' syndrome, disc herniation, facet and disc degeneration, central and lateral canal stenosis, myofascial pain and dysfunction syndromes (Kirkaldy-Willis and Burton, 1992; Hills, 2004).

For the purpose of this study, the focus was on acute lumbar facet and sacroiliac joint syndrome. Acute was defined as the subjects presenting
within 4 weeks of initial onset of LBP (Bigos et al., 2004). According to Kirkaldy-Willis and Burton (1992), acute lumbar facet syndrome is the result of rotation or compressive strain sometimes due to a major, but more often is due to a minor episode of spinal trauma. The mechanism they propose is that the trauma to the lumbar region results in lumbar facet joint sprain, i.e.: small capsular tears resulting in a minimal degree of joint subluxation. Joint subluxation is defined as an aberrant relationship between two adjacent articular structures that may have functional or pathological sequelae, causing an alteration in the biomechanical and or body systems that may be directly or indirectly affected (Bergmann et al., 1993). The various pathological changes that occur subsequent to this regional trauma often lead to synovitis. This inflammation can stimulate pain sensitive receptors and also affect blood supply to surrounding musculature, by intense protective spasm, thus increasing joint dysfunction and decreasing mobility (Kirkaldy-Willis and Bernard, 1999; Seaman and Cleveland, 1999).

An earlier study by Cavanaugh and Ozaktay (1997), laid the foundation for evidence of this proposed mechanism. In this study, adult male, white rabbits weighing 3 to 4 kg were anaesthetised, and neurophysiological studies were performed on the lumbar facet joints as well as the surrounding tissues.

A lumbar laminectomy was performed in the rabbits to expose the lumbar dorsal roots under study. The L3/L4 or L4/L5 facet joints were kept intact, and recordings were made from the appropriate dorsal roots on (L4 or L5). A ventral ramus rhizotomy was performed at the appropriate level so that nerve discharge from peripheral tissues would not be recorded. They showed that inflammatory mediators could produce ongoing background discharge in sensory nerves of joints and sensitise the nerves to mechanical stress. Thus, facet joint pain fibres that would normally fire only when mechanical stress is clearly noxious, can fire at much lower stresses and hence thresholds in the presence of these chemicals and maintain a background discharge even without mechanical stress. With respect to the current study, we extrapolate that noxious mechanical stress in humans
may lead to the production of inflammatory mediators which would sensitise the nerves of the facet joints. It is this peripheral nerve sensitisation that may be contributing to the ongoing episodes of LBP.

2.3. CORTISOL
Cortisol, biochemically known as 17 hydroxycorticosterone, is the primary endocrine secretory product of the adrenal gland, present in the peripheral blood of humans (DeGroot et al., 1989). A steroid hormone, it is secreted by the *zona glomerulosa* of the adrenal cortex and is regulated by the hypothalamic-pituitary axis (HPA) (Guyton and Hall, 2000).

2.3.1 DIURNAL NATURE OF CORTISOL SECRETION
Cortisol is not synthesised continuously during the day. It has a diurnal variation, which is characterised by the plasma levels being elevated in a cyclical manner throughout the day. This diurnal pattern results from a series of discontinuous bursts of secretory activity during the early part of the day, with virtual cessation of secretion for several hours just before and after midnight (Kacsoh, 2000; Briegel et al., 1994).

Cortisol secretion is increased by physical and emotional stress. Such stress-related stimuli override the baseline regulatory mechanism, mediated as a biofeedback in the hypothalamus. The resultant increments are superimposed on the usual diurnal pattern. Both the stress response and the baseline cortisol secretory pattern depend on adreno-cortico-trophic hormone (AcTH) released by the anterior pituitary under the influence of the hypothalamic corticotrophin releasing factor (CRF). It is documented that cortisol concentrations are highest at 8am to about 10am and steadily decreases throughout the rest of the day (Guyton and Hall, 2000). Cortisol should spike immediately after a stress is placed on the body and should thereafter decrease. The rate at which cortisol decreases will depend on body clearance i.e.: renal function and the amount of circulating cortisol in the body (Naidoo, 2004). Gray and James (1983), DeGroot et al. (1989), and Besser and Thorner (2002) also mention that cortisol is released in response to stress but are uncertain as to exactly how soon cortisol is
released following a stressor. Normal values for male serum cortisol range between 190nmol/l - 690nmol/l in the mornings and 55nmol/l - 250nmol/l in the evening (Naidoo, 2004). These laboratory values are based on King Edward VIII Hospital values. Currently, despite an exhaustive literature search (books, Internet, journals) there appears to be no studies determining serum cortisol variation amongst the different ethnic populations.

2.3.2. ANTI-INFLAMMATORY EFFECTS OF CORTISOL

Cortisol’s potent anti-inflammatory effects are thought to maintain endothelial integrity (Oppert et al., 2000). This anti-inflammatory effect is brought about by cortisol interfering with the actual synthesis of inflammatory components (Guyton and Hall, 2000).

Cortisol has two basic anti-inflammatory effects:-

1. Cortisol blocks the early stages of the inflammation process before inflammation even begins (Guyton and Hall, 2000).
2. If inflammation has already begun, cortisol causes rapid resolution of the inflammation and increases the rapidity of the healing process (Guyton and Hall, 2000).

These effects are explained further as follows: cortisol prevents the development of inflammation by the following effects:

- Cortisol stabilises the lysosomal membranes.
- Cortisol decreases the permeability of the capillaries.
- Cortisol decreases both migration of white blood cells into the inflamed area and phagocytosis of the damaged cells.
- Cortisol suppresses the immune system, causing lymphocyte proliferation to decrease markedly.
- Cortisol lowers fever mainly because it reduces the release of interleukin-1 from the white blood cells.

Thus cortisol has an almost global effect in reducing all aspects of the inflammatory process (Guyton and Hall, 2000).

---

1 Verbal communication Dr P. Naidoo, senior chemical pathologist (February 2004)
2.4. SPINAL MANIPULATION

SMT is a specific form of articular manipulation, using either long or short leverage techniques, with specific contacts. It is characterised by a dynamic thrust of controlled high velocity, amplitude and direction (Bergmann et al., 1993). SMT has been documented to be efficacious in the treatment of mechanical LBP (Shekelle et al., 1992; Opperman, 1997; Kruger, 1999; Whelan et al., 2002; Smith, 2003), as it results in improved flexibility, reduced pain and increased joint mobility (Gatterman, 1990).

There are many hypotheses on the effects of spinal manipulation on the body e.g. neural and endocrine effects, (Leach, 1994), but the one of interest to this study is Dvorak Inflammatory Model. Dvorak (1985) proposed that segmental dysfunction or local joint dysfunction creates both a mechanical and the chemical stimulation necessary for the activation of nociceptors and spinothalamic tract activity. His model proposed that segmental dysfunction would have reflex effects on the muscle thus increasing the muscle spindle discharge of alpha motor neuron fibres post-muscle contraction. If this effect occurs for prolonged periods, it will lead to local muscle spasm. This spasm will result in a sensory discharge, leading to the same muscle contracting i.e. shortening of postural slow-twitch muscle fibres, which can then either result in inflammatory histochemical changes or cause relative local hypoxaemia. These effects result in damage to the muscle causing muscular pain and muscular imbalance, which in turn results in disturbed joint movement. This theory could be considered as the foundation for Kirkaldy-Willis' and Burton's (1992) explanation of joint inflammation (synovitis) found in mechanical LBP syndrome.

Selye (1956) determined that the nervous system is also involved in the hypophyseal-adrenocortical response to stress by a number of pathways. Experiments with animals showed that a neurogenic stressor triggers a neural response through the hypothalamus to the anterior lobe of the pituitary where AcTH is secreted into the systemic circulation. Thus the
interaction between the neuroendocrine and central nervous system was demonstrated (Selye, 1956).

However, there appears to be little documented literature whether SMT per se has any anti-inflammatory or neuroendocrine effects (Christian et al., 1988).

2.5. STUDIES CONCERNING SALIVARY CORTISOL AND SMT

To date, there are two documented studies regarding salivary cortisol and SMT (Tuchin, 1998; Whelan et al., 2002). The study by Tuchin (1998) was conducted to assess if spinal manipulation has the potential to effect salivary cortisol levels. This may also have some implications in the prevention of stress-related medical conditions such as hypertension, coronary artery disease, migraines, peptic ulcers, rheumatoid arthritis, inflammatory bowel disease and other significant disease conditions that are known to have a correlation with stress. Both these studies utilised Selye's definition of stress as "the non-specific response of the body to any demand" (Selye, 1956).

Nine subjects, six male and three females participated in Tuchin's study. Subjects acted as their own controls and were informed they were receiving a therapeutic treatment to the area of spine that was affected. Saliva specimen was collected at 12 noon on Wednesdays and Saturdays. These days have been shown to be the most and least stressful periods of the week, respectively (Bassett, 1982). The procedure required a minimum of 2ml of saliva to be collected in a centrifuge tube and stored immediately, on ice. The study consisted of establishment of each individual's baseline cortisol level, a 2 week pre-experimental evaluation of the subject's cortisol levels, 2 week's of experimental evaluation in conjunction with pre-treatment and post-treatment evaluation of the salivary cortisol levels and 1 week of post-experimental re-evaluation of the subject's salivary cortisol levels. The subjects were not restricted from their usual daily activities during the course of this study.
The results of Tuchin's study showed statistically significant reduced levels \((p<0.001)\) of salivary cortisol over the complete five-week study period. There was no apparent change in the salivary cortisol levels immediately preceding and 15 minutes after the spinal manipulation. This study however, did not include control for gender bias on cortisol measurement, because both men and women were used as subjects. The relatively small sample size and the lack of a control group, make conclusions drawn from this study somewhat limited.

The objective of the study conducted by Whelan et al., (2002) was to determine if basal salivary cortisol levels can be properly detected and whether manipulation of the cervical spine had any direct effect on basal salivary cortisol levels in humans. Subjects were thirty, asymptomatic adult male, students attending a chiropractic college. The subjects where divided into three groups; namely, a control group, the “sham” group and the chiropractic cervical manipulation group. The control group received no manipulation or vertebral positioning, the sham group received spinal positioning without being taken to the end-range of motion and the spinal manipulation group received a high velocity low amplitude manipulation to the cervical spine. No reason has been given as to why a cervical spine manipulation was chosen for this study. Salivary samples were collected for 5 weeks. Subjects were requested to refrain from eating, exercising, using tobacco, and consuming any drinks other than water for 1 hour before the test sample collection. This was presumably done to prevent any of these activities from affecting the cortisol levels. Disposable, plain cotton salivettes were used for the quick and hygienic collection of the saliva sample. Chewing on the cotton-wool swab for 30 to 60 seconds collected saliva. During Week I, samples were collected by the students at home, upon waking and stored in their personal freezers until the final day of the testing. Home samples were transported to the laboratory on ice on the final day of testing. During Weeks 2 through 5, home samples were collected upon waking and were followed by an additional time course of samples collected in laboratory settings before and after manipulation. All laboratory testing
occurred between 8am and 10am, each test day, were collected on ice and stored at -80°C until biochemical analysis.

The results of the study showed that cervical spinal manipulation did not significantly change basal salivary cortisol levels. The time course of acute changes to cortisol levels was independent of the testing week and group. A decrease in salivary cortisol was detected over time on each trial-testing day. The results of the study suggest that the physical component of manipulation of the cervical spine is not a potent enough stressor to disrupt homeostatic mechanisms and override the hypothalamic-pituitary-adrenal axis.

However, in the study by Whelan et al., (2002), a difference in temperature between the personal freezers and laboratory freezers added another variable that has not been considered in the study. Furthermore, it is possible that the transport of the samples on ice to the laboratory could also have affected the results, depending on the quantity of ice used for the transportation process. Considering these shortcomings, the results of the study could be questioned.

2.6. POSSIBLE EXPLANATION OF EFFECTS OF SMT ON CORTISOL
To explain how SMT may affect cortisol release from the adrenal cortex, it is necessary to briefly introduce and discuss the mechano-sensitive units (also known as mechanoreceptors i.e. they detect mechanical deformation of the receptor or of the tissues adjacent to the receptor) found in the facet joints of the lumbar spine. An assumption is made that mechano-sensitive units found in the lumbar facets of rabbits represent those of humans (Yamashita et al., 1990).

Yamashita et al. (1990) conducted a study with the purpose of characterising mechano-sensitive units of the lumbar facet joint in rabbits, which may play a central role in LBP. In these rabbits, twenty-four units were identified (by means of laminectomy) in the region of the facet joint: ten, in the capsule of the joint; twelve, in the border regions between
capsule and muscle or tendon; and two in the ligamentum flavum. Of these units, two had a conduction velocity that was slower than 2.5 meters per second (mps) (Group IV), fifteen had a velocity ranging from 2.5 to twenty mps (Group III), and seven had a velocity faster than twenty mps. Fourteen other mechano-sensitive units were found in the muscle, tendon, and interspinous ligaments. Seven units in the facet joint responded to movement of the joint when stimulated electrically with a bipolar electrode. Grigg et al., (1986) reported that inflammation of the joint sensitised Group-III and Group-IV units and increased their responsiveness to movement under an inflammatory condition, thus increasing pain threshold. The clinical relevance of the finding of the study of Yamashita et al. (1990), is that the facet joint contains Group-III and Group-IV mechano-sensitive units with low to high thresholds and that several units responded to movement of the joint.

A study by Pickar and McLain (1995) tested the hypothesis that Group III and Group IV afferents with receptive endings in the lumbar spine respond to passive manipulation of the lumbar facet joint in cats. They noted that sensitive afferents were found in all tissues of the lumbar spine, including tissues of the facet joint, connective tissue immediately surrounding the facet joints, para-spinal muscle, and fascia distant from the facet joint. Distraction of the facet activates these sensory receptors. A hemilaminectomy approach was developed that permitted physiologic loading of the lumbar facet without disturbing its overlying musculature. Recordings of single unit afferent activity were made from filaments teased from the L5 dorsal root. This study showed that Group III and Group IV afferents located in tissues throughout the low back respond to forces applied through the lumbar facet.

These findings indicate the presence of a complex network of small diameter neural elements in the low back area capable of responding to movements of the lumbar spine. This network may play an important role in the normal function of the spinal column and may contribute to somatic and autonomic reflexes. In addition, stimulation or modulation of this system
may explain the beneficial effects many patients receive through physical therapy, bracing as well as spinal manipulation. Neuroanatomically, the lumbar facet joint receives sensory and postganglionic sympathetic fibres, which ipsilaterally and segmentally innervated by the sensory nervous system. Suseki et al., (1997) support the hypothesis that sensory pathways from the L4-L5 facet joint to L1 or L2 dorsal root ganglia pass through the sympathetic trunk. The lumbar segment of the sympathetic trunk may therefore transmit afferent impulses monitoring LBP caused by lumbar facet lesions.

Once action potentials in the dorsal column-medial lemniscal system (Suseki et al., 1997) are initiated by administered SMT, it is possible that they travel up the dorsal column-medial lemniscal system to the thalamus from which inter-neurons stimulate the hypothalamus to secret corticotrohin-releasing hormone (CRH). This causes the anterior pituitary to secrete adreno-corticotrophic hormone (AcTH), which causes the adrenal cortex to release cortisol (Guyton and Hall, 2000). Cortisol then functions as an anti-inflammatory agent (Guyton and Hall, 2000) and thus prevents further injury to joint complex, enhancing the recovery phase.
The proposed mechanism for cortisol release is summarised in the flow diagram:

- Patient with acute low back facet syndrome
- Spinal Manipulation Therapy applied
- Stimulation of articular apophyseal mechanoreceptors and muscle spindles in paraspinal muscles
- Neurons in network stimulated
- Action potentials travel up the dorsal column-medial lemniscal system to thalamus where interneurons stimulate the hypothalamus.
- Hypothalamus secretes corticotrophin-releasing hormone (CRH)
- CRH causes the anterior pituitary to secrete adenocorticotropic hormone (AcTH)
- AcTH causes the adrenal gland to increase cortisol levels
- Effect on inflamed tissue: Decreases inflammation
- Preventing further injury to joint complex and enhancing recovery phase.

2.7. SUMMARY
Mechanical LBP is a common condition; however there exists a disagreement amongst researchers on the aetiology and treatment protocols. Most authors agree that there is some degree of inflammation that occurs in the muscles and joints of the affected areas. SMT utilised for
treating mechanical LBP has been documented to be of therapeutic value. How exactly SMT eliminates or reduces LBP is not known. A possible explanation may lie in the neuroendocrine theory. This theory supports the idea that SMT may trigger neural and endocrine effects. One hypothesis proposed in this study states that lumbar SMT may lead to a release of cortisol, a known anti-inflammatory agent, and this may lead to a decrease in inflammation of the affected area, hence a decrease in the symptom of pain.
CHAPTER 3
MATERIALS AND METHODS

3.1 INTRODUCTION
This chapter includes a detailed description of the study design, patient inclusion criteria to participate in this study and the interventions used. The measurements obtained and the statistical procedures used in the analysis of the data are also discussed.

3.2 RESEARCH DESIGN
The research design was in the form of a randomised clinical trial where the effect of SMT was tested for its proposed anti-inflammatory effect by means of an immediate increase in serum cortisol level in subjects with mechanical LBP, particularly those with acute lumbar facet syndrome or sacroiliac syndrome, following SMT.

3.3 STUDY DESIGN PROTOCOL
3.3.1 OBJECTIVES
The aim of the study was to determine if serum cortisol increases immediately after lower back manipulative therapy. For the purpose of this study immediately was defined as occurring within five minutes of the first blood specimen drawn and assayed for serum cortisol level.

It was hypothesised that if SMT increases cortisol levels, then it is possible to initiate an anti-inflammatory cascade which may result in a decrease in joint inflammation thereby leading to relief of LBP.

3.3.2 THE SUBJECT DEMOGRAPHICS

3.3.2.1 SUBJECT RECRUITMENT
Subjects had to be residents of the greater Durban functional region and were selected from those people who responded to advertisements
(Appendix 9) placed in public places (e.g. gymnasia), pamphlet distribution and newspaper advertisements. There were no restrictions on ethnicity, cultural or socioeconomic background. The subjects had to present with acute mechanical lumbar facet or sacroiliac syndrome. For the purpose of this study, acute was defined as the subject presenting within 4 weeks of initial onset of LBP (Bigos et al., 1994).

3.3.2.2 SAMPLING AND GROUP ALLOCATION

Convenience sampling was utilized in this study. Subjects who responded to the advertisements were selected and randomly allocated into two groups of fifteen by drawing the group code, (1 or 2). This code was written on a piece of paper, folded and placed in a box which was shaken to mix the pieces of paper. Subjects in Group 1 were phlebotomised, received a low back spinal manipulation and then immediately (within 5 minutes) re-phlebotomised. Subjects in Group 2 were phlebotomised, rested for 5 minutes, re-phlebotomised, then received a low back spinal manipulation and immediately thereafter re-phlebotomised. In Group 2, the subjects were phlebotomised thrice, through the butterfly venous catheter, to obviate repeated punctures.

3.3.3 INCLUSION AND EXCLUSION CRITERIA

3.3.3.1 INCLUSION CRITERIA

1) The study was limited to male subjects who were between the ages of 18 and 35. Males younger than 18 years of age would need parental consent and according to DeGroot et al., (1989) older males are more likely to have unstable cortisol levels which could have affected the final results.

2) Subjects diagnosed with acute facet or sacroiliac syndrome of the low back as described by Kirkaldy-Willis and Bernard (1999)

3) Subjects with a Numerical Pain Rating Scale (NRS) of 5 to 10. This was used to homogenise the sample size, based on similar pain ratings.
3.3.3.2 EXCLUSION CRITERIA

1) Subjects with LBP of non-mechanical origin or other mechanical conditions e.g. disc herniation and other serious pathological conditions (excluded by means of case history and physical examination findings).

2) Subjects apprehensive of needles.

3) Subjects with contact allergies to disinfectant used in the research.

4) Hypertensive subjects i.e.: patients with a blood pressure of 140/90 mmHg or greater (Longmore et al., 2001)

5) Subjects with any haematological disorders e.g. haemophilia and anaemia.

6) Subjects with any cortisol abnormality e.g.: Cushing’s syndrome / Addison’s disease.

7) Subjects with contra-indications to SMT including: joint ankylosis, joint hypermobility, infection in the area that will be receiving treatment, malignancy in the area that will be receiving treatment, fractures, inflammatory arthritis, metabolic bone disease (Bergmann et al., 1993; Kirkaldy-Willis and Bernard, 1999)

8) Female subjects due to their menstrual cycles which affect cortisol levels (DeGroot et al., 1989).

9) Subjects who were medicated or received any other forms of treatment for LBP between the first and second consultation.

10) Subjects who received any form of treatment for a condition that may have arisen between the first and second consultation e.g. paracetamol for headaches.

11) Subjects who partook in any kind of exercise between the first and second consultation.

12) Subjects who arrived 10 minutes after the stipulated time of 07:30 hours.

3.3.4 PATIENT PROCEDURE

At the initial consultation, once the subjects were randomly assigned to their groups, subjects were given an information sheet (Appendix 1 or 2) outlining the research procedure, which was personally explained to them by the
researcher. Each subject signed an informed consent form (Appendix 3 or 4) allowing the researcher to begin the research with the subjects understanding that they were able to withdraw from the study at any stage, with no constraints or repercussions. Each subject was informed not to take any pain-relief medication or perform any strenuous physical activity, including exercise, between Days 1 and 2 of the study. Each patient underwent a complete case history (Appendix 5), physical examination (Appendix 6) and lumbar regional examination (Appendix 7). Subjects were also required to fill in a NRS (Appendix 8). Subjects with an NRS score of less than 5 were excluded from this study. This was done on Day 1 of the study.

3.3.5 RESEARCH PROCEDURE

Day 1- Case history, physical and regional examinations were done for both Groups 1 and 2 at the Durban Institute of Technology's Chiropractic Day Clinic. During this consultation subjects were also required to complete an NRS form. Subjects received explanations that the substance (cortisol) being tested has circadian rhythm (changes at different times of the day) therefore it was imperative that they arrived at the times stipulated by the researcher on Day 2. The times at which the subjects were phlebotomised were crucial to the final results of the study due the circadian nature of cortisol (Guyton and Hall, 2000).

Day 2- Subjects were required to arrive before 7:30am to the Community Health and Indigent Programme Services clinic. On arrival, subjects were questioned on the use of any medication for their LBP or participation in any exercise on Day 1 of the study. Subjects who had taken medication or any other form of treatment, or engaged in strenuous exercise were excluded from the study. The subjects of both groups had their weights and heights recorded by the researcher and were motion palpated.
Motion palpation is described as that aspect of palpation, which assesses the physiological range of motion possible in the different axes of motion, both generally and specifically for the joints of the spine (that is, a dynamic evaluation of the spine). This evaluation determines if a joint or motion unit of the lumbar spine/sacroiliac area has natural movement or if this movement is relatively increased or decreased (Ames, 1991). The evaluation continues until a joint fixation is found. Joint fixation is described by Haldeman (1992) as the state whereby the joint has become temporarily immobilized in a position that it may normally occupy during any phase of a physiological range of movement. This means that an affected motion unit of the spine may become hypomobile.

The subject was then rested supine, on a chiropractic examination bed for 10 minutes at normal room lighting, without the interference of sunlight due to the fact that sunlight has an effect on the biochemical pathway of cortisol (DeGroot et al., 1989).
After the 10 minutes had elapsed, blood pressure was measured on the subject's dominant arm. Blood pressure was determined on the dominant forearm side due to blood pressure being higher on this side of the patient (Vawda, 1995). Subjects were then rested for a further 5 minutes and blood pressure was re-measured on the selected side. Blood pressure was measured to prevent undiagnosed hypertensive or hypotensive subjects from participating in the research. Co-supervisor, Professor G.H.M. Vawda, phlebotomised the subjects, as described below.
Subject’s dominant hand was cleansed on the dorsal aspect for catheterising a suitable superficial vein.

Figure 3.4: Tourniquet placed about 4cm proximal to the wrist

The dominant upper limb was slightly flexed and supported by the chiropractic couch. A tourniquet was placed and appropriately tightened at about 4 cm proximal to the wrist joint.

Figure 3.5: Dorsal surface of hand being cleansed

An appropriate sized, but easily accessible, vein was punctured with a disposable sterile 21 gauge Vacutainer systems butterfly catheter.
In the absence of this catheter (21 gauge Vacutainer systems butterfly catheter), a 21 gauge Venisystems butterfly catheter was used. The butterfly catheter was removed from a needle guard after being angled at about 30°. The plastic end of the needle was attached to a clear plastic holder called the sharp gauge that had a plastic tube clipped on its free end.
Once the collection tube was pushed into the sharp gauge it filled with blood from the patient due to the vacuum the tube contains. This tube did not contain any anticoagulant substances.

Once 8 -10 ml of blood had been drawn, the tube was removed. The tube was labeled as either Group 1 or 2, Pre-Treatment X (X was the number allocated to the subject in the group of 15, selected subjects).
The subjects of Group 1 were first manipulated in the low back region according to the technique described by (Bergmann et al., 1993). The subjects were then re-phlebotomised; this sample being labeled Post-Treatment X.

A typical low back manipulation may be described as follows: the subject lies in the lateral position with the headpiece elevated, for comfort. The subject’s lower arm is tractioned laterally, folded over the shoulder of the opposite arm and stabilised with the indifferent hand, while cephalad traction is provided.

Figure: 3.10 Subject’s forearms being crossed over to opposite shoulders
The subject's leg is bent at the knee while the thigh is flexed at the hip, with the foot placed into the popliteal fossa of the opposite leg.

Figure 3.11: Subject's upper leg being flexed at the hip and knee joints

Figure 3.12: Foot of upper limb being placed in the region of the popliteal fossa of the lower limb
The doctor takes up the “Fencer” stance and places the subject’s upper bent knee between manipulator's thighs.

The doctor’s pelvis should be at the level of the lesion. The subject’s thigh is flexed while the doctor monitors the interspinous movement of the segments cranial and caudad to the lesion. The pelvis and thighs are stabilised at the point of the start of any movement of the involved spinous process by downward transfer of the doctor’s weight. The doctor’s forward leg carries the majority of the manipulator’s body weight. Skin slack is removed by cephalad traction of the indifferent hand, while a pisiform contact is made with the caudad hand on the mamillary process of the superior segment. The fingers should be spread, facing cephalad and with the fifth digit parallel to the spinal column. The cephalad hand is placed on the subject's upper shoulder, used to stabilize the torso and prevent excessive torque. The thrust is a body drop with a sudden impulse and small amplitude. (Szaraz, 1990)
The subjects of Group 2 rested supine for 5 minutes, then phlebotomised for the second time. They then received a low back spinal manipulation (Szaraz, 1990) and were phlebotomised for the third time. This third sample (labeled Post Treatment 2X1) was done as soon as it was possible after the low back manipulation. A 5-minute cut off time was used to standardise the procedure as well as to consider the immediate action of cortisol to increase after a “stressor” (Naidoo, 2004).
The tourniquet was then released, the tube removed, cotton wool placed on the site of the punctured skin, pressure applied, the needle removed and disposed in a "sharps" container, to prevent accidental needle prick injury. A strip of clear adhesive tape was placed on the cotton wool and the subject was asked to apply pressure to the area for three minutes, so as to prevent extravasation. This procedure was applied to both groups of subjects.

The researcher filled in blood test requisition form and the sample tubes were labeled appropriately. The sample request form contained the code of the research and subject's name. Identical labeling was used for the test tubes. The researcher personally took the specimens and form to the Department of Chemical Pathology laboratory on the 1st floor of the Nelson R. Mandela School of Medicine, within 5 minutes of collection. The specimens were handed to Dr. P. Naidoo [senior chemical pathologist, Nelson R. Mandela School of Medicine] (or in her absence the personnel of the laboratory on duty.) The blood samples were centrifuged and separated before freezing. Storage of the cortisol samples had no adverse effects on the final results of the study and in fact, prevented batching errors (Robertson, 2004).

The following is a flow diagram summarizing the research procedure:

```
Group 1
15 subjects

Day 1
Examination

Day 2
Subject phlebotomised at approximately 7:50am, then manipulated and then re-phlebotomised.

Group 2
15 subjects

Day 1
Examination

Day 2
Blood drawn at approximately 7:50am, subject rests supine for a few minutes and then phlebotomised. Subject then rests for 5 minutes, is then manipulated and re-phlebotomised within 5 minutes of the manipulation.
```
3.4 MEASUREMENTS AND OBSERVATIONS

3.4.1 THE DATA
The data was in two forms, primary data and secondary data.

3.4.1.1 THE PRIMARY DATA
The primary data was obtained from the following:
- Height and weight of the subjects
- Serum cortisol concentrations at the different times of the research process

3.4.1.2 THE SECONDARY DATA:
The secondary data was collected from a variety of different sources as all the available literature was screened and the relevant data selected for this particular study. These sources included journal articles, textbooks and the Internet.

3.5 STATISTICAL ANALYSIS
The statistical package SPSS (as supplied by SPSS Incorporated, Marketing Department- 1999, Chicago, USA) was used to input data and for analysis of the data in this study.

3.5.1 METHODS OF DATA ANALYSIS
Intra-group analyses: the data collected on the outcome measure of cortisol levels was checked for normality of distribution using histograms and skewness statistics. For normally distributed data, paired t-tests were used for comparisons of pre- and post- (Blood 1 and Blood 2) cortisol measurements. For data that is not normally distributed, Wilcoxon signed ranks tests were used to compare pre-measurements and post-measurements. Box and whisker plots were used to graphically show distributions of data pre-manipulation and post-manipulation.

Inter-group analyses: Baseline comparisons were made between the two groups using the Mann-Whitney test. For cortisol levels that were normally
distributed, repeated measures ANOVA were used to compare the two groups over time and examined for a time by group interaction which would be indicative of a treatment effect. For cortisol levels that are not normally distributed, the difference between the Blood 1 and Blood 2 measurements and Blood 1 and Blood 3 measurements were calculated and compared between the two groups using a Mann-Whitney-U test.

Non-parametric descriptive methods and statistical tests were used due to the skewness of the data and the small sample size. Intra-group comparisons were achieved by Wilcoxon signed ranks tests for two paired groups, while inter-group comparisons were done with Mann-Whitney-U tests for two independent groups (Kirkwood and Stern, 2003). Demographic variables were compared between groups using independent samples t-tests because these variables did not show significant skewness. An alpha level of 0.05 was used to classify statistical significance.

Hypotheses:
Two sets of hypotheses were tested viz.:

1) Serum cortisol levels would be increased post-low back SMT (The Null Hypothesis (Ho)). The Alternate Hypothesis (Ha) states that serum cortisol levels would decrease or be unaffected post-low back SMT.

2) Short-term rest will have no affect on serum cortisol levels (The Null Hypothesis (Ho)). The Alternate Hypothesis (Ha) states that short-term rest will in fact increase or decrease serum cortisol levels.
CHAPTER 4
STATISTICAL ANALYSIS AND RESULTS

4.1. RESULTS

4.1.1. Demographics
Thirty male subjects between the ages of 18 and 35 years were selected by convenience sampling to participate in the study. They were randomized into two groups, Group 1 (n=15), and Group 2 (n=15). Table 1 shows that, as expected (since randomisation distributes all baseline values evenly between groups, therefore, there were no expected baseline differences) there was no significant difference in weight (p = 0.831) or height (p = 0.481) between the subjects of the two groups.

Table 1: Comparison of weight and height between participants from Groups 1 and 2 (n =30)

<table>
<thead>
<tr>
<th></th>
<th>Group</th>
<th>n</th>
<th>Mean</th>
<th>Std. Deviation</th>
<th>Std. Error Mean</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>WEIGHT (kg)</td>
<td>1</td>
<td>15</td>
<td>74.5</td>
<td>15.1</td>
<td>3.9</td>
<td>0.831</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>15</td>
<td>73.5</td>
<td>10.0</td>
<td>2.6</td>
<td></td>
</tr>
<tr>
<td>HEIGHT (m)</td>
<td>1</td>
<td>15</td>
<td>1.8</td>
<td>.08</td>
<td>.02</td>
<td>0.481</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>15</td>
<td>1.7</td>
<td>.07</td>
<td>.02</td>
<td></td>
</tr>
</tbody>
</table>
4.1.2 INTRA-GROUP ANALYSIS OF SERUM CORTISOL CONCENTRATIONS (nmol/l)

4.1.2.1. Group 1

Group 1 was only measured pre-spinal manipulation and post-spinal manipulation. There were three missing values for these time points in Group 1. The missing values are due to haemolysis of serum samples prior to analysis. Results of the Wilcoxon signed ranks tests for comparison between the median cortisol levels at these two time points are shown in Figure 4.1 and Table 2. There was a non-significant decrease in the cortisol levels ($p = 0.126$). Table 2 shows that in 9 of the 12 participants the serum cortisol values decreased between pre-treatment and post-treatment, and in 3 of the participants in this group the values increased from pre-treatment to post-treatment. There were 0 participants whose values were tied at the two time points.

![Figure 4.1: Median serum cortisol levels (nmol/l) at pre- and post-treatment in Group 1](image)
### Table 2: Wilcoxon signed ranks test for paired intra-group comparison in Group 1

<table>
<thead>
<tr>
<th>Post-Treatment B</th>
<th>Negative Ranks</th>
<th>n</th>
<th>Mean Rank</th>
<th>Sum of Ranks</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pre - Treatment A</td>
<td>9(a)</td>
<td>6.50</td>
<td>58.50</td>
<td></td>
<td>0.126</td>
</tr>
<tr>
<td>Positive Ranks</td>
<td>3(b)</td>
<td>6.50</td>
<td>19.50</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ties</td>
<td>0(c)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>12</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

- a Post-treatment B < Pre-Treatment A
- b Post-treatment B > Pre-Treatment A
- c Post-treatment B = Pre-Treatment A

### 4.1.2.2. Group 2

Group 2 was measured pre-treatment A, pre-treatment B and post-treatment C. There was a significant decrease in cortisol levels from pre-treatment A to pre-treatment B ($p = 0.018$). This is shown in Table 3. There was one missing value due to haemolysis of serum samples prior to analysis. Of the 14 participants 12 showed a decrease between pre-treatment A and pre-treatment B, while 2 showed an increase.

### Table 3: Wilcoxon signed ranks test for paired intra-group comparison in Group 2 between pre-treatment A and pre-treatment B

<table>
<thead>
<tr>
<th>Pre-treatment B</th>
<th>Negative Ranks</th>
<th>n</th>
<th>Mean Rank</th>
<th>Sum of Ranks</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pre-treatment A</td>
<td>12(a)</td>
<td>7.50</td>
<td>90.00</td>
<td></td>
<td>0.018</td>
</tr>
<tr>
<td>Positive Ranks</td>
<td>2(b)</td>
<td>7.50</td>
<td>15.00</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ties</td>
<td>0(c)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>14</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

- a Pre-treatment B < Pre-treatment A
- b Pre-treatment B > Pre-treatment A
- c Pre-treatment B = Pre-treatment A

There was a borderline decrease in median cortisol levels from pre-treatment B to post-treatment C ($p = 0.064$) in Group 2, as shown in Table...
4. There were two missing values due to haemolysis of serum samples prior to analysis. Eleven subjects out of 13 showed a decrease between these time points, while only two showed an increase.

Table 4: Wilcoxon signed ranks test for paired intra-group comparison in Group 2 between pre-treatment B and post-treatment C

<table>
<thead>
<tr>
<th></th>
<th>n</th>
<th>Mean Rank</th>
<th>Sum of Ranks</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Post-Treatment C</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Negative Ranks</td>
<td>11(a)</td>
<td>6.55</td>
<td>72.00</td>
<td>0.064</td>
</tr>
<tr>
<td>Positive Ranks</td>
<td>2(b)</td>
<td>9.50</td>
<td>19.00</td>
<td></td>
</tr>
<tr>
<td>Ties</td>
<td>0(c)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>13</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

a Post-treatment C < Post-treatment B  
b Post-treatment C > Post-treatment B  
c Post-treatment C = Post-treatment B

There was also a significant decrease overall in Group 2 between pre-treatment A and post-treatment C ($p = 0.019$) (Table 5). The overall decrease is shown in Figure 4.2. There was one missing value due haemolysis of serum samples prior to analysis. Twelve of 14 participants showed a decrease while only 2 showed an increase.

Table 5: Wilcoxon signed ranks test for paired intra-group comparison in Group 2 between pre-treatment A and post-treatment C

<table>
<thead>
<tr>
<th></th>
<th>n</th>
<th>Mean Rank</th>
<th>Sum of Ranks</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Post-Treatment C</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Negative Ranks</td>
<td>12(a)</td>
<td>7.50</td>
<td>90.00</td>
<td>0.019</td>
</tr>
<tr>
<td>Positive Ranks</td>
<td>2(b)</td>
<td>7.50</td>
<td>15.00</td>
<td></td>
</tr>
<tr>
<td>Ties</td>
<td>0(c)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>14</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

a Post-treatment C < Pre-treatment A  
b Post-treatment C > Pre-treatment A  
c Post-treatment C = Pre-treatment A
4.1.3. INTER-GROUP ANALYSIS

There was no significant difference between the median baseline cortisol levels of the two groups \( (p = 0.400) \). This is shown in Table 6 and Figure 4.3. There was one missing value at baseline in Group 1 due to haemolysis of the serum sample. Median serum cortisol for Group 1 at baseline was 289.5 nmol/l (range 214 to 656 nmol/l). The median for Group 2 was 387 nmol/l (range 119 to 591 nmol/l).

Table 6: Mann-Whitney test for baseline (pre-treatment) serum cortisol level comparison between Group 1 and 2

<table>
<thead>
<tr>
<th>GROUP</th>
<th>n</th>
<th>Mean Rank</th>
<th>Sum of Ranks</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pre-treatment A</td>
<td>1</td>
<td>14</td>
<td>13.61</td>
<td>190.50</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>15</td>
<td>16.30</td>
<td>244.50</td>
</tr>
<tr>
<td>Total</td>
<td>29</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Figure 4.3: Distribution of pre-treatment serum cortisol levels (nmol/l) of Group 1 and 2

There was no significant difference between the post-treatment cortisol levels of the two groups ($p = 0.981$). This is shown in Table 7 and Figure 4.4.

Table 7: Mann-Whitney test for post-treatment serum cortisol level comparison between Group 1 and 2

<table>
<thead>
<tr>
<th>GROUP</th>
<th>n</th>
<th>Mean Rank</th>
<th>Sum of Ranks</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Post-treatment</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>serum cortisol levels</td>
<td>1</td>
<td>13</td>
<td>14.08</td>
<td>183.00</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>14</td>
<td>13.93</td>
<td>195.00</td>
</tr>
<tr>
<td>Total</td>
<td>27</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Figure 4.4: Distribution of post-treatment serum cortisol levels (nmol/l) of Groups 1 and 2

The change in cortisol from baseline to final reading was calculated for each participant from both groups. Median change was compared between the groups. Table 8 shows that the median change in Group 1 was -5nmol/l and in Group 2 it was -33nmol/l. Thus Group 2 showed a larger decrease in cortisol between baseline and final measurement than Group 1. This was statistically significant, as shown in Table 9. Figure 4.5 shows the distributions of the change in cortisol, by group.

Table 8: Descriptives for change in serum cortisol levels between baseline (pre-treatment) and post-treatment reading by Groups

<table>
<thead>
<tr>
<th>GROUP</th>
<th>Median</th>
<th>Minimum</th>
<th>Maximum</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>-5.0000</td>
<td>-31.00</td>
<td>24.00</td>
</tr>
<tr>
<td>2</td>
<td>-33.0000</td>
<td>-83.00</td>
<td>167.00</td>
</tr>
<tr>
<td>Total</td>
<td>-20.0000</td>
<td>-83.00</td>
<td>167.00</td>
</tr>
</tbody>
</table>
Table 9: Mann-Whitney test for comparison of change in serum cortisol by Groups

<table>
<thead>
<tr>
<th>GROUP</th>
<th>n</th>
<th>Mean Rank</th>
<th>Sum of Ranks</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>CHANGE IN</td>
<td>1</td>
<td>12</td>
<td>17.88</td>
<td>214.50</td>
</tr>
<tr>
<td>SERUM</td>
<td>2</td>
<td>14</td>
<td>9.750</td>
<td>136.50</td>
</tr>
<tr>
<td>CORTISOL LEVEL</td>
<td>Total</td>
<td>26</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Figure 4.5: Distribution of the change in serum cortisol levels (nmol/l) of Groups 1 and 2

4.3. SUMMARY

Figure 4.6 shows the profile plot of both groups' median serum cortisol values over time. Group 2 showed the largest decrease between pre-treatment A and pre-treatment B. There was only a minor change after SMT. Thus it appears that resting between measurements had a greater impact on serum cortisol levels than the manipulation had.
Figure 4.6: Profile plot of median serum cortisol levels (nmol/l) over time by Groups
CHAPTER 5
DISCUSSION

5.1. INTRODUCTION
This chapter discusses the results obtained through the statistical analyses of the objective data.

The sample size of the present study consisted of thirty male subjects who presented with mechanical LBP, attributable purely to acute lumbar facet syndrome or acute sacroiliac syndrome. These patients had no other diagnosed pathological problems of the lumbarsacral spine.

The hypothesis proposes that low back SMT may lead to a release of endogenous cortisol which effects a decrease in inflammation in the affected area. It was also hypothesised that a short-term rest period would not affect the release of cortisol.

5.2. Interpretation of Data

5.2.1. Demographical data
The demographical data pertaining to this study sample was presented in Chapter Four under 4.2.1. The study by Tuchin (1998) used a sample size of 9 subjects (3 females and 6 males). The study by Whelan et al. (2002) used a sample size of 30 males. Both these studies failed to report the use of any other demographic data.

The demographical data used in this present study were height and weight. The height and weight of Group 1 was compared to that of Group 2. There was no significant difference between the height and weight of both groups. The average Body Mass Index (BMI) of Group 1 was 24.3 and that of Group 2 was 24.5. These values fall in the upper part of the acceptable range (Haslett et al., 1999). It is thus possible that these subjects were symptomatic with LBP as a result of being slightly overweight which is in
keeping with several studies that concluded that an increase in weight increases the incidence of LBP (Orvieto et al., 1994; Van der Meulen, 1997). There is no indication in the literature that height has an effect on serum cortisol levels but there have been documented findings that overweight people generally have higher serum cortisol levels (DeGroot et al., 1989).

5.2.2 Serum cortisol analyses
The thirty male subjects were randomly allocated to one of two groups i.e. Group 1 (n=15) or Group 1 (n=15). The subjects in Group 1 received pre-treatment A and post-treatment B phlebotomies. The subjects of Group 2 received pre-treatment A, pre-treatment B and post-treatment C phlebotomies, via the single phlebotomy catheter, cannulated prior to commencement of any procedures.

![Figure 5.1: Schematic Diagram illustrating cortisol levels pre-and post-intervention](image)

The common finding, in both intra-group and inter-group comparisons, was a decrease in serum cortisol levels. This decrease in serum cortisol levels may possibly demonstrate the natural circadian drop in basal cortisol levels. This is not in keeping with our proposed hypothesis that low back spinal manipulation would cause a stress on the body significant enough to
increase cortisol levels. It was also found that short-term rest caused a significant decrease in serum cortisol levels. It is worth noting that any activity resulting in a stress response on the body should increase serum cortisol levels (Guyton and Hall, 2000). However, short-term rest is actually a period of inactivity and as such, there is no stressor on the body and this could possibly explain the decrease in serum cortisol levels after short-term rest.

Baseline serum cortisol levels differ in individuals depending on a few factors which are mentioned below:

- **Sexual dimorphism**: females have more unstable serum cortisol levels due to their menstrual cycle having an influence on cortisol levels hence females were excluded from this study (DeGroot et al., 1989)

- **Age ranges**: research has shown that the older the individual the higher the baseline serum cortisol levels hence one of the reasons this study was limited to males between the ages of 18 to 35 (Beale et al., 2002).

- **Occupational variations**: cortisol is considered a stress hormone and is released when an individual is exposed to a stressor. Occupational factors do result in individuals experiencing large amounts of stress (Selye, 1956) and this could result in them having higher baseline cortisol levels.

- **Diurnal variations**: are often disturbed in individuals who work alternating day and night shifts. The diurnal cycle is based on 24 hours but if the day is lengthened to more than 24 hours the cycle also lengthens thus affecting baseline serum cortisol levels (Ganong, 2001).

The researcher did not investigate the occupational and diurnal variations on serum cortisol levels in this study, and therefore cannot comment on how these factors could have influenced the final results of this study and
recommend that the effect of occupational and diurnal variations be investigated in future studies.

Group 2, in fact, showed a larger decrease in serum cortisol between baseline and final measurements, than Group 1. This was statistically significant \( p=0.005 \). This larger decrease in serum cortisol levels is possibly due to the fact that in Group 2 there was a greater time interval between baseline and final measurements of serum cortisol.

Group 1 showed a minimal decrease in serum cortisol levels after a low back spinal manipulation. Group 2 showed the largest decrease between pre-treatment A and pre-treatment B (i.e. rest interval). Thus, it appears that resting between measurements had a greater impact on serum cortisol levels than a low back spinal manipulation. These results are not in keeping with the proposed hypothesis that low back spinal manipulation may possibly cause a short-term increase in serum cortisol levels. It was possible that this could have lead to a decrease in inflammation on the affected area since cortisol is an anti-inflammatory hormone. Therefore we fail to accept the null hypothesis and accept the alternate hypothesis with respect to SMT and serum cortisol release and the effect of short-term rest and serum cortisol release. The results of the present study showed a minimal decrease in serum cortisol levels after SMT which are in keeping with the results of Tuchin (1998) and Whelan et al. (2002) who both showed a decrease in salivary cortisol levels after SMT. Even though the results of all three studies are the similar, each study had methodological differences which are mentioned below.

When comparing the present study to previous studies, it was found that the study by Tuchin (1998) questioned the use of blood testing due to possible rises in the cortisol levels because of the invasive nature of vein puncture required for blood sampling. He further questioned false increases in cortisol may occur due to the physical stress of sampling. It was therefore expected in this present study, due to the invasive nature of intravenous samples that
the cortisol would most certainly have risen at the least to borderline significance \((p=0.005)\).

The results of this present study have shown the opposite, serum cortisol levels in fact significantly decreased considering intravenous sampling was used. This then suggests that the use of salivary cortisol in Tuchin (1998) should be questioned since salivary cortisol only closely reflects the plasma levels of cortisol and does not emulate serum cortisol levels (Kahn \textit{et al.}, 1988).

The results of Whelan \textit{et al.} (2002) should therefore also be questioned due to the use of salivary cortisol rather than serum cortisol. Whelan \textit{et al.} (2002) concluded from their results that spinal manipulation had no significant affect on salivary cortisol level. He went on further to suggest that the physical component of spinal manipulation is not a potent enough stressor to disrupt homeostatic mechanisms and activate the hypothalamic-pituitary-adrenal axis. The results of the present study show that spinal manipulation did have an effect on serum cortisol albeit cortisol levels decreased hence the physical component of spinal manipulation is able to affect the homeostatic mechanisms and activate the hypothalamic-pituitary-adrenal axis.

The findings, although not supporting the initial hypothesis proposed, showed a decrease in short-term serum cortisol levels after low back spinal manipulation. This unexpected decrease in serum cortisol levels gives rise to numerous unexplained questions. These questions result in the formulation of the following explanations.

\textbf{5.3. PROPOSED EXPLANATIONS FOR OBSERVED FINDINGS:}

\textbf{Explanation One}

The following explanation is based on the physiology of cortisol catabolism as outlined in Ganong (2001). Cortisol is metabolised by the liver, which is the principle site of glucocorticoid catabolism. During an individual's
exposure to stress, the rate of hepatic inactivation of cortisol is depressed. It is possible that cortisol was released immediately after a subject was exposed to SMT (SMT is considered a stressor). This resulted in a depression of liver catabolism of cortisol, thus the gradient on Figure 4.1 appears shallow. However when we look at the gradient on Figure 4.2 between Pre-treatment A and Pre-treatment B, we see that is much steeper than gradient between Pre-treatment B and Post-treatment C (when the individual was exposed to SMT). The individual was resting during the interval Pre-treatment A and Pre-treatment B, was not exposed to a stressor and thus the liver metabolism of cortisol was essentially normal or possibly increased. This indicates the normal response of a body to a stressor or rest with respect to serum cortisol catabolism and not really linked to a neural pathway from the lumbar spine to the adrenal glands.

Explanation Two
After a 5 minute rest period and spinal manipulation there is an effect on the neuroendocrine system resulting in the release of serum cortisol together with another substance that for purpose of this research shall be referred to as "Substance X". However, "Substance X", possibly a neuropeptide, secretes negative substances that have a denaturing effect on circulating cortisol, hence causing a quantitative decrease of circulating serum cortisol. The question that arises and requires further research is does a "Substance X" exist and if so, what is the nature and control mechanism of this endogenous chemical. This further research has a tremendous impact on clinical medicine, in the future. After an exhaustive search in the literature (books, journals, Internet and personal communication with a chemical pathologist (Naidoo, 2005)) the researcher could not find anything similar in the human body where two substances are secreted simultaneously and one denatures the other.

Explanation Three
Cortisol is metabolised rapidly, mainly by the liver, and has a plasma half-life of approximately 2 hours. The metabolic clearance rate is 200L per day. It is not known how soon after a "stressor", cortisol is expected to be
released but what is known is that there is a several minute lag time in humans between AcTH stimulation and cortisol release (DeGroot et al., 1989). It is also possible that the blood samples were taken too early to detect any changes in the serum cortisol levels. Due to budget constraints we were also not able to carry out renal sampling over at least one hour.

In keeping with the above, it is proposed that in patients with inflammation do in fact have their cortisol released after a spinal manipulation but the cortisol is denatured at a faster rate than in patients without inflammation. It is reasoned that the levels of cortisol in this study therefore decreased due to the analysis of the hormone levels in symptomatic patients (Ganong, 2001).

**Explanation Four**

Inflammation has nothing to do with cortisol release and possibly other substances are actually involved in the process. The presence or absence of such substances needs to be elucidated in future studies.

**Explanation Five**

Spinal manipulation interferes with microcirculation in the adrenal cortex, which may give rise to relative ischaemia and therefore temporarily decreases circulating blood levels in systemic circulation (Leach, 1994). This is proposed on the basis of the anatomical proximity of the blood supply of the adrenal gland to the lumbar spine as well as alterations in the curvature of the lumbar lordosis during the SMT (Gray, 1974).

**Explanation Six**

The study was conducted in a young sample population of otherwise healthy males. It is proposed that since the resting basal levels were sufficiently "high" for the anti-inflammatory needs of this study group, the SMT did not result in a further increase. It is suggested that if a similar study were conducted in an older patient group, the levels would significantly increase after SMT. The rationale for this hypothesis is that
relative and temporary arterial ischaemic processes involving the adrenal gland is much more prominent in an older age group (Beale et al., 2002).

**Explanation Seven**

Since there are no significant reasons for the decrease in the 2 groups it therefore stands to reason that the higher the initial level, the more dramatic is the decrease and this could only be brought about by a “mass effect” of denaturing a large amount of circulating cortisol.

**Explanation Eight**

This study protocol catered for the analysis of the cortisol levels after a five-minute interval, post-SMT. It is proposed that the increase occurs well after this period, the exact period of time is unknown at present and further studies are indicated in this area. For this reason, in the present study, an objective increase in the cortisol level was not noted in the study group in view of the time interval elapsed between the SMT and sample collection.

**5.4. Study limitations**

Missing values for statistical analysis was due to haemolysis of samples prior to being analysed. These missing values could certainly have affected the final statistical analysis of the present study.

Room lighting may also have an affect on cortisol levels and therefore should also be considered (Ganong, 2001). In this study the effect of ambient lighting was minimized on the test subjects by making the subjects wear a standard airline issue eye shades prior to the collection of samples. Ideally the test subjects should be placed in a darkened room for collection of cortisol samples. However, this would not be practical and may even add to the “stressor” effect on the subjects.

Further studies could possibly include estimation of serum AcTH levels which could not be incorporated into the present study for reasons of financial constraints.
CHAPTER 6
CONCLUSIONS AND RECOMMENDATIONS

6.1. CONCLUSION
The aim of this study is to determine if serum cortisol increases immediately after low back SMT. Our hypothesis proposed an increased serum cortisol level post-SMT. This could have lead to a decrease in inflammation on the affected area. The following conclusions maybe drawn based on the objective laboratory tests carried out in this study:

- The cortisol assays in this study indicate that there was a non-significant decrease in serum cortisol levels post-low back spinal manipulation but there was a significant decrease in serum cortisol levels between the initial blood sample and the 5-minute rest period. These findings are contradictory to what was theoretically expected to happen.

- The results of this study support the fact that a neural and endocrine effect can be stimulated by rest and to a lesser extent low back spinal manipulation, albeit a decrease in serum cortisol occurred. This mechanism that results in the decrease of cortisol is not established and requires further investigation.

- Finally, this study has been unable to show an increase in serum cortisol levels after a low back spinal manipulation. Further research is necessary to determine what role SMT may have on decreasing facet joint inflammation and its proposed neural and endocrine affects that have been advanced based on previous studies.

6.2. RECOMMENDATIONS
The author is of the opinion that the following recommendations could improve the validity of future studies investigating the effects of low back
spinal manipulation on serum cortisol levels, which may lead to a decrease in inflammation in the affected area.

- In the present study, the sample size was limited to thirty subjects. A larger sample size would minimise the chances of a Type II error and result in the generation of valid study data.
- Future studies must correlate the salivary cortisol levels with serum cortisol assays in the study patients. Financial constraints in the present study precluded this aspect of the analysis.
- The effect of low back spinal manipulation on long term serum cortisol levels should be assessed i.e. the post-treatment blood test for cortisol assays should be done at least an hour or more after the low back spinal manipulation.
- The present study suggests that there are indication to perform serum AcTH assays in future studies involving LBP.
- Correlating laboratory results with clinical data e.g. NRS or clinical case history and examination findings.
- Studies to determine the time of cortisol release following a stressor and the time and nature of its degradation.
- Correlating serum cortisol levels and release with its effect on inflammatory markers such as C reactive proteins (CRP) and erythrocyte sedimentation rate (ESR).
- Studies to determine the effect of ethnicity, seasonal and dietary variations on baseline serum cortisol levels.
REFERENCES


45. Robertson, E.J. 2004. Professor and Head: Department of Chemical Pathology. *Personal communication*.


Dear participant:

TITLE OF STUDY: THE IMMEDIATE EFFECT OF LOW BACK MANIPULATION ON SERUM CORTISOL LEVELS IN ADULT MALES WITH MECHANICAL LOW BACK PAIN.

Thank you for agreeing to participate in my study. The aim of this study is to show the immediate effect of lumbar spinal manipulation on serum cortisol levels.

Serum cortisol also known as blood cortisol is to date known as the most accurate method of measuring cortisol. Cortisol is a hormone released by the body's adrenal gland. Cortisol follows a circadian rhythm which refers to the variation of cortisol in the blood throughout the day, it is at its highest between 7am to 8am and progressively decreases throughout the day. Cortisol has many important functions but the one most important to this study is its ability to decrease inflammation and thus speed up the recovery phase of an injury.

It is thought that inflammation of the small joints of the spine (facet joints) can give rise to acute mechanical low back pain. Manipulation, or more commonly known as an adjustment, is a high velocity, low amplitude thrust directed onto a specific joint (in this study a lower back joint) to improve joint alignment, range of motion and quality of movement.

Theoretically it seems possible that spinal manipulation can increase serum cortisol levels, which may decrease inflammation in the low back. Thirty people will be required to participate in this study, provided that they fall within the prescribed inclusion criteria ie: males between the ages of 18 to 35, patients who are diagnosed with acute facet syndrome of the low back and patients who are not apprehensive of needles. The participants will be
randomly divided into two groups by drawing a letter from a hat, therefore having an equal chance of being in either group.

The treatment will be carried out over a two-day period.

Day 1- a full history will be taken followed by a physical examination and the relevant regional examination from both groups at the Chiropractic Day Clinic (Berea). Patients will also be required to complete an Numerical Pain Rating Scale(NRS). This first visit should be approximately 2 to 2½ hours long.

Day 2- blood will be drawn from both groups at approximately 7:50 am at the Nelson R. Mandela school of Medicine (Umbilo). This second visit should be approximately 1 hour long.

One group of fifteen will receive a low back spinal manipulation at 7:55 am and a blood test thereafter; the other group of fifteen will rest on their back for 5 minutes and then have a second blood test, they will then rest for a further 5 minutes, receive a low back spinal manipulation and then have a third blood test.

Both groups will receive one more free manipulation at a later date in consultation with the researcher. Thus all thirty patients will receive a total of two manipulations throughout the entire study. All free manipulations done after Day 2 of the study will be done at the Chiropractic Day Clinic, to the convenience of the researcher and patient but within a week following Day 2 of the study.

Due to the circadian nature of cortisol (explained above) it is important to the results of the study that all patients abide by the times specified by the researcher. A medical doctor will draw blood samples and new sterile disposable equipment (Brand new sterile needle which will be opened in front of you and then immediately disposed of after use.) will be used as per HIV prevention protocol at the Nelson R. Mandela School of Medicine.

All treatments will be performed under the supervision of a qualified chiropractor and general practitioner and will be free of charge. Your participation in this study is voluntary and you may withdraw at anytime. You
are assured that confidentiality will be maintained and the results will be used for research purposes only. If there are any complaints about this study, please contact my supervisor or co-supervisor.

Thank you for participating in the study.

Keseri Padayachy
(Chiropractic intern / Researcher- ph. 031-2042512)

Dr J. Shaik
Supervisor (ph. 031-2042588)

Professor G.H.M. Vawda
Co-supervisor (ph. 031-2604576)
INGWADI YESAZISO

ISIHLOKO SESITADI: UMPHUMELA WOKUQONDISA KWAMALUNGA EQOLO KWISERUM CORTISOL LEVELS KUMUNTU WESILISA OMDALA OPHATWA YIQOLO.

Siyabonga ukuthi uzibandakanye kulesisitadi. Injongo yalesiita di ukutshengisa umphumela wokuqondiswa kwamalunga eqolo emazingeni eserum cortisol.

Iserum cortisol yaziwa njenge cortisol yegazi ekunamhlanje yaziwa njengendlela ekuyiyona yona ukukala icortisol. Icortisol ingumkhiqizo womzimba wama-adrenal gland. Icortisol ilandela ukwehlukanisa nge circadian ephathelena nazinhlobo ze cortisol ezitholakala egazini ekuhambeni kwelanga, isemazingeni aphezulu phakathi kukia 7am to 8am bese iqhubeka yehla ngokuhamba kwelanga. Icortisol inemisebenzi ebalulekile kodwa obaluleke kunayoyohe kulesisitadi ukwehlisa ubukhubela ngalokho yenyusa emazinga okuphila.

Kucatshangwa ukuthi ubukhubela kwamalunga omgogodla kungaholela ekuphathweni iqolo. Ukuqondiswa kwamalunga omzimba ukusebenzisa amandla esheshayo asezingeni eliphansi elungeni elithila lomzimba(eqolo ngokwalesisitadi) ukuze amalunga omzimba ame ngendlela okuyiyokayona, kanye nokuhamba kwawo, kanye namazinga.

Ngokwezifundo kubukeka sengathi ukuqondiswa kwamalunga omzimba kungenyusa amazinga ecortisol, okunehlisa ubukhubela beqolo. Abantu abangamashumi amathathu bazodingeka ukuba bazibandakanye kulesisitadi, uma bengena kuloluhta: abesilisa abaphakathi kweminyaka engu 18 kuya ku 35, iziguli eziphethwe yiqolo(amalunga eqolo) neziguli ezingesabi izinaliti. Abazozibandakanya bazohlaniswa babe ngamaqembu amabili
ngokutonyulwa kwamagama esigqokweni ukuze babe nethuba ellilinganayo ukuba kunoma iliphi iqembi.

Ukelashwa kuzothatha izinsuku ezimbili. Ngosuku lokuqala kuzothathwa iminginingwane egcwele ngeqolo elikuhluphayo bese-uyaxilongwa ekliniki/emtholampilo we Chiropractic (odokotela bomgogodla namalunga omzimba) eBerea. Iziguli kuzodingeka ukuba zigcwallise iNRS. UkuvAkasha emtholampilo kokuqala kunzaththa 2 kuyaku 2.5 wemahora.

Usuku lxesibili kuzothatha igazi kuwo womabili amaqembu ngo 7:50 ekuseni eNelson R. Mandela School of Medicine(Umbilo). Ukuvasha esibhdedlela kangathatha ihora elilodwa. Iqembu elilodwa lizothola ukuqondiswa kwamalungu eqolo ngo 7:55 ekuseni bese kuthathwa igazi, elinaye iqembu lesibili lizophumula ngemihlanie imizuzu emihlanu besa kuthathwa igazi kwesibili, bese beyaphumula eminye imizuzu emihlanu, bese bethole ukuqondiswa kwemalungu omgogodla, bese kuthathwa igazi okwesithathu.

Womabili emeqembu azobe esethole ukwelashwa mahhala uma sekuphela isitadi. Ngakhoke zonke iziguli ezingamashumi amathathu zizothola ukuqondiswa kwamalungu eqola kabilie ngokuqhubeke kwesitadi. Ukwalashwa kwamahhala emva kosukulwesibi kuzokwenzelwa e Chiropractic Clinic, kuza yo ngohuthanda kwesiguli nomqhubi wesitadi evikini emva kosuku lxesibiliwesitadi.

Ngenxa yemvelo yecortisol(esichaziwe ngaphezulu) kubelulekile ukuba imiphumela yalesisitadi ukuba zonke iziguli zigcine isikhathi ngokusho kowengamele isitadi. Udokotela uzothatha igazi ngamjovo omusha ohlanziwe osebenza kanye (umjovo omusha ozovulwe phambi kwesiguli ulahlwe uma ususebenzile) ngenxa yokuvikela ugawulayo nentsholongwane yawo e Nelson R. Mandela School of Medicine.

Konke ukwelashwa kuzokwenziwa phemb/ ngokwengamela kwe Chiropractor noDokotela, kuzebe kumahlala. Ukuzindakanya kwakho kukuwe ungayeka
noma kunini. Uyaqinisekiswa ukuthi iniminingwane yakho iyimfihlo egciniwe
nemiphumela seyitadi izosetshenziswa ngenhloso yesitadi kuphela. Uma
unokungathokozi ngesitadi ungaxhumana nowengemala isitadi.
Siyabonga ngokuzibandakanya kulesisitadi.

…………………………
Keseri Padayachy
(Chiropractic intern/ Researcher)/ umqhubi wesitadi

…………………………
Dr J. Shaik
Owengamele isitadi( ph. 031-2042588)

…………………………
Professor G.H.M. Vawda
Obmbisene nowengamele isitadi (ph 031-2604576)
INFORMED CONSENT FORM
(To be completed by patient/subject)

Date: June 07, 2004

Title of research project:
The immediate effect of lumbar spinal manipulation on serum cortisol levels in adult males with mechanical low back pain.

Name of supervisor: Dr. J. Shaik (MTech.chiro)
Name of co-supervisor: Professor G.H.M. Vawda (BSC, MBChB, FCS(1) SA, PhD. Wits)
Name of research student: Keseri Padayachy

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

If you have answered NO to any of the above, please obtain the necessary information before signing.

Please Print in block letters:
Patient/Subject Name: __________________________ Signature: ________________
Witness Name: ________________________________ Signature: ________________
Research Student Name: ______________________ Signature: ________________
INCWADI YEMVUMO
(yokugcwaliswa isiguli)

Usuku: November 11, 2004

Isihloko sesitadi:
Umphumela wokuqondiswa kwamalunga eqolo kwiserum cortisol levels
kumuntu wesilisa omdala ophathwa iqolo.

Igama lowengamele: Dr J. Shaik (M.Tech.Chiro)
Igama lobambisena nowengemele: Professor G.H.M. Vawda
(BSc, MBChB, FCS(1)SA, PhD. Wits)

Igama lomqhubi/umfundisi: Keseri Padayachy

Sicela wenze inginiliza kwimpendulo okuyiyi

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<td>2.</td>
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<td>3.</td>
<td>Kungabe uthole izimpendulo eziculisayo?</td>
</tr>
<tr>
<td>4.</td>
<td>Ulitholile ithuba lokubonisana ngesitadi?</td>
</tr>
<tr>
<td>5.</td>
<td>Ulitholile ulwazi olwanele ngesitadi?</td>
</tr>
<tr>
<td>6.</td>
<td>Uyayizwa imigomo yesitadi nokuzibandakanya?</td>
</tr>
<tr>
<td>7.</td>
<td>Uyazi ukuthi ungayeka noma kunini kulesisitadi?</td>
</tr>
<tr>
<td></td>
<td>a. nomaphasi isikhathi</td>
</tr>
<tr>
<td></td>
<td>b. ngale kokubeka isizathu sokuyeka</td>
</tr>
<tr>
<td></td>
<td>c. ngale kokubeka engozini impilo yakho</td>
</tr>
<tr>
<td>8.</td>
<td>kungabe uvuma ngokwakho ukuzibandakanya?</td>
</tr>
<tr>
<td>9.</td>
<td>Ubani okhulume naye?</td>
</tr>
</tbody>
</table>

Uma uth cha ezimphendulweni ezingaphezulu, sicela uthole ulwazi olufanele
ngaphambili kokusayina.

Sicela ubhale ngokwehlukanisa:

<p>| | |</p>
<table>
<thead>
<tr>
<th></th>
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<tbody>
<tr>
<td>Igama lesiguli:</td>
<td>sayina:</td>
</tr>
<tr>
<td>Igama lobonayo:</td>
<td>sayina:</td>
</tr>
<tr>
<td>Igama lomqhubi Wesitadi:</td>
<td>sayina:</td>
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</tbody>
</table>
Intern's Case History:

1. Source of History:

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

3. Present Illness:

<table>
<thead>
<tr>
<th>Complaint 1</th>
<th>Complaint 2</th>
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<tbody>
<tr>
<td>Location</td>
<td></td>
</tr>
<tr>
<td>Onset: Initial:</td>
<td></td>
</tr>
<tr>
<td>Recent:</td>
<td></td>
</tr>
<tr>
<td>Cause:</td>
<td></td>
</tr>
<tr>
<td>Duration</td>
<td></td>
</tr>
<tr>
<td>Frequency</td>
<td></td>
</tr>
<tr>
<td>Pain (Character)</td>
<td></td>
</tr>
<tr>
<td>Progression</td>
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</tr>
<tr>
<td>Aggravating Factors</td>
<td></td>
</tr>
<tr>
<td>Relieving Factors</td>
<td></td>
</tr>
<tr>
<td>Associated S &amp; S</td>
<td></td>
</tr>
<tr>
<td>Previous Occurrences</td>
<td></td>
</tr>
<tr>
<td>Past Treatment</td>
<td></td>
</tr>
<tr>
<td>Outcome:</td>
<td></td>
</tr>
</tbody>
</table>

4. Other Complaints:

5. Past Medical History:
   - General Health Status
   - Childhood Illnesses
   - Adult Illnesses
   - Psychiatric Illnesses
   - Accidents/Injuries
   - Surgery
   - Hospitalizations
6. **Current health status and life-style:**
   - Allergies
   - Immunizations
   - Screening Tests incl. x-rays
   - Environmental Hazards (Home, School, Work)
   - Exercise and Leisure
   - Sleep Patterns
   - Diet
   - Current Medication
     - Analgesics/week:
   - Tobacco
   - Alcohol
   - Social Drugs

7. **Immediate Family Medical History:**
   - Age
   - Health
   - Cause of Death
     - DM
     - Heart Disease
     - TB
     - Stroke
     - Kidney Disease
     - CA
     - Arthritis
     - Anaemia
     - Headaches
     - Thyroid Disease
     - Epilepsy
     - Mental Illness
     - Alcoholism
     - Drug Addiction
     - Other

8. **Psychosocial history:**
   - Home Situation and daily life
   - Important experiences
   - Religious Beliefs
9. Review of Systems:

- General
- Skin
- Head
- Eyes
- Ears
- Nose/Sinuses
- Mouth/Throat
- Neck
- Breasts
- Respiratory
- Cardiac
- Gastro-intestinal
- Urinary
- Genital
- Vascular
- Musculoskeletal
- Neurologic
- Haematologic
- Endocrine
- Psychiatric

13 Jan 2003
Appendix 6

DURBAN INSTITUTE OF TECHNOLOGY
CHIROPRACTIC DAY CLINIC
PHYSICAL EXAMINATION

Patient: ___________________________ File#: _________ Date: _________

Clinician: __________________________ Signature: __________________

Student: __________________________ Signature: __________________

1. **VITALS**

   Pulse rate: ________________________ Respiratory rate: _________
   Blood pressure: R L ________________________ Medication if hypertensive:
   Temperature: ________________________ Height: __________________
   Weight: ____________________________ Any change Y/N If Yes: how much gain/loss _________
   Over what period _________

2. **GENERAL EXAMINATION**

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

3. **CARDIOVASCULAR EXAMINATION**

   1) Is this patient in Cardiac Failure?
   2) Does this patient have signs of Infective Endocarditis?
   3) Does this patient have Rheumatic Heart Disease?

   Inspection - Scars
   - Chest deformity:
   - Precordial bulge:
   - Neck -JVP:

   Palpation: - Apex Beat (character + location):
   - Right or left ventricular heave:
   - Epigastric Pulsations:
   - Palpable P2:
   - Palpable A2:

Auscultation: - heart valves (mitral, aortic, tricuspid, pulmonary) - Murmurs (timing, systolic/diastolic, site, radiation, grade).

4. RESPIRATORY EXAMINATION

1) Is this patient in Respiratory Distress?

Inspection - Barrel chest: - Pectus carinatum/cavatum: - Left precordial bulge: - Symmetry of movement: - Scars:

Palpation - Tracheal symmetry: - Tracheal tug: - Thyroid Gland: - Symmetry of movement (ant + post) - Tactile fremitus:

Percussion - Percussion note: - Cardiac dullness: - Liver dullness:

Auscultation - Normal breath sounds bilat.: - Adventitious sounds (crackles, wheezes, crepitations) - Pleural frictional rub: - Vocal resonance - Whispering pectoriloquy: - Bronchophony: - Egophony:

5. ABDOMINAL EXAMINATION

1) Is this patient in Liver Failure?

Inspection - Shape: - Scars: - Hernias:

Palpation - Superficial: - Deep = Organomegally: - Masses (intra- or extramural) - Aorta:

Percussion - Rebound tenderness: - Ascites: - Masses:

Auscultation - Bowel sounds: - Arteries (aortic, renal, iliac, femoral, hepatic)
6. **G.U.T EXAMINATION**

External genitalia:
- Hernias:
- Masses:
- Discharges:

7. **NEUROLOGICAL EXAMINATION**

Gait and Posture
- Abnormalities in gait:
- Walking on heels (L4-L5):
- Walking on toes (S1-S2):
- Rombergs test (Pronator Drift):

Higher Mental Function
- Information and Vocabulary:
- Calculating ability:
- Abstract Thinking:

**G.C.S.:**
- Eyes:
- Motor:
- Verbal:

Evidence of head trauma:

Evidence of Meningism:
- Neck mobility and Brudzinski's sign:
- Kernigs sign:

Cranial Nerves:

I Any loss of smell/taste:
- Nose examination:

II External examination of eye:
- Visual Acuity:
- Visual fields by confrontation:
- Pupillary light reflexes = Direct:
- = Consensual:
- Fundoscopy findings:

III Ocular Muscles:
- Eye opening strength:

IV Inferior and Medial movement of eye:

V a. Sensory
- Ophthalmic:
- Maxillary:
- Mandibular:

b. Motor
- Masseter:
- Jaw lateral movement:

c. Reflexes
- Corneal reflex
- Jaw jerk

VI Lateral movement of eyes
VII  a. Motor - Raise eyebrows:
    - Frown:
    - Close eyes against resistance:
    - Show teeth:
    - Blow out cheeks:

b. Taste - Anterior two-thirds of tongue:

VIII  General Hearing:
Rinnes = L: R:
Webers lateralisation:
Vestibular function - Nystagmus:
    - Rombergs:
    - Wallenbergs:
Otoscope examination:

IX & VIII Gag reflex:

X  Uvula deviation:
Speech quality:

XI  Shoulder lift:
S.C.M. strength:

XII  Inspection of tongue (deviation):
Motor System:
  a. Power
    - Shoulder = Abduction & Adduction:
    = Flexion & Extension:
    - Elbow = Flexion & Extension:
    - Wrist = Flexion & Extension:
    - Forearm = Supination & Pronation:
    - Fingers = Extension (Interphalangeals & M.C.P's):
    - Thumb = Opposition:
    - Hip = Flexion & Extension:
    = Adduction & Abduction:
    - Knee = Flexion & Extension:
    - Foot = Dorsiflexion & Plantar flexion:
    = Inversion & Eversion:
    = Toe (Plantarflexion & Dorsiflexion):
  b. Tone
    - Shoulder:
    - Elbow:
    - Wrist:
    - Lower limb - Int. & Ext. rotation:
    - Knee clonus:
    - ankle clonus:
  c. Reflexes
    - Biceps:
    - Triceps:
    - Supinator:
    - Knee:
    - Ankle:
    - Abdominal:
    - Plantar:
Sensory System:

a. Dermatomes
   - Light touch:
   - Crude touch:
   - Pain:
   - Temperature:
   - Two point discrimination:

b. Joint position sense
   - Finger:
   - Toe:

c. Vibration:
   - Big toe:
   - Tibial tuberosity:
   - ASIS:
   - Interphalangeal Joint:
   - Sternum:

Cerebellar function:

Obvious signs of cerebellar dysfunction:
   = Intention Tremor:
   = Nystagmus:
   = Truncal Ataxia:

Finger-nose test (Dysmetria):
Rapid alternating movements (Dysdiadochokinesia):
Heel-shin test:
Heel-toe gait:
Reflexes:
Signs of Parkinsons:

8. SPINAL EXAMINATION: (See Regional examination)

Obvious Abnormalities:
Spinous Percussion:
R.O.M:
Other:

9. BREAST EXAMINATION:

Summon female chaperon.

Inspection
   - Hands rested in lap:
   - Hands pressed on hips:
   - Arms above head:
   - Leaning forward:

Palpation
   - masses:
   - tenderness:
   - axillary tail:
   - nipple:
   - regional lymph nodes:
Appendix 7

TECHNIKON NATAL CHIROPRACTIC DAY CLINIC
REGIONAL EXAMINATION - LUMBAR SPINE AND PELVIS.

PATIENT: ____________________________________________

FILE #: ___________________ DATE: ________________

INTERN/RESIDENT: ________________________________

SUPERVISING CLINICIAN: ____________________________

STANDING:

<table>
<thead>
<tr>
<th>Posture</th>
<th>Spinous Percussion</th>
</tr>
</thead>
<tbody>
<tr>
<td>Minor's Sign</td>
<td>Schober's Test (6cm)</td>
</tr>
<tr>
<td>Skin</td>
<td>Treadmill</td>
</tr>
<tr>
<td>Scars</td>
<td>Body Type</td>
</tr>
<tr>
<td>Discoloration</td>
<td>Attitude</td>
</tr>
<tr>
<td>Muscle Tone</td>
<td></td>
</tr>
<tr>
<td>Bony &amp; Soft Tissue Contours</td>
<td></td>
</tr>
</tbody>
</table>

RANGE OF MOTION

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

SUPINE:

<table>
<thead>
<tr>
<th>Skin</th>
<th>Observe abdomen</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hair</td>
<td>Fasciculations</td>
</tr>
<tr>
<td>Nails</td>
<td>Abdominal Reflexes</td>
</tr>
<tr>
<td>Palpate Abdomen/groin</td>
<td></td>
</tr>
<tr>
<td>Pulses (abdomen)</td>
<td></td>
</tr>
</tbody>
</table>
Pulses (extremities)
SLR
Bowstring
Plantar Reflex
Circumference (thigh, calf)
Leg Length:
- actual
- apparent
Sciatic Notch
Patrick FABERE
Gaensien's Test
Gluteus Maximus Stretch
Hip Medial rotation
Psoas Test
Thomas' Test:
  - hip joint
  - Rectus Femoris

**LATERAL RECUMBENT**

S-I Compression
Ober's Test
Femoral Nerve stretch
Myotomes:
  - QL
  - Gluteus Medius

**NON ORGANIC SIGNS**

Pin Point Pain
Axial Compression
Trunk Rotation
Burn's Bench Test
Flip Test
Hoover's Test
Ankle Dorsiflexion Test.

**GAIT**

Rhythm
On toes (standing)
On Heels (standing)
Half squat on one leg

**PRONE**

Gluteal skyline
Skin rolling
Iliac crest compression
Facet joint challenge
S-I tenderness
Erichson's Test
Pheasant's Test
Myotome:
  - Glut. Max
Active MF Trigger Pts:
  - QL
  - Glut. Med
  - Glut. Min
  - Glut. Max
  - Piriformis
  - Hamstrings
  - TFL

**Rhythm**

On toes (standing)
On Heels (standing)
Half squat on one leg
# Neurological Examination

<table>
<thead>
<tr>
<th>Dermatomes</th>
<th>Myotomes</th>
<th>Reflexes</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>L</strong></td>
<td><strong>R</strong></td>
<td><strong>L</strong></td>
</tr>
<tr>
<td>T12</td>
<td>Hip Flex</td>
<td>Pat.</td>
</tr>
<tr>
<td>L1</td>
<td>Hip int rot</td>
<td>Achil</td>
</tr>
<tr>
<td>L2</td>
<td>Hip ext rot</td>
<td>H/S</td>
</tr>
<tr>
<td>L3</td>
<td>Hip abd</td>
<td></td>
</tr>
<tr>
<td>L4</td>
<td>Hip add</td>
<td></td>
</tr>
<tr>
<td>L5</td>
<td>Knee flex</td>
<td></td>
</tr>
<tr>
<td>S1</td>
<td>Knee ext</td>
<td></td>
</tr>
<tr>
<td>S2</td>
<td>Dorsiflex</td>
<td></td>
</tr>
<tr>
<td>S3</td>
<td>Plantarflex</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Eversion</td>
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<td></td>
<td>Ext. hal. long</td>
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</tbody>
</table>

Tripod
Kemp's Test

# Motion Palpation and Joint Play:

**Left:**
- Upper Thoracics:
- Lumbar Spine:
- Sacroiliac Joint:

**Right:**
- Upper Thoracics:
- Lumbar Spine:
- Sacroiliac Joint:

Basic Exam: Hip
Case History:

ROM: Active:
Passive:
RIM:
Orthopaedic/Neuro/
Vascular:
Observ/Palpation:

Basic Exam: Thoracic Spine
Case History:

ROM: Motion Palp:
Active:
Passive:
Orthopaedic/Neuro/
Vascular:
Observ/Palpation:
DURBAN INSTITUTE OF TECHNOLOGY CHIROPRACTIC DAY CLINIC

Name of patient:

Date:

NUMERICAL RATING SCALE 101

Please indicate on the line below the number between 0 and 10 that best describes the pain of your major problem at this point, when it is at its WORST. A zero (0) would mean “no pain at all” and a ten (10) would mean “pain as bad as it could be”

Please write only one number.

0 __________________________________________ 10
Are you 
MALE 
and aged between 
18 - 35 years 
and suffer from 
LOWER BACK 
PAIN?

Research is currently being carried out 
at the 
DURBAN INSTITUTE OF TECHNOLOGY 
CHIROPRACTIC DAY CLINIC.

FREE TREATMENT 
Is available to those who qualify to take part in this study.

AS PART OF THIS STUDY, BLOOD TESTS WILL BE TAKEN BY THE RESEARCHER.

for more information 
Contact 
KESERI PADAYACHY 
on 
(031) 2042205/2512. or 
084 3716 438
RESEARCH ETHICS COMMITTEE

Student: KESERI PADA-YACHY
Student No.: 20000673

Research Title: THE IMMEDIATE EFFECT OF LOW BACK MANIPULATION ON SERUM CORTISOL LEVELS IN ADULT MALES WITH MECHANICAL LOW BACK PAIN.

A. The proposal meets the professional code of ethics of the Researcher

☑ Yes ☐ No

B. The proposal also meets the following ethical requirements

<table>
<thead>
<tr>
<th>YES</th>
<th>NO</th>
</tr>
</thead>
<tbody>
<tr>
<td>☑ Provision has been made to obtain informed consent of the participants</td>
<td></td>
</tr>
<tr>
<td>☑ Potential psychological and physical risks have been considered and minimised</td>
<td></td>
</tr>
<tr>
<td>☑ Provision has been made to avoid undue intrusion with regard to participants and community</td>
<td></td>
</tr>
<tr>
<td>☑ Rights of participants will be safeguarded in relation to:</td>
<td></td>
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<tr>
<td>- Measures for the protection of anonymity and the maintenance of Confidentiality.</td>
<td>☑</td>
</tr>
<tr>
<td>- Access to research information and findings.</td>
<td>☑</td>
</tr>
<tr>
<td>- Termination of involvement without compromise</td>
<td>☑</td>
</tr>
<tr>
<td>- Misleading promises regarding benefits of the research</td>
<td>☑</td>
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</tbody>
</table>

Signature of Student

Signature of Supervisor

Signature of Head of Department

Signature of Chairperson of the Faculty

Date 26/08/05
Date 26 August 2005
Date 26/08/05
Date 26/08/05