

**The Effect of Cervical and Thoracic Spinal Manipulations on  
Blood Pressure in Normotensive Males.**

By

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Dissertation submitted in partial compliance with the requirements for the  
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I, Angela Niky Pastellides, do declare that this dissertation is representative  
of my own work in both conception and execution (except where  
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## **DEDICATION**

I'd like to dedicate this work and accomplishment in memory of my dearest Yia-yia, you are forever in my thoughts, forever in my heart, forever missed. Love you always.

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## **ABSTRACT**

### **The Effect of Cervical and Thoracic Spinal Manipulations on Blood Pressure in Normotensive Males.**

#### **BACKGROUND**

A distinguishing feature of chiropractic is manipulation that is a load delivered by hand, to specific tissues (usually a short lever bony prominence) with therapeutic intent. Chiropractic spinal manipulation results in somatovisceral reflexes, which can affect the cardiovascular system and thereby reduce blood pressure. Areas of the spine known to cause such effects are the upper cervical region and the upper thoracic region. Increased blood pressure/hypertension is a global disorder. The incidence is increasing and leads to complications of cardiovascular disease and cerebral vascular accidents

#### **OBJECTIVES**

The objectives of the study were to determine whether spinal manipulation evokes somatovisceral reflexes and causes a reduction in blood pressure following an atlanto-axial ( $C_0/C_1$ ), and Thoracic segments one to five manipulations ( $T_1-T_5$ ).

#### **METHODS**

Forty, asymptomatic, normotensive males between the ages of 20 – 35 years of age participated in the study. All subjects underwent four consecutive days of intervention. Day one was sham laser. Day two was  $C_0/C_1$  spinal manipulation. Day three was  $T_1-T_5$  thoracic manipulation. Day four was a combination of  $C_0/C_1$  and  $T_1-T_5$  spinal manipulations.

#### **RESULTS**

The results of this study suggest that blood pressure decreases following a cervical or a thoracic manipulation, however a combination of the manipulations does not have a significant cumulative effect on the reduction of blood pressure.

## **CONCLUSIONS**

Somatovisceral reflexes are evoked following a spinal manipulation, causing a reduction in blood pressure after an upper cervical or upper thoracic manipulation. Neurophysiological effects occurring as a result of spinal manipulation may inhibit or excite somatosomatic reflexes, which changes heart rate and blood pressure.

## **DEFINITIONS**

### **AGONIST MUSCLE:**

Also known as the prime mover, the muscle that is contracting, primarily responsible for a specific movement is controlled by the opposing simultaneous contraction of another muscle (Dorland's Medical Dictionary, 1994; Web, 2008).

### **ANTAGONIST MUSCLE**

A muscle that acts in opposition to the agonist muscle (Web, 2008).

### **CARDIAC OUTPUT**

The stroke volume and heart rate determines cardiac output. Stroke volume is the amount of blood in litres per minute that is pumped out by the left ventricle in one contraction; this multiplied by the heart rate is cardiac output, thus the amount of blood pumped through the circulatory system in one minute (Web, 2008)

### **CHIROPRACTIC:**

A health profession directed towards the diagnosis, treatment and prevention of disorders affecting the neuromuscular system, and its effects on general health. A particular emphasis is on its manual techniques of manipulations or joint adjustment with a focus on subluxations (WHO, 2005).

### **CREEP**

Physical properties of materials resulting in progressive deformation with a constant load over time, allowing soft tissues to tolerate loads by lengthening (Dorland's Medical Dictionary, 1994).

### **HYSTERESIS**

The lagging of a physical effect on a body due to conditions and forces (Dorland's Medical Dictionary, 1994).

**INCIDENCE:**

The frequency that something such as a disease occurs in a particular area or population. The frequency with which something, such as a disease, appears in a particular population or area. Regarding disease incidence is the number of newly diagnoses cases within a specific time. (Web, 2008)

**INTERNEURON:**

An interneuron is defined as a neuron that receives information from another neuron and transmits it to one or more other neurons (Leach, 1994).

**MALIGNANT HYPERTENSION:**

This is blood pressure that increases rapidly, leading to death within a year or two. Characteristically it consists of a diastolic blood pressure above 120mmHg, renal failure, retinal haemorrhages and exudates with papilledema. It usually occurs in an addition to benign hypertension although it can occur in normotensive individuals (Boon, Colledge, Walker and Hunter, 2006).

**NIDUS:**

The origin of a morbid process. (Dorland's Medical Dictionary, 1994)

**PRESSOR REFLEX:**

The pressor reflex involves neural receptors within a muscle which respond to contraction and are responsible for a set of physiologic effects that occur to increase blood pressure, forcing blood through the contracted muscle (Knutson, 2001).

**PREVALENCE:**

The number of individuals within a population having a particular disease at a specific time (Dorland's Medical Dictionary, 1994).

**SCLEROTOME:**

The area innervated from a single spinal segment (Dorland's Medical Dictionary, 1994) that arose from the same somite during the embryological development of the foetus. This therefore includes all muscular (myotomal), skin (dermatomal), reflex and visceral (enterotomal) structures supplied by one spinal nerve / spinal level (Cramer and Darby, 1995).

**SCLERATOMALLY:**

This is an adjective that derives from "sclerotome" indicating that the structures are related to the same spinal nerve level (Cramer and Darby, 1995).

**SUBLUXATION:**

A dysfunction of a motion segment in which alignment, integrity of movement and physical function are altered, contact between the joint surfaces is intact. It may affect biomechanical and neural integrity (WHO, 2005).



## ABBREVIATIONS

<b>“&lt;”</b>	-	Less than
<b>“&gt;”</b>	-	Greater than
<b>BMI</b>	-	Body Mass Index
<b>C<sub>0</sub>/C<sub>1</sub></b>	-	Atlanto-occipital articulation.
<b>C<sub>1</sub>/C<sub>2</sub></b>	-	Atlanto-axial articulation.
<b>CAM</b>	-	Complementary alternative medicines
<b>CASA</b>	-	Chiropractic Association of South Africa
<b>CAT</b>	-	Complementary alternative therapies
<b>CCEI</b>	-	The Councils on Chiropractic Education International
<b>F</b>	-	The F value is the factor to test for variance.
<b>HIV/AIDS</b>	-	Human Immunodeficiency Virus/Acquired Immunodeficiency Syndrome.
<b>Kg</b>	-	Kilograms
<b>M</b>	-	Metres
<b>mmHg</b>	-	Millimetre’s mercury
<b>MN</b>	-	Motor Neuron
<b><i>p</i> value</b>	-	Measure of how much evidence we have against the null hypothesis by calculating the probability of observing the results from your sample of data or a sample with results more extreme, assuming the null hypothesis is true. The smaller the <i>p</i> -value, the greater the inconsistency
<b>T<sub>1</sub>-T<sub>5</sub></b>	-	Thoracic spinal segments one to five.
<b>VSC</b>	-	Vertebral Subluxation Complex
<b>WFC</b>	-	World Federation of Chiropractic
<b>WHO</b>	-	World Health Organization

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# CHAPTER 1

## INTRODUCTION

### 1.1. INTRODUCTION

Chiropractic is a manual therapy distinguished from other therapies through its manipulation techniques, load delivered by hand, to specific tissues (usually a short lever bony prominence) with therapeutic intent (Haldeman, 2005; World Health Organisation (WHO), 2005). The load is usually of a high velocity, low amplitude, single frequency, to a specific anatomic location, with a specified direction of force and of short impulse duration (Bergmann, Peterson and Lawrence, 1993; Kaptchuk and Eisenberg 1998; Meeker and Haldeman, 2002). The result is to move the vertebrae beyond the clinical physiological range (Vernon and Mrozek, 2005) or elastic barrier (Sandoz, 1976a; Sandoz, 1976b) of the joint without disrupting the anatomical integrity of the joint. On a mechanical level, the result is an increase in range of motion, and a release of synovial gasses causing a cracking sound (Kaptchuk and Eisenberg 1998; Meeker and Haldeman, 2002), whereas on a neurological or physiological level, changes have been associated with sclerotomally associated structures (somatovisceral or somatosomatic reflexes) (Pottenger, 1931; Pottenger and Ussher; Pottenger, 1953; Nansel, Jansen R, Cremata E, Dhami and Holley D, 1991; Herzog, Scheele and Conway, 1999; Hopkins and Ingersoll, 2000; Leach, 2004; Hillermann, 2005).

These effects are thought to be mediated through the nervous system which is responsible for regulating a set of sclerotomal levels (Bergmann, Peterson and Lawrence, 1993). Thus with manipulation the increase in neurological activity (neurological facilitation) that results from this procedure, is thought to correct physiological changes in related sclerotomal structures (Bergmann, Peterson and Lawrence, 1993; Herzog *et al.*, 1999; Pickar, 2002). These physiological changes are either as a result of inhibited activity or stimulatory activity (Pickar, 2002). It can therefore be postulated that manipulation could have an effect on blood pressure.

In terms of Sharma's research, (1992) blood pressure is determined by two endogenous factors: the cardiac output and total peripheral resistance, which is determined by the surface area and luminal size of the arteries and arterioles. Sharma, (1992) also stated that the physiological mechanisms that maintain blood pressure are the autonomic nervous system responses, the capillary shift mechanism, hormonal responses as well as kidney and fluid balance mechanisms.

Research has found that with blood pressure being affected by the autonomic nervous system, there is speculation that cervical spine manipulation could reduce blood pressure following a manipulation (Yates, Lamping, Abraham and Wright, 1988; Sato, 1989; Slosberg, 1998; Budgell, 1999; Bakris, Dickholtz, Meyer, Kravitz, Avery, Miller, Brown, and Bell, 2007). This is further supported by literature indicating that the effects of upper thoracic spine manipulation on blood pressure resulted in a lowering effect on blood pressure (Korr, 1979a; Yates *et al.*, 1988; Sato, 1989; Budgell and Polus, 2006).

It was therefore hypothesised that there may be a synergistic effect if both regions were manipulated (viz. cervical and thoracic spine regions) and thus this study was structured to investigate the effect of cervical and thoracic spinal manipulation on blood pressure in normotensive male patients.

## **1.2. AIM OF THE STUDY**

The aim of this investigation was to determine the effect of spinal manipulations on various spinal areas on blood pressure, as well as a combination of manipulations of the thoracic and cervical spine having a combined lowering effect on blood pressure as well as a cumulative effect on blood pressure in case-controlled normotensive males.

### 1.3. OBJECTIVES OF THE STUDY

The **first objective** was to determine the effect of sham laser on blood pressure in normotensive males. Blood pressure was measured five, 15 and 30 minutes post sham laser treatment.

Hypothesis One:

There would be no change in the blood pressure of the normotensive males with the sham cervical intervention.

The **second objective** was to determine the effect of cervical manipulation on blood pressure in normotensive males. Blood pressure was measured five, 15 and 30 minutes post cervical manipulation.

Hypothesis Two:

There would be no change in the blood pressure of the normotensive males with the active intervention (cervical manipulation).

The **third objective** was to determine the effect of thoracic manipulation on blood pressure in normotensive males. Blood pressure was measured five, 15 and 30 minutes post thoracic manipulation.

Hypothesis Three:

There would be no change in the blood pressure of the normotensive males with the active intervention (thoracic manipulation).

The **fourth objective** was to determine the effect of a combination of a cervical manipulation and thoracic manipulation on blood pressure. Blood pressure was measured five, 15 and 30 minutes post manipulation.

Hypothesis Four:

There would be no change in the blood pressure of the normotensive males with the active intervention (cervical and thoracic manipulation).

The **fifth objective** was to determine the effect on baseline (basal) blood pressure, following each day of intervention, which was measured at the start of every day of intervention.

Hypothesis Five:

There would be no change in the basal blood pressure of the normotensive males with the active interventions (sham laser, cervical and thoracic manipulation).

#### **1.4. RATIONALE**

Cervical spine manipulations alone and thoracic spine manipulations alone have been shown to decrease blood pressure significantly. The effects of a combination of cervical and thoracic spine adjustments together have thus far not been investigated (Sato, 1989; Slosberg, 1998; Murphy, 1999; Pickar, 2002; Bakris *et al.*, 2007).

It is postulated that a combination of cervical and thoracic manipulations will have a synergistic effect on the reduction of blood pressure. This may indicate that spinal manipulative therapy on different regions of the spine can have synergistic effects on the autonomic nervous system.

The results of the study will help to add to the existing body of literature on the somatovisceral effects of manipulation on blood pressure, should the findings indicate a significant decrease with a combination of a cervical and thoracic spinal manipulation, which might result in further studies on hypertensive patients to determine whether chiropractic is a form of treatment for hypertension.



## **1.5. LIMITATIONS OF THE STUDY**

Subjects participating within the study were asked to refrain from certain factors that could affect blood pressure, such as caffeine found in tea, coffee and energy drinks. Exercise and factors which the subjects could have perceived as stress would have caused an increase in blood pressure. It was therefore requested that subjects limit the effect of these stressors on the cardiovascular system, however the researcher had to rely on subject compliance as well as the honesty and openness of the subjects in terms of ensuring these criteria were met. A relaxation period of 30 minutes prior to treatment may have also aided to eliminate some of these factors.

## **CHAPTER 2**

### **REVIEW OF THE RELATED LITERATURE**

#### **2.1. INTRODUCTION**

This chapter presents the literature regarding Chiropractic and the effects of spinal manipulation and subluxation. In addition to the concepts of hypotension, hypertension and their neurological regulation will be discussed, as well as the connection between subluxation, manipulation and its impact on hypotension and hypertension.

#### **2.2. CHIROPRACTIC**

Chiropractic is a manual therapy, which belongs to the complementary alternative therapies (CAT) or complementary alternative medicines (CAM) (Haldeman, 2005; WHO, 2005). In this context the World Health Organisation defines Chiropractic as a health care profession that concerns itself with providing the most popular form of manual therapy and is thus concerned with the diagnosis, treatment and prevention of conditions related to the neuromuscular and skeletal systems thereby limiting the effects of these conditions and improving a patient's health and quality of life (WHO, 2005). In congruence with this the World Federation of Chiropractic (WFC) (2009) mandates a Chiropractor to be a practitioner that is responsible for dealing with the health needs of the public. This is achieved through the development of a chiropractor as a primary health care practitioner who pays particular attention to the relationship between the structural and neurological aspects of the body in health and disease; and is able to assess, diagnose, treat, manage / co-manage / refer a patient as is necessary in the best interests of the patient's wellbeing (The Councils on Chiropractic Education International (CCEI), 2004; WFC, 2009). It is therefore not unexpected that the Chiropractic Association of South Africa defines Chiropractic in the South African context as a profession that is responsible for

“the diagnosis, treatment and prevention of mechanical disorders of the musculoskeletal system and the effects of these disorders on the function of the nervous system and general health” and that the Allied Health Professions Council mandates, for purposes of registration in South Africa, that Chiropractors are required to assess and diagnose patients in order to determine any physical defect or illness and thus treat and prevent further defect or illness to the spine, pelvis and general neuromusculoskeletal structure (Chiropractic Association of South Africa (CASA), 2009; Act 63 of 1982 (as amended)).

### **2.2.1. MANIPULATION**

Notwithstanding these umbrella definitions as pertinent to the legislative control and professional management of the Chiropractic profession, the most distinguishing feature of the Chiropractic profession in terms of the interaction with patients is manipulation that is utilised in the treatment of the patients. Manipulation is defined as a load that is delivered by hand, to specific tissues (usually a short lever bony prominence) with therapeutic intent. The load is usually of a high velocity, low amplitude, single frequency, to a specific anatomic location, with a specified direction of force and of short impulse duration (Bergmann, Peterson and Lawrence, 1993; Kaptchuk and Eisenberg, 1998; Meeker and Haldeman, 2002). The therapeutic intent is achieved by the resultant movement of the vertebral joints beyond the clinical physiological range (Vernon and Mrozek, 2005) or elastic barrier (Sandoz, 1976a) without disrupting the joints anatomical integrity. On a mechanical level, the result is an increase in range of motion, and a release of synovial gasses causing a cracking sound (Kaptchuk and Eisenberg, 1998; Meeker and Haldeman, 2002), whereas on a neurological physiological level, changes within associated sclerotomal structures (somatovisceral or somatosomatic reflexes) are also seen (Pottenger, 1931; Pottenger and Ussher, 1933; Pottenger, 1953; Nansel, Waldorf, Cooperstein, 1993; Herzog *et al.*, 1999; Hopkins and Ingersoll, 2000; Leach, 2004; Hillermann, 2005).

These effects are thought to be mediated through the nervous system which is responsible for regulating set sclerotomal levels within the domain of a nerve (Bergmann *et al.*, 1993). Thus with manipulation the increase in neurological activity

(neurological facilitation) that results from this procedure, is thought to correct physiological changes in related sclerotomal structures (Bergmann *et al.*, 1993; Herzog *et al.*, 1999; Pickar, 2002). These physiological changes are either as a result of inhibited activity or stimulatory activity (Pickar, 2002).

It therefore stands to reason that manipulation could have an effect on blood pressure, but before the constructs behind this clinical effect is discussed, it is important to define and contextualise blood pressure.

### **2.3. CARDIOVASCULAR NEUROANATOMY AND PHYSIOLOGY OF BLOOD PRESSURE**

The heart muscle maintains an autonomic rhythm due to specialized muscle fibres which are capable of self excitation; these fibres are connected within a bundle and are customized for rapid conduction. The rhythm originates within the body of fibres known as the sinoatrial node in the right atrium of the heart. Impulses from this node are conducted to the atrio-ventricular node; from here the impulses are conducted to the ventricles via the Purkinje fibres. Co-ordination of this system is via the parasympathetic and sympathetic innervation to the heart (Masarsky and Todres-Masarsky, 2001; Dimmick, Young and Newell, 2006), which stems principally from the cervical spine region.

Specifically the parasympathetic innervation to the heart originates from the cardiac branches of the vagus nerves. The vagus nerve originates from the nucleus ambiguus and the dorsal motor vagal nucleus and has contributing fibres from the hypothalamus (Masarsky and Todres-Masarsky, 2001; Dimmick *et al.*, 2006) before it exits the skull through the jugular foramen. In so doing it runs closely alongside the carotid artery (left and right) / brachiocephalic trunk (right) on its way to the base of the arch of the aorta. At the bifurcation of the brachiocephalic trunk the vagus gives rise to a branch known as the recurrent laryngeal, before proceeding down the brachiocephalic trunk to the base of the aorta, where it forms part of the aortic plexus before it crosses over the pericardium (over the right atrium) on its path to the diaphragmatic opening where the aorta passes into the abdomen. On the left the vagus passes along the carotid to its origin, before giving rise to the left recurrent laryngeal branch (that hooks around the arch of the aorta), before

contributing to the aortic plexus and then passing posteriorly over the pericardium associated with the left ventricle on its way to meet the right vagus nerve at the arcuate ligament of the diaphragm (Tobias, Arnold and Allen, 1988; Cramer and Darby, 1995; Abrahams and Hutchings, 1998; Moore and Dalley 1999; Boon, Colledge, Walker and Hunter, 2006; Drake, Vogl, Mitchell, Tibbitts and Richardson, 2008 ).

Through its connections in the aortic plexus, the vagus nerve allows for vagal stimulation of the sinoatrial node, which slows the rhythm of the sinus node and thus decreases the heart rate (Guyton and Hall, 1997).

On the converse the sympathetic innervation to the heart originates from fibres within the spinal cord (via the lateral horn). These fibres pass to the upper thoracic portion of the spinal cord, before passing out the vertebral canal as part of the spinal nerve. Once outside of the intervertebral foramen, the sympathetic fibres leave the spinal nerve in the form of the white ramus communicants and enter the sympathetic chain. Once at the level of the sympathetic chain, the fibres can either (Tobias *et al.*, 1988; Cramer and Darby, 1995; Abrahams and Hutchings, 1998; Moore and Dalley 1999; Boon *et al.*, 2006; Drake *et al.*, 2008):

- Synapse at the level of entry and pass out of the chain via the grey rami communicants to re-enter the spinal nerve at that level.
- Or they can pass to the superior or inferior ganglia within the sympathetic chain, before they synapse at that location and then pass out via a grey ramus communicants in order to enter the spinal nerve at that level. The superior cervical ganglion (stellate ganglion), which is located alongside the second and third cervical vertebrae, receives its sympathetic input from the first five thoracic spinal nerves, as a result of sympathetic fibres travelling up the sympathetic chain before synapsing within the cervical ganglia.
- Or they can pass through the ganglion of entry and synapse in a terminal end organ (such as the adrenal gland), where the post synaptic neuron is the tissue of the terminal organ. The sympathetic transmission of information is passed along the greater, lesser and least splanchnic nerves from sympathetic chain to the organs. In this system the heart receives its sympathetic transmission via the greater

splanchnic nerves, which pass from the sympathetic chain (cervical through upper thoracic ganglia) inferior and anteriorly along the vasculature (normally along the carotid arteries, the brachiocephalic trunk and aorta) to the heart's aortic plexuses (Guyton and Hall, 1997). Through its transmission the sympathetic stimulation has been noted to increase the heart rate (Masarsky *et al.*, 2001; Dimmick *et al.*, 2006).

Both the sympathetic and parasympathetic systems may be influenced by a number of factors internal to the body, known endogenous factors, these include but are not limited to the renin-angiotension system and its feedback system with aldosterone, the baroreceptor reflex system, and cardiovascular factors such as cardiac output, total peripheral resistance and total blood volume (Kaplan, 1986; Seedat, 1989; Fox, 1999).

According to Sharma (1992), the physiological mechanisms that maintain normal blood pressure are those related to, influenced by and influence the autonomic nervous system responses, the hormonal response systems, the capillary shift mechanism and the kidney and fluid balance mechanisms:

### **2.3.1. THE INFLUENCES OF THE AUTONOMIC NERVOUS SYSTEM ON BLOOD PRESSURE**

In this context, the autonomic nervous system is the most rapid blood pressure regulator; it receives continuous information from stretch receptors known as baroreceptors, located in the walls of the large arteries (Guyton and Hall, 1997). These baroreceptors are situated principally in the carotid sinus (internal carotid arteries) and the aortic arch, their information is relayed to the vasomotor centre via the glossopharyngeal nerve to the brainstem. The aortic baroreceptors usually result in the brainstem activating the vagus nerve, whereas the baroreceptors of the carotid sinus usually result in the brainstem activating the sympathetic nervous system. Therefore both systems are essential in maintaining normal constant arterial blood pressure during postural changes (Cramer and Darby, 1995; Moore and Dalley 1999). Failure of these baroreceptors can cause hypotension which can result in syncope and vertigo (Sharma, 1992; Masarsky and Todres-Masarsky, 2001), where a decrease in blood pressure will normally cause the sympathetic nervous system to

be activated and thus to increase the hearts contractility as well as cause vasoconstriction of the peripheral vessels to facilitate a normalised blood pressure. This is achieved in that the sympathetic chain ganglia distribute vasoconstrictor fibres to all blood vessels allowing a vasoconstrictor tone to be maintained even in the resting position. The opposite is true of hypertension; however the vagal system has a limited ability to respond to consistent high arterial blood pressure as tissue fatigue as well as processes of hysteresis and creep causing localised damage to the heart and vessel walls, predisposing the patient to further disease and complications (Boon *et al.*, 2006).

### **2.3.2. THE INFLUENCES OF THE HORMONAL RESPONSE SYSTEM ON BLOOD PRESSURE**

Hormonal control of blood pressure can act in different ways to affect blood pressure; it can cause vasodilation, vasoconstriction, as well as altering the blood volume (Sharma, 1992). These mechanisms can therefore increase or decrease blood pressure. In the context of hypertension, adrenaline and noradrenalin, which are secreted due to sympathetic nervous system stimulation of the adrenal glands via the splanchnic nerves, are also known to aggravate high blood pressure or increase normal blood pressure. Hormonal responses from the adrenal gland are normally slower than the immediate neurological responses affected through the autonomic nervous system, but acts more quickly than the capillary fluid shifting mechanism to enhance the rapid vasoconstriction and rapid increase in cardiac output, initiated by the autonomic nervous system (Cramer and Darby, 1995).

### **2.3.3. THE EFFECT OF FLUID BALANCE ON BLOOD PRESSURE**

The capillary fluid shift mechanism is the exchange of fluid between the capillary membrane and the interstitial fluid. The movement of this fluid is controlled by interstitial fluid pressure, capillary blood pressure and the plasma's osmotic pressure. A decrease in blood pressure will result in fluid moving from the interstitial space into the circulation to increase blood volume and thus blood pressure (Sharma, 1992). This mechanism is however much slower than the autonomic response mechanism and is responsible for fine tuning homeostasis once roughly achieved through the autonomic system (Guyton and Hall, 1997).

### **2.3.4. KIDNEYS CONTROL OF BLOOD PRESSURE IN THE LONG TERM**

Long term control of blood pressure, is via the kidneys by increasing or decreasing the blood volume via the rennin-angiotensin system, which facilitates peripheral resistance and sodium homeostasis (Guyton and Hall, 1997, Beers and Berkow, 1999).

Specifically the juxtaglomerular cells of the kidney release rennin, Angiotensin I is transformed from plasma angiotensin by rennin. Angiotensin II is converted from Angiotensin I via Angiotensin Converting Enzyme (ACE). Angiotensin II is accountable for increasing peripheral resistance by directly acting on smooth muscle contraction, as well as increasing blood volume via stimulating secretions of aldosterone (formed in the adrenal cortex), and increasing distal tubular sodium reabsorption (Guyton and Hall, 1997). Thus Angiotensin II increases blood pressure and could be a cause of hypertension if pathologically produced. The kidney does however produce various substances which counterbalance the effects of angiotensin; these antihypertensive substances are prostaglandins and nitric oxide. When blood volume is reduced, sodium is reabsorbed thereby increasing blood volume. When the blood volume is too great the heart atria excrete natriuretic peptide which inhibits sodium reabsorption and thus decreasing blood volume (Beers and Berkow, 1999; Kumar, Contran and Robbins, 2003; Boon *et al.*, 2006). Blood volume affects cardiac output; which is dependent on the sodium homeostasis within



the body. Therefore normal vascular tone occurs when there is a balance between the vasoconstricting hormones (Angiotensin II and catecholamines) and vasodilating hormones (kinins; prostaglandins and nitric oxide) (Boon *et al.*, 2006).

As a result of the above discussion it can be seen that neural (autonomic nervous system) and hormonal influences determine the level of total peripheral resistance, cardiac output and blood volume. The auto regulation of these three parameters occurs as a protective mechanism, in order to ensure that perfusion to the body's tissues are not compromised (Kumar, Contran and Robbins, 2003).

## **2.4. THE PHYSIOLOGY AND PATHOPHYSIOLOGY OF BLOOD PRESSURE**

To understand the concepts related to blood pressure fully it is important to review the normal physiology of the cardiovascular system, with particular reference to the heart and how blood passes through the heart. Therefore the next section will deal with this briefly before normal blood pressures and reasons for abnormal blood pressures are presented.

### **2.4.1. NORMAL PHYSIOLOGY**

The heart is a muscular organ, comprising of four chambers. The two upper chambers are known as the atria and two lower chambers are known as the ventricles. The ventricles are thicker than the atria as they have a heavier pumping requirement. The left ventricle has a thicker wall than the right ventricle as it has to pump the blood to all areas of the body except the lungs; the right ventricle pumps the blood to the lungs (Thibodeau, 1987; Tobias *et al.*, 1988; Guyton and Hall, 1997; Moore and Dalley, 1999).

Deoxygenated blood from the capillaries, venules and veins enter the right atrium via the superior and inferior venae cavae. Atrial contraction then pumps this deoxygenated blood to the right ventricle which in turn pumps it through the pulmonary artery to the lungs whereby the blood is oxygenated. This oxygenated blood enters the left atrium via the pulmonary veins, from here the oxygenated blood is pushed into the left ventricle via atrial contraction, the left ventricle then pumps this

oxygenated blood into the aorta to distribute to all areas of the body (Thibodeau, 1987; Tobias *et al.*, 1988; Guyton and Hall, 1997; Moore and Dalley, 1999).

The systolic reading in blood pressure is during ventricular contraction, this is when the pressure reading is at its highest, and the diastolic reading is during ventricle relaxation and atrial refilling, this is when the pressure reading is at its lowest (Guyton and Hall, 1997; Fox, 1999).

In order to make allowances for physiological changes within the cardiovascular system, diagnosing hypertension or hypotension requires three consecutive recordings of blood pressure over three consecutive days, preferably in the morning, if all readings of systolic and diastolic values are within the hypertension range, a diagnosis of hypertension could be made and conversely if all readings are lower than the normal range, then a diagnosis of hypotension needs to be considered (Beers and Berkow, 1999).

#### **2.4.2. PATHOPHYSIOLOGY**

Blood pressure is defined as the force per unit area exerted on the wall of a blood vessel and is expressed in millimetres mercury (mmHg). Thus in a normotensive individual the blood pressure should range between 120 and 140 mmHg systolic and 75 and 90 mmHg diastolic readings (Guyton and Hall, 1997; Fox, 1999; Kumar *et al.*, 2003).

When the blood pressure falls outside of these reading parameters, the patient is said to have hypertension if the systolic reading is above 140 mmHg and the diastolic reading is above 90 mmHg (Guyton and Hall, 1997). Conversely if the patient has a systolic reading below 100 mmHg and a diastolic reading below 60 mmHg, the patient is diagnosed as hypotensive or having hypotension (Rutan, Hermanson, Bild, Kittner, LaBaw and Tell, 1992).

**Table 2.1: Classification of Blood Pressure for Adults:**

Classification	Beers and Berkow (1999)		Holm, Cunningham, Bensadoun and Madsen (2006)	
	Systolic (mmHg)	Diastolic (mmHg)	Systolic (mmHg)	Diastolic (mmHg)
Hypotension	<100	<60	<100	<60
Optimal	< 120	<80	< 120	<80
Normal	<130	<85	120 – 139	80 – 89
High – Normal	130 – 139	85 – 89		
Stage 1 Hypertension (mild)	140 – 159	90 – 99	140 – 159	90 – 99
Stage 2 Hypertension (moderate)	160 – 179	100 – 109	>160	> 100
Stage 3 Hypertension (severe)	>180	>110		

## **2.5. CAUSES OF HYPOTENSION**

Hypotension is considered to be the failure of the normal blood pressure compensatory mechanisms resulting in symptomatic postural hypotension. Factors that can cause or aggravate the condition are (Rutan *et al.*, 1992; Boon *et al.*, 2006):

- Postural hypotension
- Hypovolemia/decreased blood volume (resulting from excessive diuretic therapy)
- Sympathetic degeneration (Aging, Parkinson's disease and Diabetes Mellitus)
- Drug therapy (vasodilator's and anti-depressants)

## 2.6. CAUSES OF HYPERTENSION

The causes of hypertension have been classified (Boon *et al.*, 2006) as:

- Primary hypertension
- Secondary hypertension

In terms of primary hypertension, idiopathic / essential hypertension is the most common and contributes 90% to 95% of all hypertensive patients. The majority of the remaining 5% - 10% fall into the secondary hypertension category also known as 'benign hypertensive' category which is secondary to renal disease / narrowing of the renal artery (renovascular hypertension); although malignant hypertension can occur in up to 5% of hypertensive patients. Infrequently, hypertension could be secondary to diseases of the adrenal glands, such as primary aldosteronism, pheochromocytoma and Cushing's syndrome (Kumar *et al.*, 2003; Boon *et al.*, 2006).

**Table 2.2: A summary of causes of Primary and Secondary Hypertension**

**(Kumar *et al.*, 2003; Boon *et al.*, 2006):**

<ul style="list-style-type: none"><li>• <b>Renal</b><ul style="list-style-type: none"><li>➤ Acute glomerulonephritis</li><li>➤ Chronic renal disease</li><li>➤ Polycystic disease</li><li>➤ Renal artery stenosis</li><li>➤ Renal vasculitis</li><li>➤ Renin-producing tumours</li></ul></li></ul>
<ul style="list-style-type: none"><li>• <b>Endocrine</b><ul style="list-style-type: none"><li>➤ Adrenocortical hyperfunction<ul style="list-style-type: none"><li>○ Cushing's syndrome</li><li>○ Primary aldosteronism</li><li>○ Congenital adrenal hyperplasia</li></ul></li><li>➤ Exogenous hormones<ul style="list-style-type: none"><li>○ Glucocorticoids</li><li>○ Estrogen</li><li>○ Sympathomimetics</li><li>○ Monoamine oxidase inhibitors</li></ul></li><li>➤ Pheochromocytoma</li><li>➤ Acromegaly</li><li>➤ Hypothyroidism</li><li>➤ Hyperthyroidism</li><li>➤ Pregnancy</li></ul></li></ul>
<ul style="list-style-type: none"><li>• <b>Cardiovascular</b><ul style="list-style-type: none"><li>➤ Coarctation of aorta</li><li>➤ Polyarteritis nodosa</li><li>➤ Increased intravascular volume</li><li>➤ Increased cardiac output</li><li>➤ Aortic rigidity</li></ul></li></ul>
<ul style="list-style-type: none"><li>• <b>Neurologic</b><ul style="list-style-type: none"><li>➤ Psychogenic</li><li>➤ Raised intracranial pressure</li><li>➤ Sleep apnea</li><li>➤ Acute stress</li></ul></li></ul>
<ul style="list-style-type: none"><li>• <b>Exogenous Factors</b><ul style="list-style-type: none"><li>➤ Stress</li><li>➤ Drugs</li><li>➤ Smoking</li><li>➤ Alcohol</li><li>➤ Obesity</li><li>➤ Physical inactivity</li><li>➤ Nutrition (diet high in salt)</li></ul></li></ul>

With respect to the secondary hypertension diagnoses, it has been noted that they have been found to modify the genetic determinants of hypertension (Kumar *et al.*, 2003).

Although both hypertension and hypotension are clinically significant, it is hypertension that is most often associated with clinical sequelae which are associated with increased morbidity and mortality (Boon *et al.*, 2006) therefore this study will focus on hypertension.

## **2.7. MAGNITUDE OF THE HYPERTENSION PROBLEM**

Globally, hypertension is a dilemma in developed and developing countries accounting for the pandemic of cardiovascular disease (Seedat, Milne, Opie, Pinkney-Atkinson, Rayner, Veriava, Croasdale, 2006). It has been reported that 15% of the urban population of South Africa are considered hypertensive (Steyn, Fourie, Lombard, Katzenellenbogen, Bourne and Jooste, 1996; Jamison, Feachmen, Bos and Makgoba, Hofman and Rogo, 2006); with 2% of this figure constituting females affected with hypertension and the remaining 13%, representing the male contribution to this figure (Steyn *et al.*, 1996; Jamison *et al.*, 2006; Steyn, 2006). This compares favourably with the slightly higher reported findings of the rural population of South Africa where 18% are affected by hypertension, 2% representing the female contribution and the remaining 16% the male contribution (Metcalf, Hoffman, Steyn, Katzenellenbogen and Fourie, 1996, Jamison *et al.*, 2006).

As a result of these findings it was estimated that in Africa more than 20 million people are affected with hypertension, particularly those in the urban areas (Bam and Yako, 1984; Amoah and Kallen, 2000). As a result of these estimates, the prevalence of hypertension in adults aged 25-64 years seems to range from 25% to 35% amongst the African studies completed (Bam and Yako, 1984; Amoah *et al.*, 2000). This concurs with the findings of Jamison *et al.*, (2006), who noted that 8% to 25 % of the adult population in Sub-Saharan Africa are affected by hypertension. In addition the results concur with the Heart and Stroke Foundation of South Africa ([www.healthlinkorg.co.za](http://www.healthlinkorg.co.za), 2009), who estimate that 1 in 4 (10% to 25%) South African citizens between the ages of 15 to 64 years suffer from hypertension; with up to two thirds of these people being unaware of their raised blood pressure status.

This high reporting of hypertension in the South African and sub-Saharan contexts seems to be representative of the findings that malignant hypertension occurs more frequently in Black African people than any other population groups (Milne, Veriava, James, Isaacson,

1998). Furthermore, it has been reported that the Indian community have a higher incidence of Type II Diabetes Mellitus than any other population group, which has been linked to the co-morbid conditions of obesity and hypertension (Seedat *et al.*, 2006).

These population specific presentations of hypertension are further compounded by diseases that cross all ethnic groups. One such example is that of the high incidence and prevalence of HIV/AIDS (Mukadi, Maher and Harries, 2001; Nelson Mandela Foundation, 2008), as it has been shown that the prolonged use of antiretroviral drugs used in the treatment of HIV/AIDS can cause systolic hypertension (Seedat *et al.*, 2006).

Therefore in real terms, hypertension and the associated cardiovascular disease sequelae are the second highest cause of mortality in South Africa, accounting for 7.4% of all deaths (Seedat *et al.*, 2006). As a result of this high incidence and prevalence of hypertension, its sequelae and the hypertension linked co-morbid diseases contributing to hypertension; these have been extensively investigated, in order to enable more effective and efficient control and / or prevention of hypertension and thus prevent at least 250 000 deaths per annum in South Africa (Bam and Yako, 1998; Amoah *et al.*, 2000). In addition these investigations have been aimed at reducing the costs of health care associated with the assessment, treatment and management of cardiovascular disease, which was noted in 1991 to cost between R4 billion and R5 billion. At that stage this amount constituted around 7.5% of direct health care spending related to hypertension and related diseases as well as sequelae in South Africa (Seedat *et al.*, 2006).

## **2.8. SEQUELAE FROM AND COMORBID CONDITIONS OF HYPERTENSION**

### **2.8.1. SEQUELAE**

Hypertension is the most common cause of cardiovascular disease (specifically hypertensive heart disease) in Africa (Jamison *et al.*, 2006), often resulting in cerebrovascular accidents, ischemic heart disease, coronary heart disease and myocardial infarction (Bradshaw, Groenewald, Laubscher, Nannan, Nojilana, Norman, Pieterse, and Schneider, 2003; Kumar *et al.*, 2003). Specifically in Black African patients, hypertension has been commonly linked to the following sequelae: congestive cardiac failure, left ventricular hypertrophy and cerebrovascular accidents (Jamison *et al.*, 2006). Furthermore, hypertension may lead to cardiac hypertrophy, heart failure due to hypertensive heart disease, aortic dissection and renal failure in all ethnic groups, with each of these sequelae increasing in likelihood with increased age that the patient has had hypertension (Kumar *et al.*, 2003).

### **2.8.2. COMORBID CONDITIONS**

In summary and from the above discussions on hypertension, its co-morbid conditions as well as its sequelae, it can be argued that a reduction in blood pressure can reduce the risk of ischemic heart disease, heart failure and cerebrovascular accidents (Kumar *et al.*, 2003). This would therefore result in the decreased morbidity and mortality as well as decrease the costs expended on health care requirements associated with hypertension and hypertensive disease.

Co-morbid conditions occurring with hypertension are obesity, Diabetes Mellitus, metabolic syndrome, depression, dyslipidemia and HIV/AIDS (Holm, Cunningham, Bensadoun and Madsen, 2006; Evangelista and McLaughlin, 2008). In the South African context Steyn (2006) indicates that obesity in South Africa has a high incidence, occurring co-morbidly with hypertension. Abdominal obesity links insulin resistance and dyslipidemia and hypertension through complex endocrine pathways resulting in metabolic syndrome (Evangelista and McLaughlin, 2008). Therefore the



high incidence of obesity in South Africa coincides with the co-morbid conditions of Diabetes Mellitus II and hypertension.

It therefore stands to reason that if manipulation could have an effect on blood pressure, it too would contribute to a decrease in morbidity and mortality as well as decrease the costs expended on health care requirements associated with hypertension and hypertensive disease. In order to understand the hypothesised effects of manipulation on hypertension, the links between the spine and the cardiovascular system need to be discussed in detail and what follows is an outline of the neuroanatomy that links the spine to the cardiovascular system.

## **2.9. THE VERTEBRAL SUBLUXATION COMPLEX (SPINAL LESION) AND ITS NEUROANATOMY**

A motion segment consists of two adjacent vertebrae (or bones) and the intervening soft tissue structures that make the motion segment a functional unit within the spinal column (or an extremity kinematic chain) (Gatterman, 1990; Bergmann, 1992; Kirkaldy-Willis and Burton, 1992; Bergmann *et al.*, 1993; Bergmann, 1995).

Within a spinal motion segment, its function is determined by mechanical, chemical, hormonal and neurological factors (Sandoz, 1976a; Sandoz, 1976b; Korr, 1979a; Dvorak, 1985; Wyke, 1985; Patterson and Steinmetz, 1986; Gatterman, 1990; Gatterman and Goe, 1990; Mense, 1991; Leach, 2004):

### **2.9.1. MECHANICAL FACTORS**

Mechanical factors are normally principally determined by the size, shape and orientation of the facet joints and the intervertebral disc (collectively known as the three joint complex (Kirkaldy-Willis and Burton, 1992; Cramer and Darby, 1995), the postural muscles, the minor mover muscles and the major mover muscles (Cramer and Darby, 1995) as well as the ligaments that restrain/break or facilitate movement (Bogduk and Twomey, 1987; Cramer and Darby, 1995). In a pathological situation the mechanical factors that could hamper movement include, but may not be limited

to adhesion formation (Vernon and Mrozek, 2005), meniscoid entrapment (Leach, 2004), disc deformation or nuclear fragment formation (Leach, 2004; Haldeman, 2005), segmental hypermobility associated with segmental hypomobility (Gillet, 1974; Schafer and Faye, 1990; Bergmann, 1992; Bergmann, 1993; Bergmann, 1995). Furthermore mechanical dysfunction may result from joint surfaces becoming incongruent with each other as a result of asymmetrical formation or an asymmetrical force that is imparted into the motion segment (Schafer and Faye, 1990; Bergmann, 1992; Bergmann *et al.*, 1993; Bergmann, 1995; Haldeman, 2005).

### **2.9.2. CHEMICAL FACTORS**

Chemical factors include the requirements of the motion segment in terms of nutrition to that segment (e.g. glucose and oxygen) and the removal of waste products (e.g. urea and carbon dioxide). These factors are usually kept in a homoeostatic balance by the constantly fluid vascular system (Cramer and Darby, 1995), the movement available within the motion segment (as it facilitates nutrition of structures that are not vascularised) (Guyton and Hall, 1997) and the diffusion gradients created by the use and production of chemical substances (Guyton and Hall, 1997; Leach, 2004). The obstruction of supply of nutrition or the removal of waste, results in either a depletion of chemicals or a build up of chemicals. These two processes singularly or in combination are usually the causative agents for the development of an inflammatory nidus (Rippey, 2001). This inflammatory process is then responsible for activating the neurological system, leading to pain (Dvorak, 1985; Gatterman and Goe, 1990; Mense, 1991; Leach, 2004), arthrogenic muscle inhibition or muscular spasm (Nansel, Waldorf and Cooperstein, 1993; Herzog *et al.*, 1999; Hopkins and Ingersoll, 2000; Hopkins, Ingersoll, Krause, Edwards and Cordova, 2001; Hillermann, 2005) and in some instances referred pain (Sandoz, 1976a; Sandoz, 1976b; Simons and Travell, 1999; Chaitow and DeLany, 2000). On occasion it has also been reported that the patient has had visceral symptomatology associated with this process (Murphy, 2002; Leboeuf-Yde, Pedersen, Bryner, Cosman, Hayek, Meeker, Shaik, Terrazas, Tucker and Welch, 2005).

### **2.9.3. HORMONAL FACTORS**

Hormonal parameters that affect the functioning of the normal motion segment, are those related to the normal circadian rhythms (e.g. melatonin), normal metabolic function (e.g. thyroid function), stress and anxiety (e.g. adrenalin and noradrenalin; cortisol) and pain control (e.g. serotonin, endorphins and enkephalins). It has been suggested that immune control through hormonal means may also affect the functioning of a motion segment, however the information is limited. In this context the various hormonal systems have a homeostatic norm or balance; as an example melatonin is regulated by daily exposure to sunlight, this exposure is responsible for setting and regulating melatonin function through the pineal gland resulting in normal sleep patterns. These sleep patterns allow for the person to have optimal relaxation and recuperation and therefore optimal functioning for all body systems including the motion segment. Lack of exposure to sunlight and therefore a lack of normal hormonal regulation is thought to result in decreased recuperation. Thus increased susceptibility to stress (both mechanical and physiological) thereby predisposing to malfunction or dysfunction at both mechanical and physiological levels within the body and within the motion segment (Leach, 2004).

### **2.9.4. NEUROLOGICAL FACTORS**

The functioning of the motion segment is however principally controlled by the pulley and level system that is found between the muscular and bony structures within the motion segment. The co-ordination of this pulley and lever is controlled by the nervous system. In order to achieve this, the neurological system within the motion segment has (Denslow, 1975):

#### **2.9.4.1. INTRA-ARTICULAR RECEPTORS**

These receptors are specialized cells that change their properties in response to specific stimuli of various types. Receptors that respond to physical or mechanical stimuli are termed mechanoreceptors. Mechanoreceptors act to convert energy from one form, for example tension, into a specific nerve signal.

Receptors that transduce information about the relationship between body segments are proprioceptors. Joint receptors are mechanoreceptors and can also act as proprioceptors. Therefore, joint receptors have two major functions. They provide position sense or information about the relative configuration of body segments, and they initiate protective reflex mechanisms that protect and help stabilize the joint (Hopkins and Ingersoll, 2000).

The four different types of mechanoreceptors in the motion segment are (Sandoz, 1976a; Sandoz, 1976b; Wyke, 1981; Freiwald, Reuter and Engelhardt, 1999):

- Wyke Type 1 - Ruffini bodies.
- Wyke Type 2 - Vater-Pacini bodies.
- Wyke Type 3 - Golgi organelles.
- Wyke Type 4 - Free nerve endings.

#### **2.9.4.1.1. TYPE 1- RUFFINI BODIES**

These are the second most frequent receptor type in any joint. They are arranged in clusters of between 3 to 6 bodies (Freiwald *et al.*, 1999). Ruffini bodies are slow-adapting receptors that have been identified in the joint capsules. These receptors have a very low threshold and they respond to very slight changes in ligament tension and capsular pressure (Hopkins and Ingersoll, 2000).

#### **2.9.4.1.2. TYPE 2- VATER-PACINI BODIES**

This receptor type has been found in the fibrous joint capsule and ligament insertions. The function of the Vater-Pacini bodies is the rapid uptake and transport of information from the afferent-supply tissues. They are inactive in immobile joints and like-wise when the facet joint is moved at a constant velocity, they become active in the case of accelerations or decelerations. Vater-Pacini sensors have a low mechanical stimulation threshold, adapt rapidly and they act as dynamic mechanoreceptors (Freiwald *et al.*, 1999).

#### **2.9.4.1.3. TYPE 3- GOLGI ORGANELLAE**

The Golgi tendon organs in the muscle-tendon bond the muscles overlying the motion segment. Golgi organs are similar to the Ruffini bodies and as long as they are localized in the joints, some authors do not differentiate between them. These receptors help provide information about joint position (Freiwald *et al.*, 1999).

#### **2.9.4.1.4. TYPE 4- FREE NERVE ENDINGS**

Free nerve endings are non-specialized, non-encapsulated, unmyelinated (or finely myelinated) receptors. They function as pain receptors and probably provide a crude awareness of initial joint movement. These nerve endings are found throughout the joint tissue and are involved with any motion segment dysfunction (Hopkins and Ingersoll, 2000).

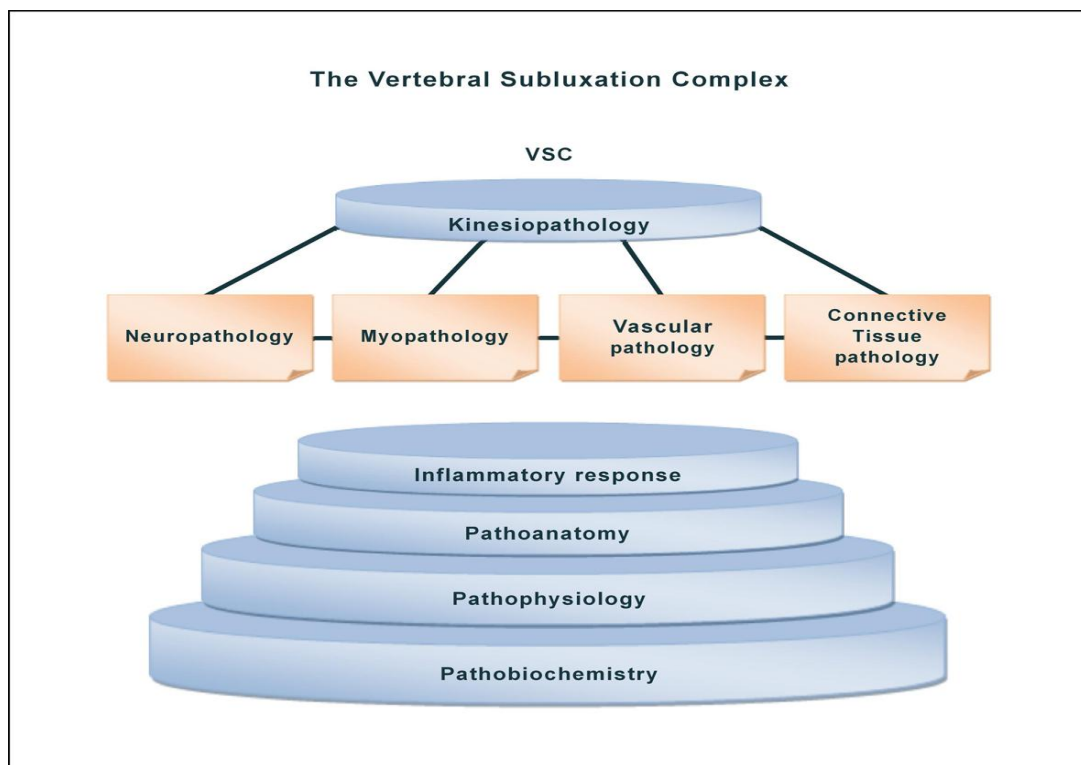
#### **2.9.4.2. EXTRA-ARTICULAR RECEPTORS**

Muscle spindles are extra-articular receptors that are located within skeletal muscle and run parallel to the skeletal muscle fibres. The fibres in the centre of the muscle spindle are known as intrafusal fibres, whilst the skeletal muscle fibres surrounding the muscle spindle are known as the extrafusal fibres. The intrafusal fibres are supplied by Type I alpha nerve fibres, which stimulate motor neurons (Solomon, Schmidt and Adragna, 1990). The afferents of the muscle spindle are type II nerve fibres that synapse with spinal ganglia cells. Type II afferents also stimulate the motor neurons but their effect is multi-segmental. They have a promoting effect on the agonist and an inhibitory effect on the antagonist muscle (Freiwald *et al.*, 1999).

Stimulation of the muscle spindle due to stretching of that muscle causes a reflex contraction. This reflex is known as the stretch reflex and brings the tension in the muscle back to normal (Solomon *et al.*, 1990). Mechanoreceptors and central nervous system mediation can also stimulate the muscle spindle fibres. Hence,

muscle spindles either directly or indirectly influence the motor neurons of the agonistic and antagonistic musculature (Freiwald *et al.*, 1999).

A vertebral subluxation, is defined as a motion segment that presents with kinesiopathology, neuropathophysiology, myopathology as well as histological and biochemical changes (signs and symptoms) that manifest locally or at a site distal to the subluxation complex (Denslow, 1975; Gatterman, 1990; Haldeman, 2005).



**Figure 2.1: Vertebral Subluxation Complex (Gatterman, 1995).**

## **2.10. THE POSSIBLE NEUROANATOMICAL LINK BETWEEN THE SUBLUXATION AND HYPERTENSION**

### **2.10.1. NEURAL PATHWAYS**

#### **2.10.1.1. THE AFFERENT PATHWAY TO THE SPINAL CORD**

Most receptors are specialized endings to sensory nerve fibres (Hopkins and Ingersoll, 2000). Sensory nerve cells contain a cell body located in a dorsal root ganglion very close to the spinal cord. The cell body then projects through the dorsal horn of the spinal cord where it can make connections with several different types of neurons (Haldeman, 1992; Haldeman, 2005).

In terms of the afferent information from the cardiovascular system, these would principally enter via the baroreceptors or pressure transducers within the walls of the carotid arteries, whereas the information from the facet joint would enter the spinal cord through the afferent Wyke receptors I to III (Wyke, 1981). In each instance this incoming information would be relayed to one of several Rexed's laminae (Cramer and Darby, 1995) present in the dorsal horn and grey matter of the spinal cord. At this stage the information is then transferred to the appropriate interneuron(s) at the level of entry for transfer to the appropriate ascending tract(s) and relayed at that level to facilitate local reflex action (Leech, 1994).

#### **2.10.1.2. THE INTERNEURON<sup>a</sup>**

Once the sensory fibre enters the dorsal horn of the spinal cord, it usually branches to synapse on one or several interneurons. A single neuron usually receives information from designated neurons and then projects it to many different neurons with the purpose of facilitating local reflex responses at a particular level as well as adjacent levels while also transferring the

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<sup>a</sup> An interneuron is defined as a neuron receiving information from a neuron and transmitting it to one or more other neurons.

information to tracts connecting on to higher thalamic, cerebellar, cerebral and limbic centres. Thus most interneurons have axons that branch widely, ascending or descending over distances of 2-3 segments (Hopkins and Ingersoll, 2000).

Interneurons are the intermediates of pathways to alpha and gamma motor neurons (MN), to autonomic efferent neurons and to ascending pathways. They receive projections from sensory afferent fibres, descending fibres and other interneurons (Hopkins and Ingersoll, 2000). Processing of afferent input into the spinal cord is mediated by inputs from other parts of the periphery and from the brain (Haldeman, 1992). Therefore the net effect of all information arriving at the interneuron is expressed in the inhibitory or excitatory response of the MN pool (Hopkins and Ingersoll, 2000).

As was indicated in Section 2.4.1, (the normal physiology of blood pressure), the effect of the baroreceptor information can either be excitatory or inhibitory depending on which receptors the information was initially received from by the interneuron, thus resulting in an efferent response that is either facilitated or depressed at that spinal cord level. Similarly, the effect of stimulating the Wyke receptors can facilitate or depress the local reflex responses. Facilitation of the spinal cord segment is initiated by stimulation of the Wyke I to III receptors (as would be seen in manipulation) (Cramer and Darby, 1995; Leach, 2004) and depression of the spinal cord segment usually results from stimulation of the Wyke IV receptors (as is seen in arthrogenic muscle inhibition) (Suter, Herzog, Conway and Zang, 1994; Suter, MacMorland, Herzog and Bray, 1999; Hopkins and Ingersoll, 2000; Suter, MacMorland, Herzog and Bray, 2002).

It can thus be seen that should a spinal cord segment be facilitated it is potentially possible to enable a response in areas that were not the intended target of an expressed intervention. This has in the past led to the development of somatovisceral, viscerosomatic, viscerovisceral and somatosomatic theories (Leach, 2004). Some of these theories have greater



literature support (e.g. somatosomatic (Sandoz, 1976b; Kirkaldy-Willis and Burton, 1992; Simons and Travell, 1999; Chaitow and DeLany, 2000), than others which are less easily tested or observed (e.g. viscerovisceral). Notwithstanding these differences the recording of such events thought to be related to these theories has formed the basis of several investigations as some clinical changes that were perceived to be unrelated to the complaint under treatment and unrelated to the treatment intervention have either improved or worsened and were directly linked to the intervention by the patients (Leboeuf-Yde *et al.*, 2005); therefore seemingly supporting the existence of these theories. The next section therefore discussed these theories.

## **2.11. SOMATOVISCERAL EFFECTS OF SPINAL MANIPULATION**

Based on the perceived neurological links through the interneurons, it has been hypothesised and shown that if sufficient stimulation is imparted to a particular spinal level, this facilitation may cause inhibition or stimulation of the efferent neurons through the connection provided by the interneurons (Denslow and Cough 1941; Brown, Goodwin and Matthew, 1969; Eldred, Hutton and Smith, 1976; Poppele and Quick, 1981; Morgan, Prochazka and Proskue, 1984; Shambaugh, 1987; Edin and Vallbo, 1988).

In this manner it is thought that manipulation (particularly of the spine) stimulates the Wyke I to III receptors, which is a mechanical stimulus to the vertebral column (Korr, 1979a; Gatterman, 1995). It is further been postulated that if manipulation is delivered at the end of the passive range of motion within a motion segment (i.e. at the elastic barrier) into the paraphysiological range (without exceeding the barrier of anatomical limits); the degree of neurological stimulation of the receptors would be maximal (Sandoz, 1976b; Vernon and Mrozek, 2005) and therefore potentially result in maximal stimulation of the corresponding spinal segmental level and thus result in increased facilitation of that level (Guyton and Hall, 1997; Dixon, 2005).

Thus with maximal stimulatory input into the spinal cord segmental level from the sensory input arising out of the paraspinal tissues, the maximum neurological reflex stimulation /

inhibition occurs in the spinal cord, which then results in an effect on the autonomic nervous system and can alter end organ function. Sympathetic outflow is inhibited by non-noxious (non-painful) stimulus to the paraspinal tissues and noxious (painful) stimulus has a stimulatory effect (Korr, 1979b; Bergmann, 1992; Budgell, 2000; Pickar, 2002). Conversely the parasympathetic outflow is stimulated by non-noxious stimuli and inhibited by noxious stimuli (Sato, 1992).

The influence of these effects has been shown to change heart rate; blood pressure; pupillary diameter; distal skin temperature; as well as endocrine and immune system (Korr, 1979b; Bergmann, 1992; Slosberg, 1998; Murphy, 1999; Budgell, 2000; Pickar, 2002; Fryer, 2003; Welch and Boone, 2008).

With particular reference to blood pressure, the effects are thought to be principally related to the cervicosympathetic reflexes (reflexes mediated through the sympathetic chain and related cervical ganglia) and activation of the pressor reflexes (Knutson, 2001).

In this instance, increased transmission from the muscle spindles and Golgi Tendon Organs of the suboccipital spine has been shown to stimulate the cervicosympathetic reflexes (responsible for blood pressure and heart rate reduction) which counteract the vestibulosympathetic reflexes (responsible for blood pressure and heart rate increase). Thus cervicosympathetic reflexes from the upper spine lower heart rate and blood pressure (Sato, 1992; Knutson, 2001). Whereas the pressor reflex stimulates sympathetic flow to the heart as a result of suboccipital muscle contraction / hypertonicity, which has as an effect by decreasing vascularisation to the region. Thus to overcome the vascular constriction, the neurological reflexes increase blood pressure via the sympathetic activation of heart function.

It is therefore evident that there is a closely regulated and neurological integrated pattern of functioning that intricately regulates the blood pressure and heart rate of a subject. The next section now links this neurological system with the subluxation in order to propose the effects that spinal manipulation of a subluxation may have on blood pressure and heart rate.

## **2.12. PROPOSED EFFECTS OF CERVICAL SPINAL MANIPULATION ON BLOOD PRESSURE**

The chiropractic subluxation is associated with muscle hypertonicity (myopathy) and neuropathophysiology (Gatterman, 1990; Bergmann, 1992; Haldeman, 1992; Bergmann *et al.*, 1993; Haldeman, 2005). Manipulation has been shown to normalise muscular activity (Korr, 1979b) and induce normal neurological control of the motion segment (Korr, 1979b; Gatterman, 1995).

Upper cervical muscular hypertonicity has been associated with hypertension and abnormal arterial flow in the area of the atlas (Janetta, Segal and, Wolfson., 1995; Knutson, 2001; Chapman-Smith, 2007). As a result of these findings, Bakris *et al.*, (2007) theorised that there is a subpopulation of hypertensive people that have 'neurogenic hypertension' as a result of the pressor reflex (Knutson, 2001). He therefore performed magnetic resonance imaging (MRI) on hypertensive and normotensive individuals, evaluating the relationship between the ventrolateral surface of the medulla and the vertebral arteries and their branches. He found that 90.6% of the hypertensive individuals had some form of arterial compression. Due to these findings Bakris *et al.*, (2007) performed a clinical trial, concluding that a subluxation of the atlas is associated with hypertension principally due to ischemia of the brainstem's and upper cervical region's circulation. This was based on the fact that it was shown that manual correction (manipulation) of the subluxation reduced the blood pressure (through removal of the pressor reflex). In this context, the reduction in blood pressure was shown to be equivalent to administering two blood pressure tablets in combination (Bakris *et al.*, 2007; Chapman-Smith, 2007). It therefore stands to reason that manipulation could be a method of treatment that could be utilised to reduce expenditure on chronic medicines for this condition (Masarsky and Todres-Masarsky, 2001; Chapman-Smith, 2007).

These effects as found by Bakris *et al.*, (2007) are thought to be mediated through the parasympathetic innervation (vagus nerve) to the heart. The vagus nerve not only arises from the nucleus ambiguus and dorsal motor vagal nucleus (Dimmick *et al.*, 2006) [motor nuclei of cranial nerves III, VIII, IX, X, and XI (Welch and Boone, 2008)] in the brainstem, but also passes through the upper cervical complex after exiting the jugular foramen

(Guyton and Hall, 1997; Masarsky and Todres-Masarsky, 2001). Therefore making it subject to both the pressor reflex (inhibiting parasympathetic function), but also subject to the effects of manipulation which removes the pressor reflex (through a decrease in the muscle hypertonicity in the sub-occipital region) and also causing an increase in the neurological stimulation at the level of the brainstem, medulla and upper cervical spinal cord regions, thus facilitating a normalisation of abnormal neurological function via the interneuronal pool (Plaughner and Bachman, 1993; Driscoll and Hall, 2000).

**Table 2.3: Cervical Manipulation Studies**

Author/Appraisal of research source:	Title:	Sample parameters/strength of evidence:	Measurement tools:	Conclusion/interpretation of results/synthesis of results regarding current research:
Nansel, Jansen, Cremata, Dhami and Holley, 1991, <i>Journal of Manipulative and Physiological Therapeutics</i> .	Effects of cervical adjustments on lateral-flexion passive end-range asymmetry and on blood pressure, heart rate and plasma catecholamine levels.	Only healthy, asymptomatic male subjects who exhibited goniometrically verified lateral-flexion passive range of motion asymmetries of 10 degrees or greater on the morning of the experiment were chosen for the study.	The biomechanical and physiological effects of a single, unilateral lower cervical spinal adjustment delivered to the most restricted side of cervical lateral-flexion passive end-range were measured.	The results of this investigation indicated that lower cervical adjustments in asymptomatic subjects, didn't induce significant alterations in the overall activity of the sympathetic nervous system.
Licht, Christensen, Højgaard, Marving, 1998, <i>Journal of Manipulative Physiology and Therapeutics</i> .	Vertebral artery flow and spinal manipulation: a randomized, controlled and observer-blinded study.	A randomized, controlled and observer-blinded study at a university hospital vascular laboratory. Twenty university students with "biomechanical dysfunction" in the cervical spine.	Doppler ultrasound used to investigate whether any changes occur in peak flow velocity in the vertebral artery after spinal manipulative therapy.	No change in peak flow velocity immediately after spinal manipulative therapy was found and no correlation between peak flow velocity and systolic blood pressure

Author/Appraisal of research source:	Title:	Sample parameters/strength of evidence:	Measurement tools:	Conclusion/interpretation of results/synthesis of results regarding current research:
Knutson, 2001, <i>Journal of Manipulative Physiology and Therapeutics</i> .	Significant changes in systolic blood pressure post vectored upper cervical adjustment vs resting control groups: a possible effect of the cervicosympathetic and/or pressor reflex.	Study took place in a private chiropractic practice. Test 1: Forty patients demonstrating signs of upper cervical subluxation and 40 patients without such signs. Test 2: Thirty patients demonstrating signs of upper cervical subluxation.	Intervention was a specific, vectored upper cervical (atlas) adjustment or similarly positioned resting. Main outcome measured: Pre-rest, post-rest, and post adjustment systolic, diastolic, and pulse rates as recorded through use of a digital oscillometric sphygmomanometer	The results indicated that palpation and vectored atlas adjustment caused a significant decrease in systolic blood pressure in patients with upper cervical subluxation.
Bakris, Dickholtz, Meyer, Kravitz, Avery, Miller, Brown and Bell, 2007, <i>Atlas Research Foundation and the NIH K25, HL68139-01A1</i> .	Atlas vertebra realignment and achievement of arterial pressure goal in hypertensive patients: a pilot study	An 8 week double blind, placebo-controlled design at a single centre, 50 drug naïve (n=26) or washed out (n=24) patients with Stage 1 hypertension.	Treatment=National Upper Cervical Chiropractic (NUCCA) procedure. Placebo= a sham procedure	Atlas realignment demonstrated sustained blood pressure reductions, similar to the use of two-drug combination therapy.

## 2.13. PROPOSED EFFECTS OF THORACIC SPINAL MANIPULATION ON BLOOD PRESSURE

The effect of thoracic manipulation affecting heart rate and blood pressure may be threefold:

1. Through the pressor reflex associated with the stimulated sympathetic efferent pathways from the superior, middle cervical and stellate ganglia can affect the heart rate and blood pressure negatively (Knutson, 2001). The effect of manipulation of the upper thoracic spine therefore will change the neurological firing pattern of the sympathetic neurons that exit at these levels; having an indirect effect on reducing the sympathetic stimulation in the upper cervical spine (suboccipital region) normalising the blood flow in the upper cervical spine region and thus negating the pressor reflex and normalising blood pressure (Knutson, 2001).

2. The presence of a subluxation in the upper thoracic spine causing abnormal neurological reflex patterns (Patterson and Steinmetz, 1986). Therefore the effect of upper thoracic manipulation may affect the normalisation of the sympathetic nervous system at the point of exit from the spinal cord ( $T_1 - T_5$ ). If this mechanism were to be the one responsible for changes in heart rate and blood pressure, then the proposed theoretical constructs of Korr (1979b), Sato (1992) and Gatterman (1995), would be the most applicable; in that they articulate with the neurological stimulation of a spinal cord segment causing facilitation of that segment and thus either inhibition or stimulation of the autonomic nervous system, causing it to no longer be influenced by abnormal neurological reflex pathways and reaching a homeostatic normal.
3. Lastly sympathetic innervation is extensive within the upper thoracic region, thus a spinal manipulation could elicit a sympathetic response, thus increasing heart rate, as well as blood pressure (Welch and Boone, 2008). Manipulation over the thoracic region has a direct mechanical effect on the heart and the heart's great vessels, activating the cardiac mechanoreceptors (Budgell and Polus, 2006). Thus the sympathetic nervous system is stimulated as a result of a thoracic manipulation and in turn the parasympathetic nervous system counteracts the sympathetic response, and it is fair to say that the autonomic nervous system is activated from a thoracic manipulation (Budgell and Polus, 2006).

**Table 2.4: Thoracic Manipulation Studies**

Author/ Appraisal of research source:	Title:	Sample parameters/strength of evidence:	Measurement tools:	Conclusion//interpretation of results/synthesis of results regarding current research:
Yates, Lamping, Abraham and Wright, 1988, <i>Journal of Manipulative and Physiological Therapeutics</i> .	Effects of chiropractic treatment on blood pressure and anxiety: a randomized, controlled trial.	Subjects were randomly assigned to one of three treatment conditions: active treatment, placebo treatment, or no treatment control. Twenty-one patients with elevated blood pressure were used.	Intervention was a thoracic manipulation at T <sub>1</sub> -T <sub>5</sub> . Dependent measures obtained pre- and post-treatment included systolic and diastolic blood pressure, and state anxiety	Spinal manipulations to T <sub>1</sub> - T <sub>5</sub> proved to statistically lower systolic and diastolic blood pressure.
Budgell, and Polus, 2006, <i>Journal of Manipulative and Physiological Therapeutics</i> .	The Effects of Thoracic Manipulation on Heart Rate Variability: A Controlled Crossover Trial	A controlled crossover trial that was conducted on 28 healthy young adults (23 men and 5 women; age range, 18-45 years; mean age, 29 ± 7 years).	Heart rate variability was measured before and after a sham procedure and a thoracic spinal manipulation.	High-velocity and low- amplitude manipulation of the thoracic spine appeared to influence autonomic output to the heart.

## **2.14. PROPOSED EFFECTS OF CERVICAL AND THORACIC SPINAL MANIPULATION ON BLOOD PRESSURE**

It is therefore possible, based on the proposed mechanism of action, that a combination of cervical and thoracic manipulation would achieve the greatest benefit as it affects both the sympathetic and parasympathetic system thus mediating a mechanism that more readily establishes a normalisation of the relationship between the two components of the autonomic nervous system.

**Table 2.5: Full Spine Manipulation Studies**

Author/ Appraisal of research source:	Title:	Sample Parameters/strength of evidence:	Measurement tools:	Conclusion/interpretation of results/synthesis of results regarding current research:
Plaughter, Long, Alcantara, Silveus, Wood, Lotun, Menke, Meeker and Rowe, 2002, <i>Journal of Manipulative Physiology and Therapeutics.</i>	Practice-based randomized controlled- comparison clinical trial of chiropractic adjustments and brief massage treatment at sites of subluxation in subjects with essential hypertension: pilot study.	Private practice outpatient chiropractic clinic Randomized controlled- comparison trial with 3 parallel groups. Twenty- three subjects aged 24 to 50 years with systolic or diastolic essential hypertension.	Interventions: Group one had two months of full-spine chiropractic care (i.e., Gonstead) consisting primarily of specific-contact, short-lever-arm adjustments delivered at a subluxation. Group two was the massage group having a brief effleurage procedure delivered at subluxed areas. Group three, the non treatment control group rested alone for a period of approximately 5 minutes in an adjustment room	Chiropractic treatment proved to be beneficial in reduction of systolic and diastolic blood pressure.
Dimmick, Young, Newell, 2006, <i>Journal of Manipulative and Physiological Therapeutics.</i>	Chiropractic Manipulation affects the difference between arterial systolic blood pressures on the left and right in normotensive subjects.	A nonrandomized, matched pair, controlled clinical trial, with the treatment group (35 people) and control group (35 people) matched for age and sex, was performed in chiropractic student clinics in London, UK. The trial took four months.	Intervention was chiropractic manipulation. Pre- intervention and post-intervention systolic and diastolic blood pressures were recorded in both arms through the use of a digital oscillometric sphygmomanometer.	Chiropractic treatment appeared to have an effect on the difference in systolic blood pressure between the arms.
Welch and Boone, 2008, <i>Journal of Chiropractic Medicine.</i>	Sympathetic and parasympathetic responses to specific diversified adjustments to chiropractic vertebral subluxations of the cervical and thoracic spine.	Forty patients (25- 55 years old) met inclusion criteria that consisted of normal blood pressure, no history of heart disease, and being asymptomatic.	Patients were evaluated pre- and post-chiropractic adjustment for the following autonomic responses: Blood pressure and pulse rate. Seven patients were measured for heart rate variability. The subjects received either a diversified cervical or thoracic segment adjustment.	Sympathetic response to thoracic manipulation was evident; however not a significant change in blood pressure was noted. Diastolic blood pressure, decreased following a cervical manipulation.



This only consists of studies incorporating full spine manipulation, without isolation of the various regions in the spine that have been hypothesised to affect blood pressure and heart rate, therefore the aim of this research was to investigate the effects of cervical and thoracic spine manipulations, cervical spine manipulations alone and thoracic spine manipulations alone on heart rate and blood pressure in normotensive males in order to establish whether the underpinning theories would support a decrease in these parameters. The clinical significance of such outcomes would be to possibly explore in greater detail the length of time these neurophysiological effects sustain on blood pressure, thus aiding in hypertension and overall care of patient. Another consideration into the clinical significance would be to aid in cervical manipulation of a subluxed hypertensive patient, generally it would be contra-indicated, however if such a patient presented, a thoracic manipulation could be administered, which theoretically would cause a reduction in blood pressure, thus a cervical spine manipulation would no longer be considered contra-indicated and the individual could be treated also aiding in blood pressure reduction.

## **CHAPTER 3**

### **MATERIALS AND METHODS**

#### **3.1. STUDY DESIGN**

This investigation was designed as a prospective, randomised controlled trial, involving a group of forty asymptomatic males between 20-35 years of age. Based on this design, the study was approved by the Faculty of Health Sciences Research and Ethics Committee at the Durban University of Technology (Appendix C), indicating that the study complied with the Belmont, Helsinki and Nuremburg guidelines (Johnson, 2005) that outline the requirements for research studies with human subjects. This means that all subjects took part in a voluntary basis and each subject was required to read and sign a Letter of Information and Consent form (Appendix B). In this context, the Letter of Information described in detail the study's protocol and subject requirements for participation and for the duration of the study. They were informed that they could withdraw from the study at any time and all information would remain confidential.

##### **3.1.1. ADVERTISING**

Subjects were recruited by advertising for volunteers at the Durban University of Technology campus and around the greater Durban area. Recruitment of subjects entailed approaching groups of students on campus, distribution of flyers and posters around the gymnasium and library (Appendix A), as well as approaching the coaches of the Durban University of Technology's sports teams and encouraging the participation of the team member's in the study. Those who were interested were asked to contact the Chiropractic Day Clinic at the Durban University of Technology and leave their details or book an appointment with the researcher.

### 3.1.2. INITIAL SCREENING OF THE PROSPECTIVE SUBJECTS

Only male subjects between 20-35 years of age were allowed into the study (Kaplan, 1986; Fox, 1999). There was no bias given to race, religion or socio-economic standing.

Preliminary questions were asked by the researcher via telephonic or personal interviews to determine whether the prospective subject was eligible for inclusion in the study:

- “What is your age?”
- “Are you a smoker?”
- “Do you have a history of high blood pressure?”
- “Are you on any blood pressure medication?”
- “Have you had any recent trauma to your neck or upper back?”

If the subject was below the age of 20 or above the age of 35, and / or if the prospective subject was a female and / or answered yes to the above questions, that subject was deemed ineligible to participate in the study.

When the subject was deemed a likely candidate he was given a clear explanation as to the nature and expectations of the research. It was explained that the objective of the study was to determine the effect of cervical and thoracic manipulations on blood pressure. Furthermore subjects were told that they would be subjected to four consecutive days of intervention with the following outline:

- Day 1 : Laser (understood by the researcher to be sham laser),
- Day 2 : C<sub>0</sub>/C<sub>1</sub> manipulation,
- Day 3 : T<sub>1</sub>-T<sub>5</sub> manipulation and
- Day 4 : C<sub>0</sub>/C<sub>1</sub> and T<sub>1</sub>-T<sub>5</sub> manipulation.

The subject was then reserved an appointment at the Chiropractic Day Clinic to meet the researcher and to read the Letter of Information and sign an Informed Consent form (Appendix B) regarding the study. During this process, the nature and hypotheses as well as the research requirements were explained verbally to each subject and any further questions or queries were also addressed.

Thereafter the potential subject underwent a case history (Appendix D), physical examination (Appendix E), cervical spinal regional examination (Appendix F) and a thoracic spinal regional examination (Appendix G). All data collected was recorded on a SOAP note (Appendix H). These examinations were done to thoroughly assess for any factors that would deem the potential subject not eligible for the study (such as bradycardia, tachycardia, hypertension and an abnormal height / weight ratio), as well as that constitute contra-indications to manipulation. Furthermore, the assessment ensured that the potential subject complied with the following inclusion and exclusion criteria.

### **3.2. INCLUSION AND EXCLUSION CRITERIA OF THE SUBJECTS:**

#### **3.2.1. INCLUSION CRITERIA:**

- Subjects in the study had to have a systolic reading between 120-140 mmHg and a diastolic reading between 75-90 mmHg, to ensure homogeneity of the group (Mouton, 1996; Guyton and Hall, 1997; Fox, 1999).
- Only healthy males between 20-35 years of age were accepted into the study, to ensure homogeneity of the group (Mouton, 1996; Guyton and Hall, 1997; Fox, 1999).
- Subjects had to have a resting heart rate of 60-100 beats per minute, to ensure homogeneity of the group (Mouton, 1996; Palatini, 1999).
- Subjects were only accepted into the study if they gave their informed consent in writing (Appendix B).

### 3.2.2. EXCLUSION CRITERIA:

- Subjects were excluded if they had a Body Mass Index above  $27 \text{ kg/m}(\text{height})^2$ , to ensure homogeneity of the group as well as eliminating the effects of obesity on blood pressure (Mouton, 1996; Kumar *et al.*, 2003).
- Females were excluded from the study, to maintain homogeneity as well as to exclude expectancy effects that differ between genders (Mouton, 1996; Parker, 2005).
- Subjects were excluded if they were on anti-hypertensive medication (Kaplan, 1986; Seedat, 1989; Fox, 1999; Sutherland, 2002).
- Subjects were excluded from the study if they were on any medication that could affect blood pressure indirectly, such as NSAIDS, heart medication, anti-bacterial medications as well as arthritic medications (Kaplan, 1986; Seedat, 1989; Fox, 1999; Sutherland, 2002).
- Smokers were excluded from the study, because smoking has been shown to affect blood pressure (Kaplan, 1986; Seedat, 1989; Fox, 1999; Sutherland, 2002; Frosh, Dierker, Rose and Waldinger, 2009).
- Subjects were excluded if they suffered from any known contra-indications to manipulation, which include but are not limited to (Kaplan, 1986; Seedat, 1989; Bergmann, 1992; Fox, 1999; Sutherland, 2002):
  - Vertebral artery insufficiency,
  - Spondylolisthesis,
  - Vertebral malignancy,
  - Vertebral disc prolapse causing nerve root entrapment,
  - Rheumatoid arthritis,
  - Severe osteoporosis,
  - Acute vertebral fracture,
  - Osteomyelitis,

- Tuberculosis and / or
- Infective arthritis.

### **3.3. MATERIALS:**

All blood pressure readings and heart rates were recorded using a fully automated digital blood pressure monitor (Microlife BP 3AG1; Microlife AG Max Schmidheiny-Strasse 201, 9435 Heerbrugg / Switzerland).

This unit was selected in order to:

- Ensure consistency in the calibration of the unit
- Ensure that placement and recordings were done systematically on each participant
- Ensure that human error, parallax errors and blood pressure gauge errors were eliminated (Bickley and Szilagyi, 2008; Douglas, Nicol, and Robertson, 2009).

The unit has been tested according to the British Hypertension Society (BHS) (Cuckson, Reinders Shabeeh and Shennan, 2002) and has the highest possible grading for Systolic (A) and Diastolic (A) measurement accuracy. The technical specifications of the unit measures a blood pressure range from 30mmHg to 300mmHg with accuracy within +/- 3mmHg, and the range of pulse rate measures within 40-200 beats per minute (Cuckson, Reinders, Shabeeh and Shennan, 2002).

The subjects' weight and height were recorded simultaneously on a height-weight scale in the Chiropractic Day Clinic of the Durban University of Technology.

### 3.4. INTERVENTION:

All subjects that passed the initial screening for inclusion into the study then underwent four consecutive days of intervention.

The blood pressure was taken as follows at pre-intervention and post intervention (at the specified time intervals):

The blood pressure was recorded using the automated digital Microlife BP3AG1 unit. The subject was instructed to sit on a chair with a backrest for support. The right arm was rested on a table top at the height of the subject's elbow, with the arm in a relaxed position. Once the cuff of the unit was wrapped around the upper arm just above the elbow, the subject was asked to remain still while the unit measured the blood pressure and heart rate. The unit was then turned on and the cuff was inflated, the reading of blood pressure and heart rate were noted and recorded and the unit was switched off.



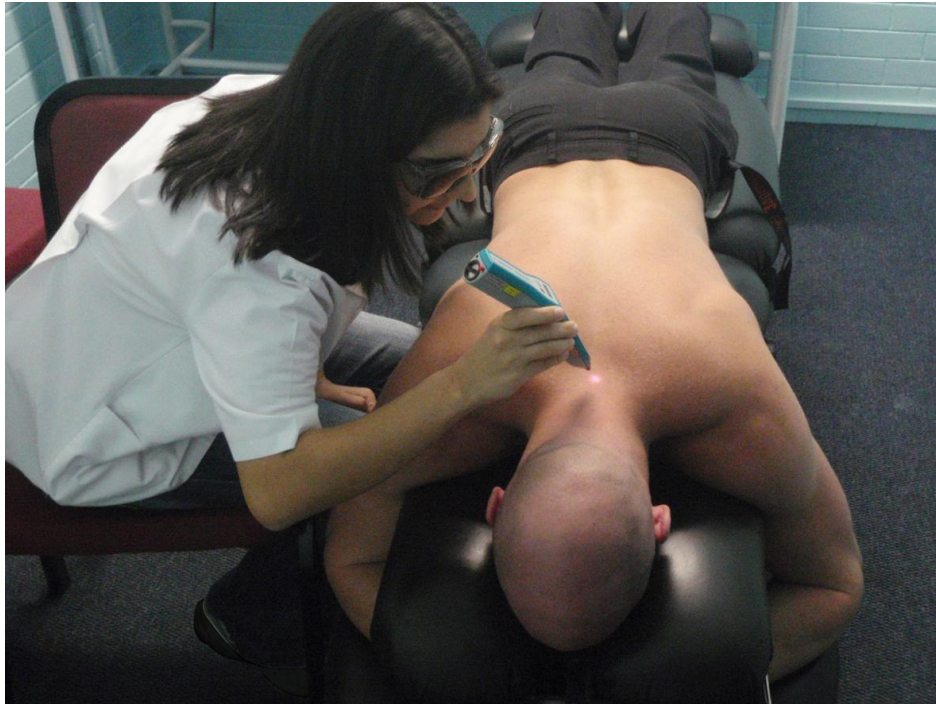
**Figure 3.1: Depicting the Method of Measuring Blood Pressure and Heart Rate**  
**(Permission for photo, Appendix I)**

❖ **Intervention A** (Day 1): Sham laser.

Once the basal blood pressure was recorded, intervention A was administered. Subjects were asked if they wished to see how the laser operated and this was demonstrated to those that positively indicated as such. The laser unit had been set to emit only a red light without the presence of a crystal, thus rendering it clinically useless, but allowing the patients to believe that it worked. This assisted in ensuring that there was a minimal placebo effect (Richardson, 2007). Subjects were then told to lie face down on a table, prior to being tested for laser therapy (sensation) as would have been done if the laser had been an active laser (Forster and Palastanga, 1985). The use of the sham treatment was to record the effects of 'treatment anxiety' on the subjects and their blood pressure readings. The use of laser was to ensure that the subject had no contact over the skin and soft tissues which could arguably have an stimulatory effect and alter the blood pressure readings.

The laser unit machine was then set to zero, the subjects were told not to attempt turning their heads to look at the laser beam, in addition the researcher wore the protective laser goggles (to further enhance the placebo effect). Subjects were informed during the treatment that they should not have expected to feel any difference in the area treated and that no side-effects should be expected. Blood pressure was then recorded another three times following the intervention, this was executed in the same manner as the previous recording. Readings were recorded five minutes, 15 minutes and 30 minutes post-intervention.





**Figure 3.2. Depicting Sham Laser Intervention**  
**(Permission for photo, Appendix I)**

- ❖ **Intervention B**, executed on Day 2 was a cervical manipulation, at the atlanto-occipital joints bilaterally in the posterior, rotation direction (Janetta *et al.*, 1995; Bakris *et al.*, 2007).

The subject's blood pressure was recorded prior to the intervention. Once the basal blood pressure was recorded using the same manner as previously discussed, intervention B was administered.

Subjects were told to lie supine on the adjusting table while the researcher stood at the subject's head. The researcher's indifferent hand supported the subject's neck by cupping the ear and supporting the cervical spine with the fingers. The contact hand was then over the joint being manipulated, thrust was given in a rotation direction to the atlanto-occipital joint (Bergmann, 1992; Murphy 1999).



**Figure 3.3: Depicting C<sub>0</sub>/C<sub>1</sub> Spinal Manipulation Intervention**  
**(Permission for photo, Appendix I)**

Blood pressure was then recorded another three times following the intervention, this was executed in the same manner as the previous recording. Readings were recorded five minutes, 15 minutes and 30 minutes post-intervention.

- ❖ **Intervention C**, executed on Day 3 was a thoracic spine manipulation at T<sub>1</sub>-T<sub>5</sub> bilaterally in extension, on the facet joints.

The subject's blood pressure was recorded prior to any intervention. Once the basal blood pressure was recorded using the same manner as previously discussed, intervention C was administered.

Subjects were instructed to lie prone on the adjusting table, while the researcher stood beside them, contacting with the lateral aspects of hypothenar eminences over the thoracic facet joints, bilaterally. A high velocity, low amplitude thrust was delivered to the joints at T<sub>3</sub> to affect the motion segment above (T<sub>2</sub>) and below (T<sub>4</sub>). (Bergmann, 1992; Murphy, 1999).

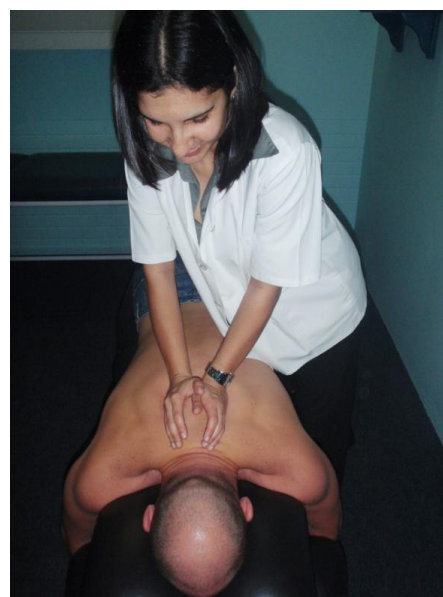
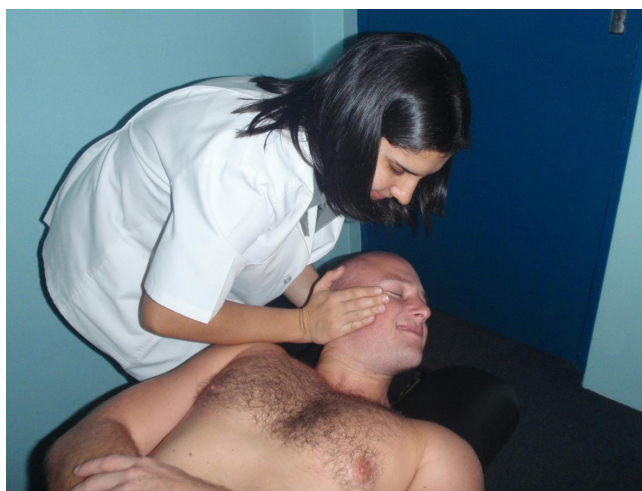


**Figure 3.4: Depicting T<sub>1</sub>-T<sub>5</sub> Spinal Manipulation Intervention**  
**(Permission for photo, Appendix I)**

Blood pressure was then recorded another three times following the intervention, this was executed in the same manner as the previous recording. Readings were recorded five minutes, 15 minutes and 30 minutes post-intervention.

- ❖ **Intervention D**, executed on Day 4 was a combination of the manipulative techniques used in intervention B and intervention C.

The subject's blood pressure was recorded prior to any intervention. Once the basal blood pressure was recorded using the same manner as previously discussed, intervention D (a combination of manipulative procedures in intervention B and C at the same joint levels) was administered.



**Figure 3.5: Depicting C<sub>0</sub>/C<sub>1</sub> and T<sub>1</sub>-T<sub>5</sub> Spinal Manipulation Intervention**  
**(Permission for photo, Appendix I)**

Blood pressure was then recorded another three times following the intervention, this was executed in the same manner as the previous recording. Readings were recorded five minutes, 15 minutes and 30 minutes post-intervention.

### **3.5. STATISTICAL METHODOLOGY:**

SPSS version 15.0 (SPSS Inc., Chicago, Illinois, USA) was used to analyze the data. A p value <0.05 was considered as statistically significant.

Normal distribution testing was completed using the One-Sample Kolmogorov-Smirnov Test. This was done to justify the use of parametric testing.

To determine the effect of cervical and thoracic manipulations on blood pressure, repeated measures ANOVA generalized linear models were used. Profile plots were generated to compare the trends visually. Simple and repeated contrasts were used to compare the change in pressure over time between all treatments.

## **CHAPTER 4**

### **RESULTS**

#### **4.1. INTRODUCTION**

This chapter encompasses the statistical analysis of the blood pressure readings recorded over the four days pre and post interventions to assess the effects of sham laser, cervical manipulation, thoracic manipulation and a combination of cervical and thoracic manipulation on blood pressure. Results obtained from the statistical analysis of the raw data are depicted in linear graphs, with explanations of the results.

##### **4.1.1. DATA**

Blood pressure readings comprised of a systolic blood pressure reading and diastolic blood pressure reading. A baseline blood pressure reading (both systolic and diastolic) was recorded on each day prior to any intervention. In addition, three post intervention readings were recorded, five minutes, 15 minutes and 30 minutes after the intervention.

## **4.2. DEMOGRAPHIC DATA**

Forty males participated in the research study, there were no subjects that did not complete the study.

Within this grouping subjects were aged between 20 – 35 years of age, resulting in a median age 24 years. Furthermore, the heights of subjects ranged from 1.6 metres to 1.9 metres, with a median height of 1.75 metres. The weight of all subjects ranged from 51 kilograms to 88 kilograms, giving a median weight of 72.5 kilograms.

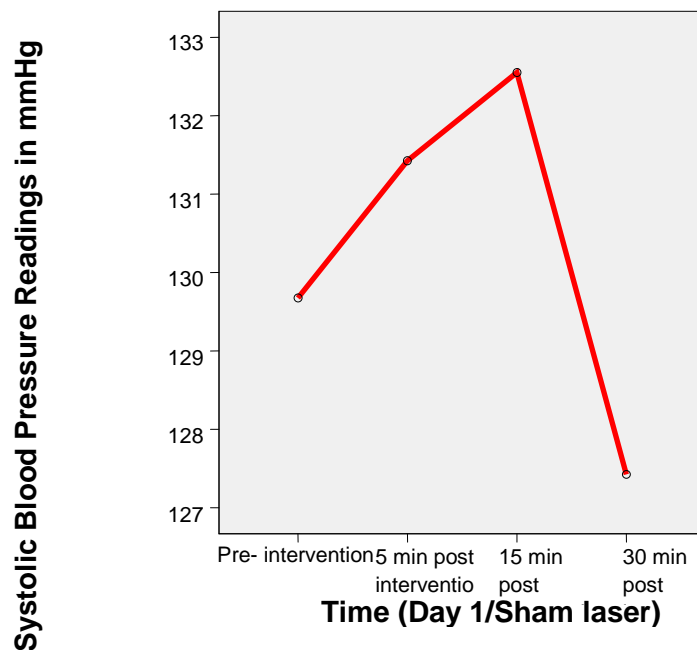
The heart rate of all subjects was required to be between 60 – 100 beats per minute. The slowest heart rate recorded was 60 beats per minute and the fastest heart rate recorded was 99 beats per minute. Thus the median heart rate was 74 beats per minute.

These parameters therefore met the inclusion and exclusion criteria for gender, age, BMI and heart rate.

### 4.3. DAY 1 READINGS

#### 4.3.1. ANALYSIS OF SYSTOLIC BLOOD PRESSURE READINGS ON DAY1 / SHAM LASER

Figure 4.1 reflects that the systolic blood pressure reading on Day 1.



**Figure 4.1: Mean Systolic Blood Pressure Readings on Day 1/Sham Laser**

The data in Figure 4.1 reflects that of systolic blood pressure with regards to sham laser treatment. Systolic blood pressure did decrease after 30 minutes, however at five and 15 minutes post intervention, systolic blood pressure increased slightly, owing to the possible effects of 'treatment anxiety'.

**Table 4.1: The Effect of Day 1/Sham Laser Intervention on Systolic Blood Pressure Readings**

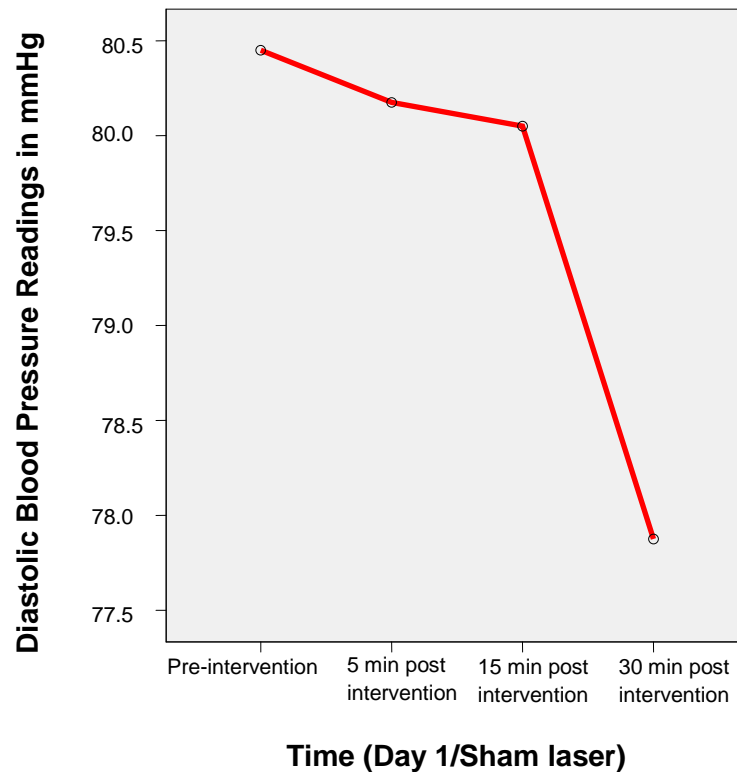
Effect		Value	F	<i>p</i>
Day 1 Systolic Blood Pressure Readings	Wilks' Lambda	.852	2.134	0.112

Table 4.1 depicts that a ( $p=0.112$ ) was calculated, thus the effects on systolic blood pressure from sham laser treatment were insignificant.



#### 4.3.2. ANALYSIS OF DIASTOLIC READINGS ON DAY 1/SHAM LASER

Figure 4.2 reflects diastolic blood pressure readings on Day 1/Sham Laser.



**Figure 4.2: Mean Diastolic Blood Pressure Readings on Day 1/Sham Laser**

Figure 4.2 shows the decline in diastolic blood pressure post sham laser treatment, which after five minutes and fifteen minutes is reduced, the greatest decline is at 30 minutes post intervention.

**Table 4.2: The Effect of Day 1/Sham Laser Intervention on Diastolic Blood Pressure Readings**

Effect		Value	F	<i>p</i>
Day 1 Diastolic Blood Pressure Readings	Wilks' Lambda	.807	2.957	0.045

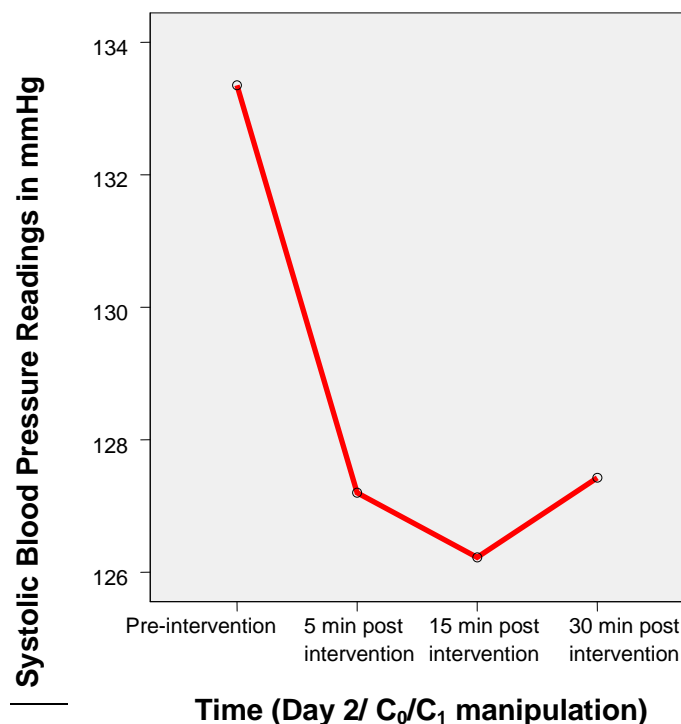
Table 4.2 reflects that the effect of the Day 1/Sham laser intervention on diastolic pressure is statistically significant ( $p=0.045$ ).



#### 4.4. DAY 2 READINGS

##### 4.4.1. ANALYSIS OF SYSTOLIC BLOOD PRESSURE READINGS ON DAY 2 / C<sub>0</sub>/C<sub>1</sub> MANIPULATION

Figure 4.3 reflects systolic blood pressure readings on Day 2/ C<sub>0</sub>/C<sub>1</sub> Manipulation.



**Figure 4.3: Mean Systolic Blood Pressure Readings on Day 2/ C<sub>0</sub>/C<sub>1</sub> Manipulation**

Figure 4.3 shows a decline in systolic blood pressure, following five minutes post intervention the reduction was substantial, the decline continued till 15 minutes, however systolic blood pressure increased at 30 minutes post intervention.

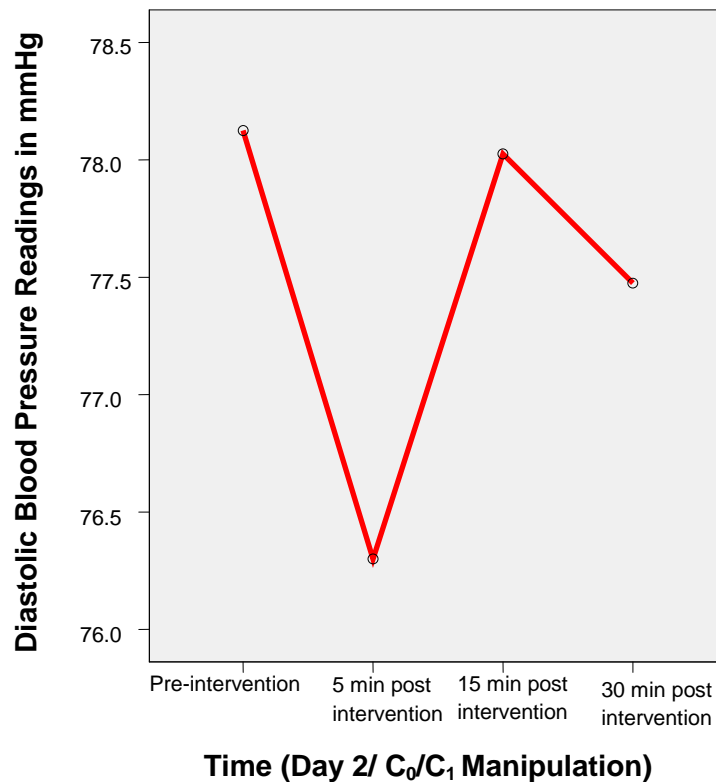
**Table 4.3: The Effect of Day 2/C<sub>0</sub>/C<sub>1</sub> Manipulation Intervention on Systolic Blood Pressure Readings**

Effect		Value	F	<i>p</i>
Day 2 Systolic Blood Pressure Readings	Wilks' Lambda	.707	5.113	0.005

Table 4.3 reflects that the effect of the Day 2/C<sub>0</sub>/C<sub>1</sub> Manipulation intervention on systolic blood pressure readings is statistically significant ( $p=0.005$ ).

#### 4.4.2. ANALYSIS OF DIASTOLIC BLOOD PRESSURE READINGS ON DAY 2 / $C_0/C_1$ MANIPULATION

Figure 4.4 reflects the change in diastolic blood pressure readings on Day 2/  $C_0/C_1$  manipulation.



**Figure 4.4: Mean Diastolic Blood Pressure Readings on Day 2/  $C_0/C_1$  Manipulation**

Figure 4.4 shows the fluctuations of diastolic blood pressure post  $C_0/C_1$  manipulation. At five minutes post intervention the decline in diastolic blood pressure is substantial, however at 15 minutes the effects of the manipulation on diastolic blood pressure are no longer occurring.

**Table 4.4: The Effect of Day 2/  $C_0/C_1$  Manipulation Intervention on Diastolic Blood Pressure Readings**

Effect		Value	F	<i>p</i>
Day 2 Diastolic Blood Pressure Readings	Wilks' Lambda	.904	1.307	0.287

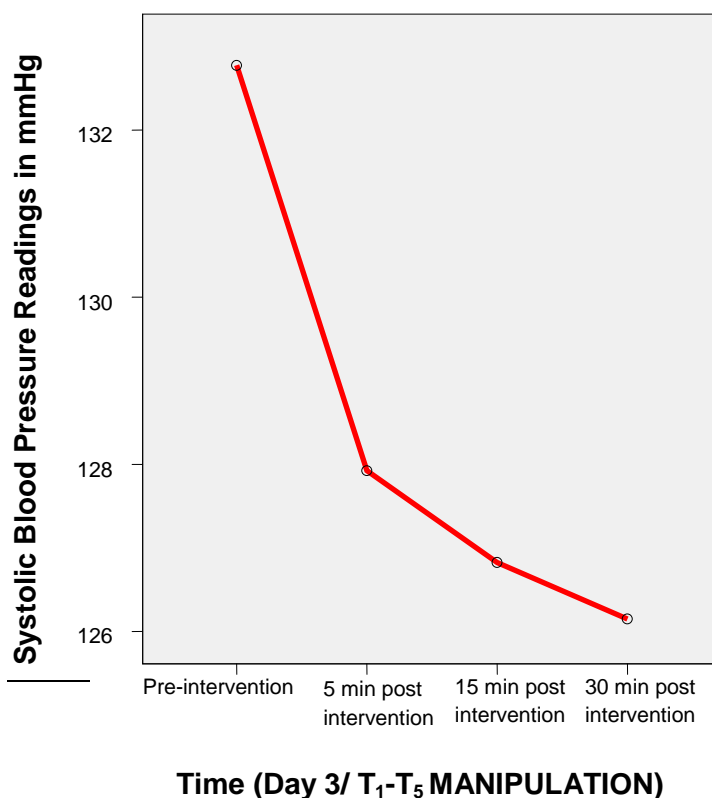
Table 4.4 reflects that the effect of the Day 2/  $C_0/C_1$  intervention on diastolic blood pressure readings is not statistically significant ( $p=0.287$ ).

## 4.5. DAY 3 READINGS

### 4.5.1. ANALYSIS OF SYSTOLIC BLOOD PRESSURE READINGS ON DAY 3 /

#### T<sub>1</sub>-T<sub>5</sub> MANIPULATION

Figure 4.5 reflects the decrease in systolic blood pressure readings on Day 3/ T<sub>1</sub>-T<sub>5</sub> manipulation.



**Figure 4.5: Mean Systolic Blood Pressure Readings on Day 3/ T<sub>1</sub>-T<sub>5</sub> Manipulation**

Figure 4.5 shows the reduction in systolic blood pressure following a thoracic manipulation, the reduction in the systolic reading is greatest at five minutes post intervention and continues to decline at 15 and 30 minutes post intervention.

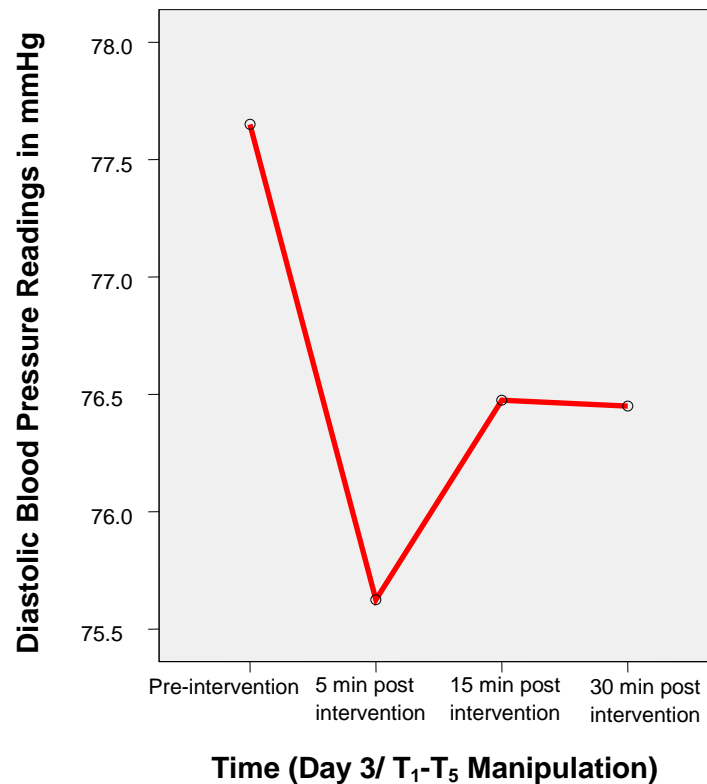
**Table 4.5: The Effect of Day 3/ T<sub>1</sub>-T<sub>5</sub> Manipulation Intervention on Systolic Blood Pressure Readings**

Effect		Value	F	P
Day 3 Systolic Blood Pressure Readings	Wilks' Lambda	.698	5.334	0.004

Table 4.5 reflects that the effect of the Day 3/T<sub>1</sub>-T<sub>5</sub> manipulation intervention on systolic blood pressure readings is statistically significant ( $p < 0.004$ ).

#### 4.5.2. ANALYSIS OF DIASTOLIC BLOOD PRESSURE READINGS ON DAY 3 / T<sub>1</sub>-T<sub>5</sub> MANIPULATION

Figure 4.6 reflects the change in diastolic blood pressure readings on Day 3/ T<sub>1</sub>-T<sub>5</sub> manipulation.



**Figure 4.6: Mean Diastolic Blood Pressure Readings on Day 3/ T<sub>1</sub>-T<sub>5</sub> Manipulation**

Figure 4.6 shows the reduction in diastolic blood pressure post thoracic manipulation, the reduction is significantly the greatest at five minutes post manipulation. Thereafter it inclines steadily at 15 minutes post manipulation and maintains that level at 30 minutes post manipulation.

**Table 4.6: The Effect of Day 3/T<sub>1</sub>-T<sub>5</sub> Manipulation Intervention  
on Diastolic Blood Pressure Readings**

Effect		Value	F	<i>p</i>
Day 3 Diastolic Blood Pressure Readings	Wilks' Lambda	.930	.929	0.436

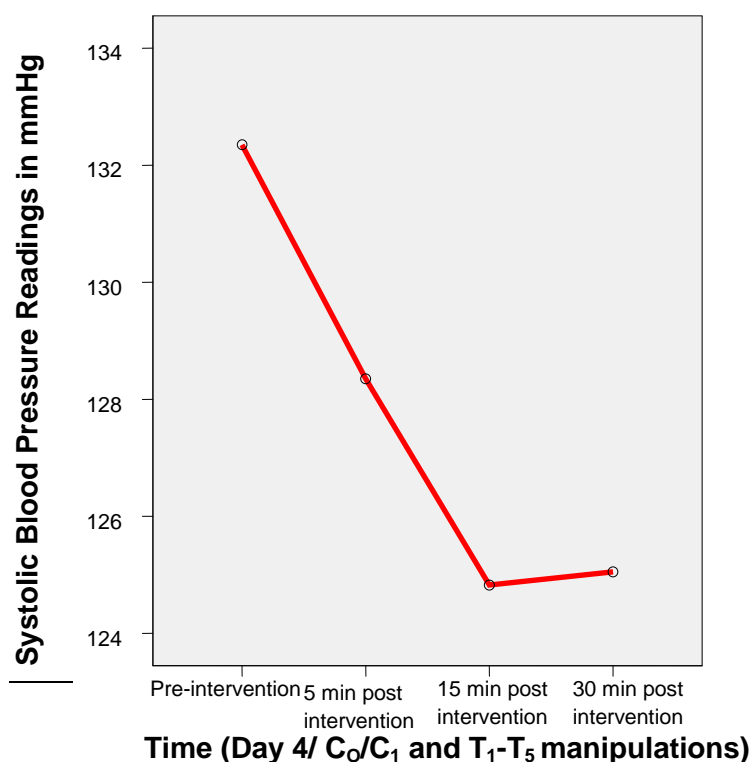
Table 4.6 reflects that the effect of the Day 3/ T<sub>1</sub>-T<sub>5</sub> intervention on diastolic blood pressure readings is not statistically significant ( $p=0.436$ ).

## 4.6. DAY 4 READINGS

### 4.6.1. ANALYSIS OF SYSTOLIC BLOOD PRESSURE READINGS ON DAY 4 /

#### $C_0/C_1$ AND $T_1-T_5$ MANIPULATIONS

Figure 4.7 reflects the change in systolic blood pressure readings on Day 4/ combination of  $C_0/C_1$  and  $T_1-T_5$  manipulations.



**Figure 4.7: Mean Systolic Blood Pressure Readings on Day 4/  
 $C_0/C_1$  and  $T_1-T_5$  Manipulations**

Figure 4.7 shows the reduction in systolic blood pressure is great at five minutes post intervention and continues to decline until 15 minutes post intervention, then rises slightly at 30 minutes post intervention.

**Table 4.7: The Effect of Day 4/  $C_0/C_1$  and  $T_1-T_5$  Manipulations Intervention  
on Systolic Blood Pressure Readings**

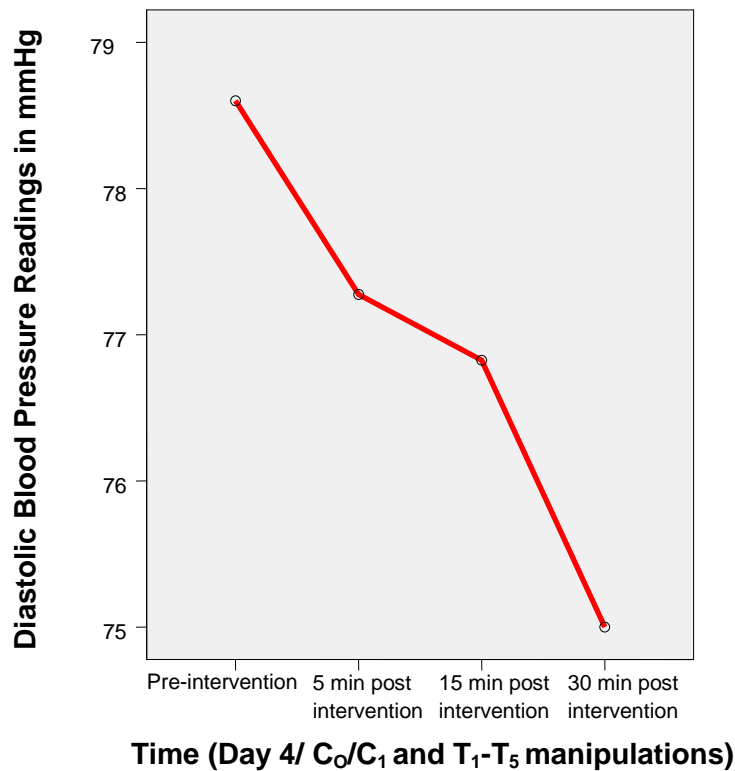
Effect		Value	F	$p$
Day 4 Systolic Blood Pressure Readings	Wilks' Lambda	.641	6.902	0.001

Table 4.7 reflects that the effect of the Day 4/  $C_0/C_1$  and  $T_1-T_5$  intervention on systolic blood pressure readings is statistically significant ( $p=0.001$ ).

#### 4.6.2.ANALYSIS OF DIASTOLIC BLOOD PRESSURE READINGS ON DAY 4 /

##### **C<sub>0</sub>/C<sub>1</sub> AND T<sub>1</sub>-T<sub>5</sub> MANIPULATIONS**

Figure 4.8 reflects the change in diastolic blood pressure readings on Day 4/ C<sub>0</sub>/C<sub>1</sub> and T<sub>1</sub>-T<sub>5</sub> manipulations.



**Figure 4.8: Mean Diastolic Blood Pressure Readings on Day 4/ C<sub>0</sub>/C<sub>1</sub> and T<sub>1</sub>-T<sub>5</sub> Manipulations**

Figure 4.8 shows the reduction in diastolic blood pressure post intervention, the blood pressure declined significantly at five minutes post intervention and continued to decline further at 30 minutes post intervention.

**Table 4.8: The Effect of Day 4 Intervention on Diastolic Blood Pressure Readings**

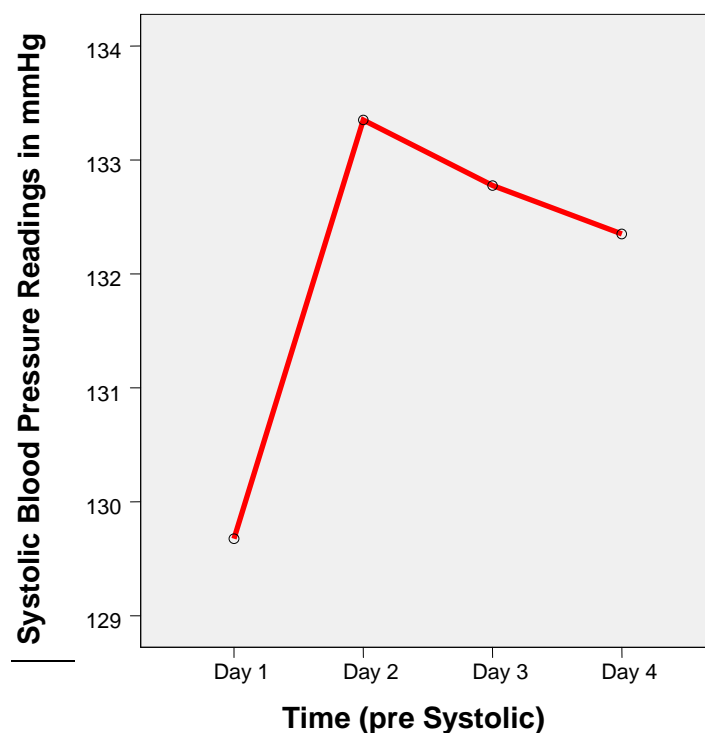
Effect		Value	F	<i>p</i>
Day 4 Diastolic Blood Pressure Readings	Wilks' Lambda	.903	1.325	0.281

Table 4.8 reflects that the effect of the Day 4/ C<sub>0</sub>/C<sub>1</sub> and T<sub>1</sub>-T<sub>5</sub> intervention on diastolic blood pressure readings is not statistically significant ( $p=0.281$ ).

## 4.7. COMPARISON OVER THE 4 DAYS

### 4.7.1. PRE TEST SYSTOLIC BLOOD PRESSURE READINGS

Figure 4.9 reflects the change in systolic blood pressure readings over the four Days.



**Figure 4.9: Mean Pre Systolic Blood Pressure Readings**

The change in basal systolic readings pre intervention over all four days is depicted in figure 4.9. The results for systolic blood pressure pre-intervention seemed to increase on Day 2, however during Day 3 and Day 4 the systolic blood pressure was lower than Day 2, which could indicate that the spinal manipulations from Day 2 of intervention could have a long-term effect on blood pressure.

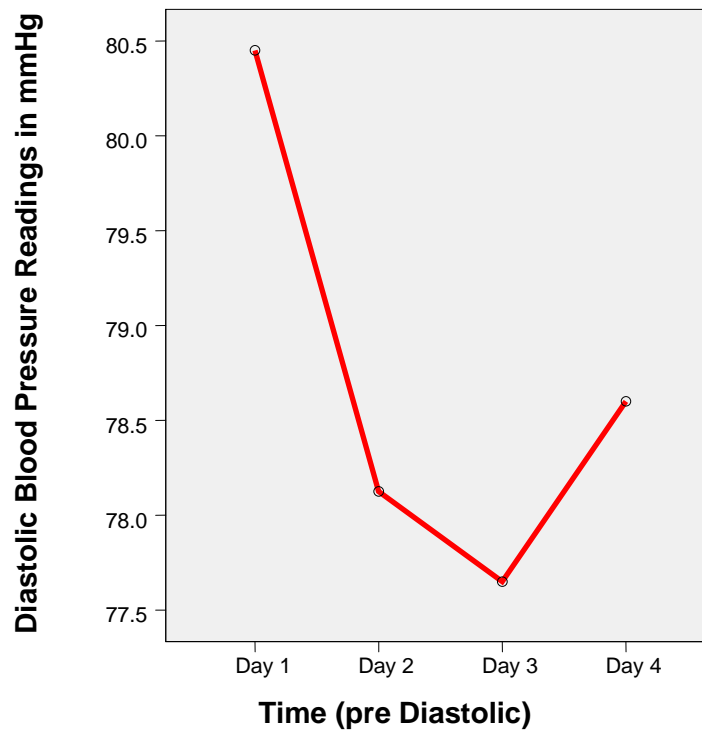
**Table 4.9: The Effect of Interventions on Baseline Systolic Blood Pressure Readings on Each Day**

Effect		Value	F	<i>p</i>
pre Systolic Blood Pressure Readings	Wilks' Lambda	.914	1.16	0.337

Table 4.9 reflects that the effect of the pre intervention on systolic blood pressure readings over the four days is not statistically significant ( $p=0.337$ ).

#### 4.7.2. PRE TEST DIASTOLIC BLOOD PRESSURE READINGS

Figure 4.10 reflects the change in diastolic blood pressure readings over the four days.



**Figure 4.10: Mean Pre Diastolic Blood Pressure Readings**

The change in basal diastolic blood pressure over all four days prior to any intervention is depicted as the lowest on Day 3, following Day 2/  $C_0/C_1$  manipulation.

**Table 4.10: The Effect of Interventions on Pre Diastolic Blood Pressure Readings**

Effect		Value	F	<i>p</i>
Pre Diastolic Blood Pressure Readings	Wilks' Lambda	.765	3.784	0.018

Table 4.10 reflects that the effect of the pre intervention on diastolic blood pressure readings over the four days is statistically significant ( $p=0.018$ ).

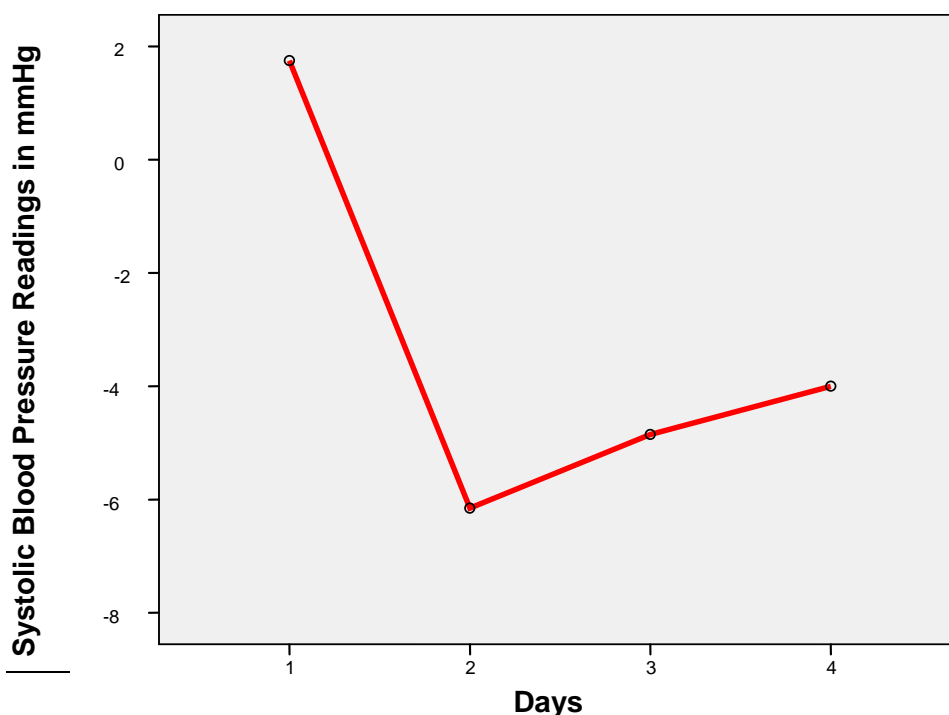


#### 4.8. CHANGE IN MEAN BLOOD PRESSURE READINGS

##### 4.8.1. SYSTOLIC BLOOD PRESSURE READING

##### 4.8.1.1. CHANGE IN MEAN SYSTOLIC BLOOD PRESSURE READING (5 min post – pre)

The change in post intervention reading from the pre intervention reading was calculated for each day by subtracting the pre intervention reading from the five min post intervention reading. The difference in the systolic blood pressure readings reflects the change in reading after each intervention.



**Figure 4.11: Mean Systolic Blood Pressure Readings Change After Five  
Minutes  
by Day of Treatment**

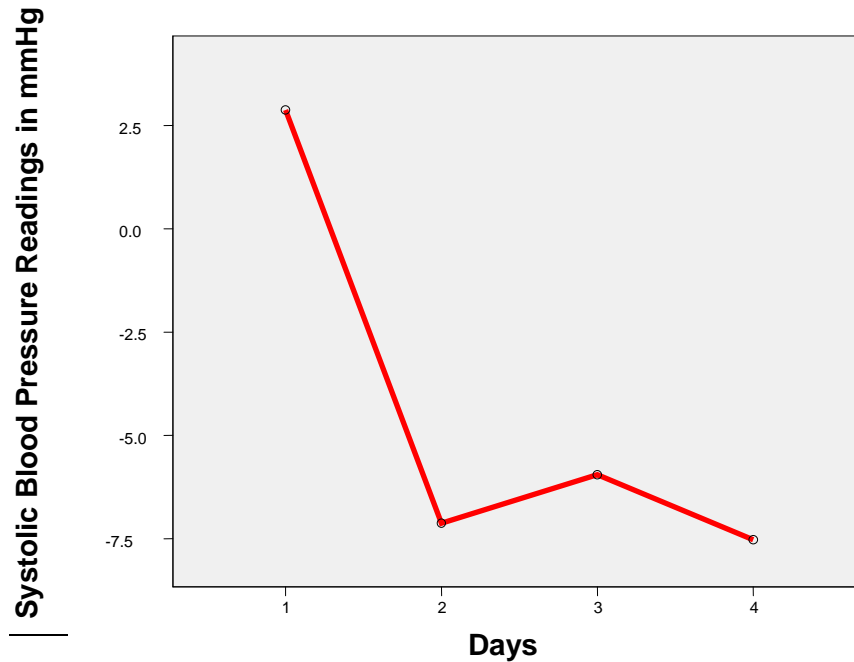
Figure 4.11, shows that the reduction in systolic blood pressure over all four days five minutes post manipulation is greatest at Day 2, following cervical spine manipulation.

**Table 4.11: Repeated Measures ANOVA Testing of the Effect of the Interventions on the Change in Systolic Blood Pressure Readings after Five Minutes on Each Day**

Effect	Statistic	Value	<i>P</i>
Systolic Blood Pressure Readings overall	Wilks' Lambda	0.817	0.056
Day 1 vs 2	F	7.964	0.007
Day 1 vs 3	F	5.781	0.021
Day 1 vs 4	F	3.399	0.073
Day 2 vs 3	F	0.376	0.543
Day 2 vs 4	F	0.894	0.250
Day 3 vs 4	F	0.185	0.669

Table 4.11 shows that there was a marginally significant difference overall between all the days ( $p=0.056$ ) but specifically the systolic blood pressure reading decreased significantly between Day 1 and Day 2 ( $p=0.007$ ) and Day 1 and Day 3 ( $p=0.021$ ). The other days showed no significant change in systolic blood pressure readings. Therefore the interventions on Day 2 and Day 3 lowered the five minute systolic blood pressure readings significantly more than the sham treatment. However, the effects of the different interventions at five minutes post treatment were not significantly different from each other.

#### 4.8.1.2. CHANGE IN MEAN SYSTOLIC BLOOD PRESSURE READINGS (15 min post – pre)



**Figure 4.12: Mean Systolic Blood Pressure Readings Change After 15 Minutes  
by Day of Treatment**

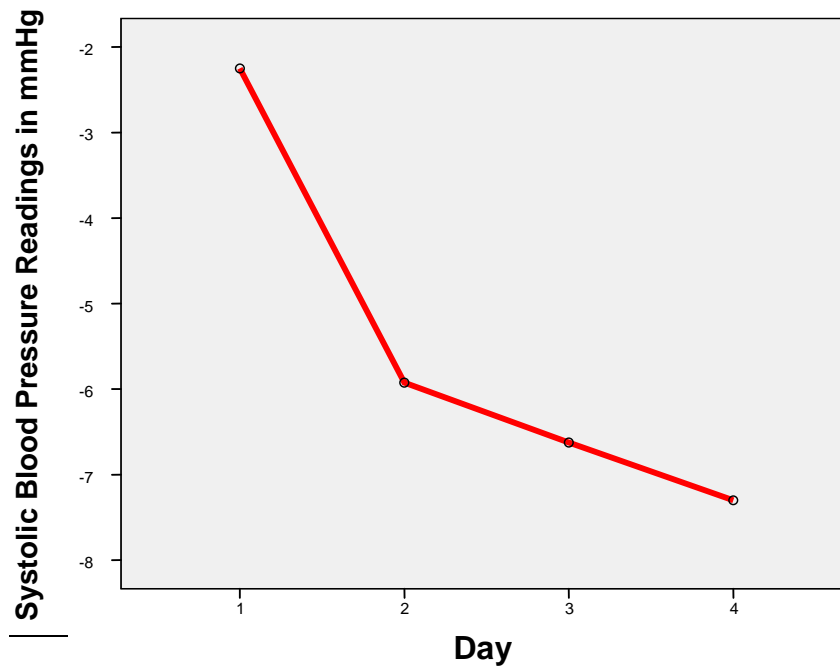
Figure 4.12 depicts the reduction of systolic blood pressure at 15 minutes post intervention to be greatest Day 2 and Day 4.

**Table 4.12: Repeated Measures ANOVA Testing of the Effect of the Interventions on the Change in Systolic Blood Pressure Readings after 15 Minutes on Each Day**

Effect		Value	<i>P</i>
Systolic Blood Pressure Readings overall	Wilks' Lambda	0.693	0.003
Day 1 vs 2	F	10.532	0.002
Day 1 vs 3	F	9.564	0.004
Day 1 vs 4	F	15.740	<0.001
Day 2 vs 3	F	0.176	0.677
Day 2 vs 4	F	0.023	0.879
Day 3 vs 4	F	0.434	0.514

Table 4.12 shows that the overall effect of the interventions were effective at 15 minutes for systolic blood pressure readings ( $p=0.003$ ). Specifically Day 2 intervention reduced the systolic blood pressure readings significantly from the sham treatment ( $p=0.002$ ), as did the intervention on Day 3 ( $p=0.004$ ) and Day 4 ( $p<0.001$ ) compared with the sham. However, the effect of interventions on Day 2, Day 3 and Day 4 did not differ from each other significantly at 15 minutes post treatment.

#### 4.8.1.3. CHANGE IN MEAN SYSTOLIC BLOOD PRESSURE READINGS (30 min post – pre)



**Figure 4.13: Mean Systolic Blood Pressure Reading Change after 30 Minutes  
by Day of Treatment**

Figure 4.13 shows the reduction in systolic blood pressure over all four days of manipulation at 30 minutes of manipulation, is greatest on Day 2 and continues to decrease to Day 4.

**Table 4.13: Repeated Measures ANOVA Testing of the Effect of the Interventions on the Change in Systolic Blood Pressure Readings after 30 Minutes on Each Day**

Effect	Statistic	Value	<i>p</i>
Systolic Blood Pressure Readings overall	Wilks' Lambda	0.923	0.388
Day 1 vs 2	F	1.727	0.197
Day 1 vs 3	F	2.488	0.123
Day 1 vs 4	F	2.964	0.093
Day 2 vs 3	F	0.082	0.777
Day 2 vs 4	F	0.268	0.607
Day 3 vs 4	F	0.096	0.759

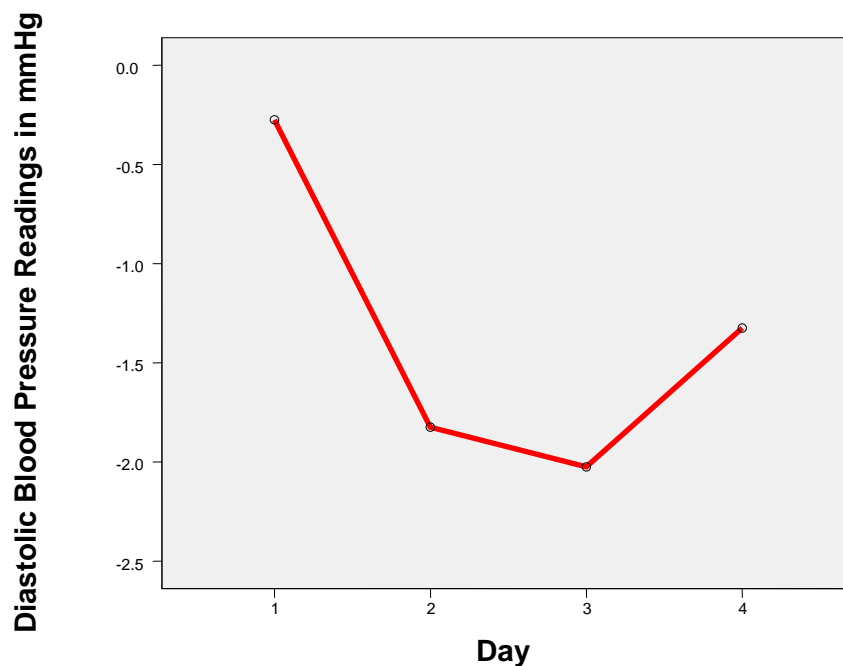
Table 4.13 reflects that the change in systolic blood pressure readings over the four days is not statistically significant overall ( $p>0.05$ ). Moreover, no individual treatment was significantly more beneficial than the sham treatment at Day 1, except for a marginally non significant effect of the treatment on Day 4 ( $p=0.093$ ). None of the individual treatments were better than the other (non with  $p>0.05$ ) at 30 minutes post treatment.

#### 4.8.1.4. CONCLUSION FOR SYSTOLIC BLOOD PRESSURE READINGS

Therefore for the systolic blood pressure reading reduction, the effect was greatest at 15 minutes post intervention and all three interventions had a significantly greater effect than the sham laser, however, the three interventions were not significantly different from each other.

#### 4.8.2 DIASTOLIC BLOOD PRESSURE READING

##### 4.8.2.1. CHANGE IN MEAN DIASTOLIC BLOOD PRESSURE READINGS (5 min post – pre)



**Figure 4.14: Mean Diastolic Blood Pressure Readings Change after Five Minutes  
by Day of Treatment**

Figure 4.14 shows that the scale of changes was insignificant on each day.

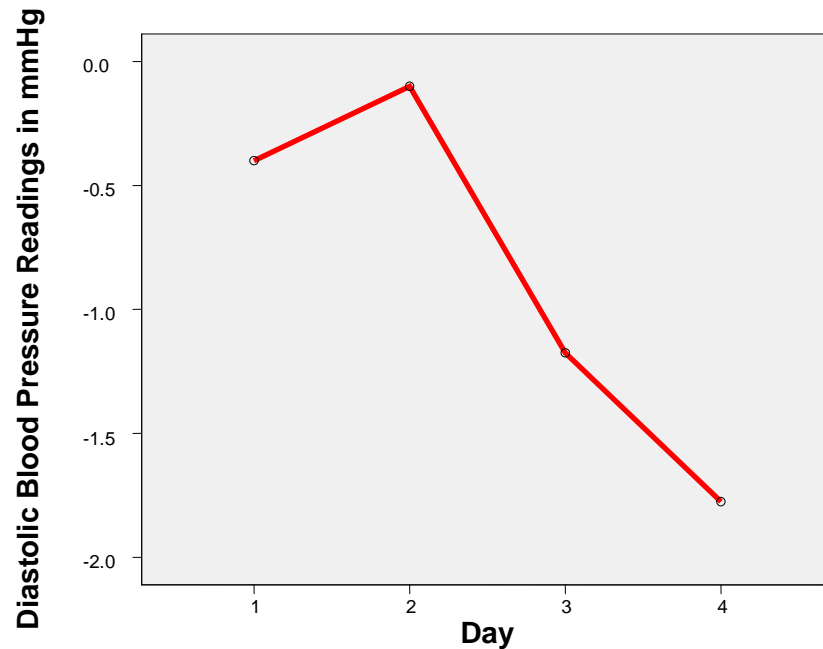
**Table 4.14: Repeated Measures ANOVA Testing of the Effect of the Interventions on the Change in Diastolic Blood Pressure Readings after 5 Minutes on Each Day**

Effect	Statistic	Value	<i>p</i>
Diastolic Blood Pressure Readings overall	Wilks' Lambda	0.968	0.744
Day 1 vs 2	F	0.808	0.374
Day 1 vs 3	F	1.210	0.278
Day 1 vs 4	F	0.240	0.627
Day 2 vs 3	F	0.020	0.889
Day 2 vs 4	F	0.054	0.818
Day 3 vs 4	F	0.150	0.701

It can be seen from Table 4.14 that there was no significant overall difference in the change in diastolic blood pressure readings between the different interventions at five minutes post treatment ( $p=0.744$ ). None of the individual treatments showed a significant difference either (none of the  $p>0.05$ ).



**4.8.2.2. CHANGE IN MEAN DIASTOLIC BLOOD PRESSURE READINGS  
(15 min post – pre)**



**Figure 4.15: Mean Diastolic Blood Pressure Readings Change  
after 15 Minutes by Day of Treatment**

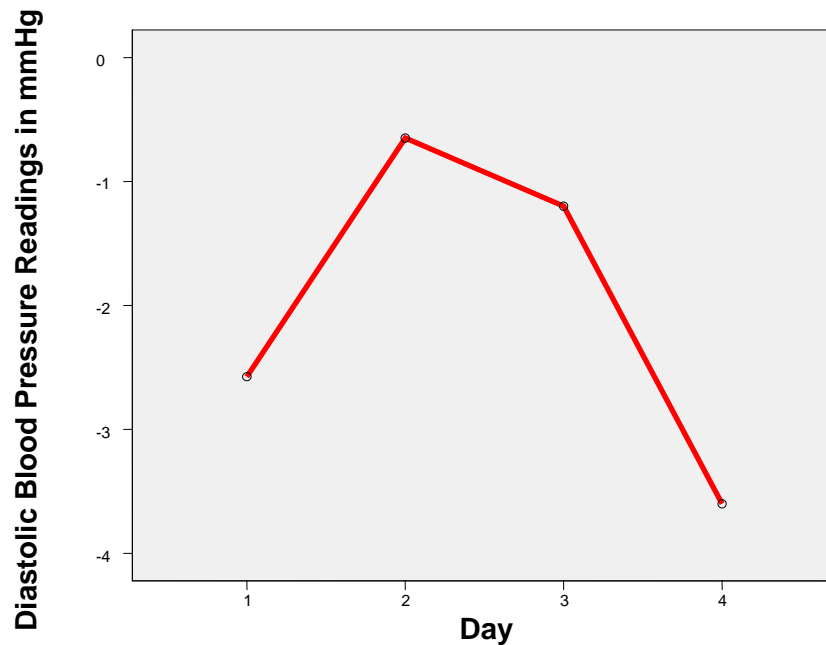
Figure 4.15 depicts the diastolic readings 15 minutes post intervention over all four days, diastolic blood pressure shows to decrease after Day 2 and continues to Day 4, possibly due to the fact that after Day 2 the interventions were manipulations.

**Table 4.15: Repeated Measures ANOVA Testing of the Effect of the Interventions on the Change in Diastolic Blood Pressure Readings after 15 Minutes on Each Day**

Effect	Statistic	Value	<i>p</i>
Diastolic Blood Pressure Readings overall	Wilks' Lambda	0.975	0.812
Day 1 vs 2	F	0.041	0.841
Day 1 vs 3	F	0.165	0.687
Day 1 vs 4	F	0.458	0.503
Day 2 vs 3	F	0.386	0.538
Day 2 vs 4	F	1.005	0.322
Day 3 vs 4	F	0.150	0.700

There was also no significant overall difference in the change in diastolic blood pressure readings between the different interventions at 15 minutes post treatment ( $p=0.812$ ). None of the individual treatments showed a significant difference either (none of the  $p>0.05$ ).

**4.8.2.3. CHANGE IN MEAN DIASTOLIC BLOOD PRESSURE READINGS  
(30 min post – pre)**



**Figure 4.16: Mean Diastolic Blood Pressure Readings Change  
after 30 Minutes by Day of Treatment**

Figure 4.16 shows the changed in diastolic blood pressure over all four days at 30 minutes post intervention. Day 3 shows a decline in diastolic blood pressure which continues to Day 4.

**Table 4.16: Repeated Measures ANOVA Testing of the Effect of the Interventions on the Change in Diastolic Blood Pressure Readings after 30 Minutes on Each Day**

Effect	Statistic	Value	<i>p</i>
Diastolic Blood Pressure Readings overall	Wilks' Lambda	0.918	0.362
Day 1 vs 2	F	1.416	0.241
Day 1 vs 3	F	0.698	0.409
Day 1 vs 4	F	0.224	0.639
Day 2 vs 3	F	0.095	0.760
Day 2 vs 4	F	1.965	0.169
Day 3 vs 4	F	1.626	0.210

Table 4.16 reflects that the change in diastolic blood pressure readings over the four days is not statistically significant overall ( $p>0.05$ ). None of the treatments reduced diastolic blood pressure readings significantly more than the sham laser at 30 minutes post treatment. Also the treatment effects were not different from each other (none of the  $p>0.05$ ).

#### **4.8.2.4 CONCLUSION DIASTOLIC BLOOD PRESSURE READING**

Therefore for diastolic blood pressure readings, none of the interventions had a significantly different effect to the sham laser treatment at five, 15 or 30 minutes post treatment. This outcome does not reject the hypothesis as the systolic reading would be affected far greater than the diastolic reading.

#### **4.9 CONCLUSION TO PRESENTATION OF RESULTS**

It can be concluded from these results that cervical manipulations at C<sub>0</sub>/C<sub>1</sub> reduce systolic blood pressure significantly. Thoracic manipulations at T<sub>1</sub>-T<sub>5</sub> are also significant in reducing systolic blood pressure. The combination of the C<sub>0</sub>/C<sub>1</sub> and T<sub>1</sub>-T<sub>5</sub> manipulations do show a reduction systolic blood pressure, it does however not have a synergistic effect. Manipulation demonstrated an effect on reducing both basal diastolic and systolic blood pressure after manipulation intervention on all days.

## **CHAPTER 5**

### **DISCUSSION**

#### **5.1. INTRODUCTION:**

This chapter encompasses the statistical analysis of the study in chapter 4 and correlates it with the demographic data.

#### **5.2. DEMOGRAPHIC DATA**

All subjects in the study had to be male between the ages of 20 – 35 years, their heart rate had to be within the normal range of 60 – 100 beats per minute (Palatini, 1999; Boon, Colledge, Walker and Hunter, 2006), and also all individuals had to be below a Body Mass Index of 27 (Guyton and Hall, 1997). This was done in order to ensure homogeneity between the subjects and thus allow for a more accurate representation of the results in this population (Mouton, 1996). Furthermore this was done in order to ensure that the effects of disease on blood pressure was limited as the majority of subjects were healthy and physically fit (Guyton and Hall, 1997; Kumar *et al.*, 2003).

Once the group was defined, all subjects were similarly screened for inclusion and exclusion criteria during the recruitment and assessment process. Thereafter all subjects received all four days of intervention according to a set format in order to ensure intervention consistency and to prevent possible variable outcomes with regards to blood pressure due to diurnal or circadian rhythms (Guyton and Hall, 1997; Leach, 2004).

### 5.3. DATA

The data collected was analysed in terms of the effects on systolic blood pressure and diastolic blood pressure. A baseline reading was recorded prior to any interventions and three readings were then recorded post interventions, at five minutes, 15 minutes and 30 minutes post intervention.

SPSS version 15.0 (SPSS Inc., Chicago, Illinois, USA) was used to capture and analyze the data. A p value  $<0.05$  was considered as statistically significant.

To determine the effect of cervical spine manipulations, thoracic spine manipulations and combined cervical and thoracic spine manipulations on blood pressure, repeated measures ANOVA generalized linear models were used. Profile plots were generated to compare the trends visually. Simple and repeated contrasts were used to compare the change in pressure over time between all treatments.

This was done in order to attain the following objectives and evaluate the following hypotheses:

The **first objective** was to determine the effect of sham laser on blood pressure in normotensive males.

Hypothesis One:

There would be no change in the blood pressure of the normotensive males with the sham cervical intervention.

The **second objective** was to determine the effect of cervical manipulation on blood pressure in normotensive males.

Hypothesis Two:

There would be no change in the blood pressure of the normotensive males with the active intervention (cervical manipulation).

The **third objective** was to determine the effect of thoracic manipulation on blood pressure in normotensive males.

Hypothesis Three:

There would be no change in the blood pressure of the normotensive males with the active intervention (thoracic manipulation).

The **fourth objective** was to determine the effect of a combination of a cervical manipulation and thoracic manipulation on blood pressure.

Hypothesis Four:

There would be no change in the blood pressure of the normotensive males with the active intervention (cervical and thoracic manipulation).

The **fifth objective** was to determine the effect on basal blood pressure, following each day of intervention.

Hypothesis Five:

There would be no change in the basal blood pressure of the normotensive males with the active interventions (sham laser, cervical and thoracic manipulation).



## 5.4. COMPARISON OF DAY 1 INTERVENTION

### 5.4.1. DAY 1 INTERVENTION ON SYSTOLIC BLOOD PRESSURE

The statistical data is located in Figure 4.1, as well as Table 4.1.

The data in Figure 4.1 reflects that of systolic blood pressure with regards to sham laser treatment. A slight increase in systolic blood pressure post sham laser is depicted, at five and 15 minutes post intervention, owing to the possible effects of 'treatment anxiety' (Dimmick *et al.*, 2006). This is possible in the context of this research study as the readings for the Day 1 intervention were taken at visit one, where the subject was expected to :

- Undergo a full case history, physical examination and relevant regional examinations in order to participate in the study.
- Read and sign the Information Letter and Informed Consent Form, which formalises the research process more strictly than would be the case if the subjects were to have presented for care outside of a research study (Richardson, 2007).
- Undergo an intervention (sham laser) which all of the subjects were unfamiliar with, therefore predisposing them to concerns about the outcomes of the intervention (Mouton, 1996).

These assertions are supported by the fact that the systolic blood pressure did decrease after 30 minutes, however the effects were not statistically significant (Table 4.1 depicts  $p=0.112$ ); indicating that the subjects were more relaxed with the process that they had been through and more amenable to understanding that the unknown intervention did not produce any adverse side effects.

In addition to the above it should also be considered that it is not known whether the subjects had consumed any stimulants (coffee or coke) or participated in any exercise or smoked or took part in any recreational drugs prior to commencing with their first visit as the subjects were only asked to refrain from such activity at that first

visit. As a result of this it is recommended that future studies consider having a first screening appointment, followed by subsequent visits in which readings are taken, so subjects are warned ahead of time what to avoid to ensure that external influencers to blood pressure can be minimised.

#### **5.4.2. DAY 1 INTERVENTION ON DIASTOLIC BLOOD PRESSURE**

The statistical data is located in Figure 4.2, as well as Table 4.2. The data in Figure 4.2 reflects that of diastolic blood pressure with regards to sham laser treatment.

From the graph it can be seen that the initial readings for the diastolic blood pressure changed minimally (five and 15 minute readings), however after the 30 minute reading the graph depicts a statistically significant ( $p=0.045$ ) decrease of diastolic blood pressure.

This supports the discussion as presented in 5.4.1 and further suggests that 'treatment anxiety' did play a role in these readings.

### **5.5. COMPARISON OF DAY 2 INTERVENTION**

#### **5.5.1. DAY 2 INTERVENTION ON SYSTOLIC BLOOD PRESSURE**

The statistical data is located in Figure 4.3, as well as Table 4.3. The data in Figure 4.3 reflects the effects of C<sub>0</sub>/C<sub>1</sub> spinal manipulation on systolic blood pressure.

Figure 4.3 shows a decline in systolic blood pressure, at the five minutes post intervention reading. The decline was greatest at the 15 minute reading, after which the systolic blood pressure increased at the 30 minute reading.

The overall decrease as reflected in Table 4.3 indicated that the results of C<sub>0</sub>/C<sub>1</sub> manipulations on blood pressure were statistically significant ( $p= 0.005$ ).

These results would seem to suggest that there's a possible significant neurological component to the regulation of blood pressure (Brown *et al.*, 1969; Eldred *et al.*, 1976; Poppele and Quick, 1981; Morgan *et al.*, 1984; Shambaugh, 1987; Edin and Vallbo, 1988; Bergmann, 1992; Slosberg, 1998; Murphy, 1999; Murphy, 2002; Pickar, 2002; Fryer, 2003) and that the stimulation / facilitation of spinal cord segments through manipulation (Sandoz, 1976; Korr, 1979a; Dvorak, 1985; Wyke, 1985; Patterson and Steinmetz, 1986; Gatterman, 1990; Gatterman and Goe, 1990; Mense, 1991; Leach, 2004) is able to effect a response that manifests physiologically within the first 15 minutes post manipulation.

With the increase in blood pressure at the 30 minute reading it suggests that since the subjects are normotensive, that manipulation would have exerted an effect to decrease the blood pressure below what would have been the subjects physiological or homoeostatic norm and therefore the manipulation is reflected in the readings of a temporary decrease in blood pressure, followed by an increase that is mediated through hormonal (Sharma, 1992), capillary fluid shifting (Sharma, 1992; Guyton and Hall, 1997) or kidney related (Guyton and Hall, 1997; Kumar *et al.*, 2003; Boon *et al.*, 2006;) mechanisms (as the neurological mechanism was affected by manipulation). This may then also correlate with the delay in response to the sudden decrease in blood pressure increasing only at the 30 minute reading.

In subjects with true hypertension that is neurologically mediated (Knutson, 2001), subjects may have maintained a decreased blood pressure at the 30 minute reading. However this would only be seen in a population that has neurologically mediated hypertension.

#### **5.5.2. DAY 2 INTERVENTION ON DIASTOLIC BLOOD PRESSURE**

The statistical data is located in Figure 4.4, as well as Table 4.4. The data in Figure 4.4 reflects the effects of C<sub>0</sub>/C<sub>1</sub> spinal manipulation on diastolic blood pressure.

The figure depicts that diastolic blood pressure dropped at the 5 minute reading post C<sub>0</sub>/C<sub>1</sub> manipulation, however at 15 minute reading post manipulation the diastolic

blood pressure had increased, whereas at the 30 minutes reading post manipulation the diastolic blood pressure had once again reduced. Table 4.4 reflects that the intervention on Day 2, on diastolic blood pressure was not statistically significant with ( $p= 0.287$ ).

The possible reasons for this seemingly abnormal response, seems to lie in the following homoeostatic possibility:

**Table 5.1: Comparison of Diastolic and Systolic Readings  
after Manipulation of the Cervical Spine:**

<b>Reading:</b>	<b>Diastolic pressure.</b>	<b>Systolic pressure (See Section 5.5.1).</b>
<b>5 minute reading.</b>	Initial decrease – see Section 5.5.1 for discussion.	Initial decrease.
At this stage the baroreceptors have suddenly decreased sending impulses to the central nervous system (as a result of the decreased systolic pressure). This reflex results in the contraction of the peripheral blood vessels in order to increase the (Sharma, 1992; Guyton and Hall, 1997) fluid shifting into the vascular system (Guyton and Hall, 1997) and activation of the renal angiotension mechanisms (Boon <i>et al.</i> , 2006) in order to increase blood flow to the heart and rectify the systolic pressure.		
<b>15 minute reading.</b>	15 minute increased diastolic pressure results from the sudden increase in blood flow to the heart after activation of the mechanisms above.	Maintains downward trajectory.
An increase in the systolic pressure is affected (Boon <i>et al.</i> , 2006) as more blood is transferred from the atria to the ventricles and into the aorta and carotid arteries (housing the baroreceptors), resulting in a relaxation of the previously baroreceptor activated mechanisms, thus resulting in changed 30 minute readings.		
<b>30 minute reading.</b>	Decrease in the diastolic pressure.	Increase in the systolic pressure.

## 5.6. COMPARISON OF DAY 3 INTERVENTION

### 5.6.1. DAY 3 INTERVENTION ON SYSTOLIC BLOOD PRESSURE

The statistical data is located in Figure 4.5, as well as Table 4.5. The data in Figure 4.5 depicts the results of systolic blood pressure readings following a thoracic manipulation at T<sub>1</sub>-T<sub>5</sub>. The systolic blood pressure reduced significantly at five, 15 and greatest at 30 minute readings post manipulation. Table 4.5 depicts the significant effects ( $p=0.004$ ). Please refer to the discussions after Section 5.6.2.

### 5.6.2. DAY 3 INTERVENTION ON DIASTOLIC BLOOD PRESSURE

The statistical data is located in Figure 4.6, as well as Table 4.6. Figure 4.6 depicts the results of thoracic manipulation on diastolic blood pressure, a reduction in diastolic blood pressure is noted 5 minute reading post manipulation, whereas at the 15 minute reading post manipulation the diastolic blood pressure increases; thereafter maintaining at the same level at the 30 minute post manipulation reading. The  $p$  value in Table 4.6, shows that thoracic manipulations are not statistically significant on diastolic blood pressure ( $p=0.436$ ).

**Table 5.2: Comparison of Diastolic and Systolic Readings  
after Manipulation of the Thoracic Spine:**

Reading:	Diastolic pressure.	Systolic pressure (See Section 5.5.1).
<b>5 minute reading.</b>	Initial decrease – see Section 5.5.1 for discussion.	Initial decrease.
At this stage the baroreceptors have suddenly decreased sending impulses to the central nervous system (as a result of the decreased systolic pressure). This reflex results in the contraction of the peripheral blood vessels in order to increase the (Sharma, 1992; Guyton and Hall, 1997;) fluid shifting into the vascular system (Guyton and Hall, 1997) and activation of the renal angiotension mechanisms (Boon <i>et al.</i> , 2006) in order to increase blood flow to the heart and rectify the systolic pressure.		

**Table 5.2: Comparison of Diastolic and Systolic readings  
after manipulation of the thoracic spine (continued):**

<b>15 minute reading.</b>	15 minute increased diastolic pressure results from the sudden increase in blood flow to the heart after activation of the mechanisms above.	Maintains downward trajectory.
An increase in the systolic pressure is affected (Boon <i>et al.</i> , 2006) as more blood is transferred from the atria to the ventricles and into the aorta and carotid arteries (housing the baroreceptors), resulting in a relaxation of the previously baroreceptor activated mechanisms, thus resulting in changed 30 minute readings.		
<b>30 minute reading.</b>	Maintains after the initial neurological activity “spike”.	Slowing in the decrease of the systolic pressure.

The difference in the cervical spine manipulation (Day 2) versus the thoracic spine manipulation (Day 3) may lie in the fact that there is limited vagal stimulation during the manipulation of the thoracic spine and therefore the degree to which the neurological baroreceptor reflex reacts and initiates a response is not as great, therefore the homoeostatic norms are not approximated as quickly as seen in the cervical spine manipulation group. However, this does not mean that it did not happen and was perhaps only missed because the research method in this study did not have further readings beyond 30 minutes. Therefore it is suggested that future research studies look into having further readings to measure intermediate and long term effects of manipulation as well as to track and establish the time points at which different neurological and / or physiological reflex mechanisms react to the externally imposed neurological influence.

## **5.7. COMPARISON OF DAY 4 INTERVENTION**

### **5.7.1. DAY 4 INTERVENTION ON SYSTOLIC BLOOD PRESSURE**

The statistical data is located in Figure 4.7, as well as Table 4.7. The data in Figure 4.7 depicts the results of systolic blood pressure following a combination of a C<sub>0</sub>/C<sub>1</sub> and T<sub>1</sub>-T<sub>5</sub> spinal manipulations on systolic blood pressure. The systolic blood pressure decreased at the five minute reading and at the 15 minute reading post intervention. However, at the 30 minute reading post intervention, the systolic blood pressure began to increase marginally.

Thus Table 4.7 depicts the statistical significance of the combined manipulations on systolic blood pressure was significant with ( $p=0.001$ ).

Please see discussion after Section 5.7.2.

### **5.7.2. DAY 4 INTERVENTION ON DIASTOLIC BLOOD PRESSURE**

The statistical data is located in Figure 4.8, as well as Table 4.8. Figure 4.8 depicts the decline in diastolic blood pressure following a combination of a C<sub>0</sub>/C<sub>1</sub> and T<sub>1</sub>-T<sub>5</sub> spinal manipulations. The reduction in diastolic blood pressure is greatest at 30 minutes post intervention. However the results of Table 4.8, show that there is an insignificant difference in the changes ( $p= 0.281$ ), indicating that the intervention was unsuccessful in affecting diastolic blood pressure.

**Table 5.3: Comparison of Diastolic and Systolic Readings  
after Manipulation of the Cervical and Thoracic Spine Regions:**

<b>Reading:</b>	<b>Diastolic pressure.</b>	<b>Systolic pressure (See Section 5.5.1).</b>
<b>5 minute reading.</b>	Initial decrease – see Section 5.5.1 for discussion.	Initial decrease.
At this stage the baroreceptors have suddenly decreased sending impulses to the central nervous system (as a result of the decreased systolic pressure). This reflex results in the contraction of the peripheral blood vessels in order to increase the (Sharma, 1992; Guyton and Hall, 1997) fluid shifting into the vascular system (Guyton and Hall, 1997) and activation of the renal angiotension mechanisms (Boon <i>et al.</i> , 2006) in order to increase blood flow to the heart and rectify the systolic pressure.		
<b>15 minute reading.</b>	15 minute decreased diastolic pressure seems to result from the fact that the cervical manipulation induced baroreceptor response (Table 5.2), seems to have been negated by the manipulation of the thoracic spine. This concurs with Korr (1979) and Patterson and Steinmetz (1986) who indicated that there is a possibility of negating or modifying reflex responses by manipulating a second segment. This is based on the theoretical construct that facilitation of spinal cord segment will initiate neurological reflex responses whose pathways will be modified by a superimposed neurological overload (e.g. manipulation).	Maintains downward trajectory.
A decrease in the systolic pressure is therefore affected (Boon <i>et al.</i> , 2006) as the blood transference is not affected (with the atria not receiving increased amounts of blood).		



**Table 5.3: Comparison of Diastolic and Systolic Readings after Manipulation of the Cervical and Thoracic Spine Regions (Continued):**

<b>30 minute reading.</b>	Maintains a downward trajectory, until such time as physiological mechanisms outside of the neurological are able to restore blood flow to the atria.	The slow decrease of the systolic pressure indicates that there is a slow change capillary fluid shift, hormonal and kidney mechanisms.
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## **5.8. COMPARISON OF PRE TEST SYSTOLIC READINGS OVER THE 4 DAYS**

The statistical data is located in Figure 4.9, as well as Table 4.9. The change in basal systolic blood pressure readings pre intervention over all four days is depicted in Figure 4.9. The results were not favorable (statistically insignificant,  $p=0.337$ ), as systolic blood pressure pre-intervention seemed to increase on Day 2. However Day 3 and Day 4 the systolic blood pressure was lower than Day 2, which could indicate that the spinal manipulations from Day 2 of intervention could have a long-term effect on blood pressure.

It would seem that the overriding aspect in the systolic blood pressure is ‘treatment anxiety’ (Dimmick *et al.*, 2006). It is possible in the context of this research study as the pre test readings for the Day 1 could have been influenced by the subject having to:

- Undergo a full case history, physical examination and relevant regional examinations in order to participate in the study.
- Read and sign the Information Letter and Informed Consent Form, which formalises the research process more strictly than would be the case if the subject were to have presented for care outside of a research study (Richardson, 2007).
- Undergo an intervention (sham laser) which all of the subjects were unfamiliar with, therefore predisposing them to concerns about the outcomes of the intervention (Mouton, 1996).

The subjects, who were for the most part naive to manipulation, received their first manipulation / chiropractic intervention on Day 2. Furthermore these same subjects would have been anxious about the outcomes of their perceived treatment from Day 1 and may

have been apprehensive in providing the researcher with the appropriate data (Dimmick *et al.*, 2006; Richardson, 2007).

Therefore in both instances the blood pressure would have been expected to be raised or increasing, until after the subject had realised that they were fulfilling expectations of the research; and that manipulation was not as “bad” as they may have perceived it to be. This would then have resulted in more relaxed subjects after them having overcome the initial anxieties (Richardson, 2007).

To add to this the effects of the manipulation (see Sections 5.5, 5.6 and 5.7) may also have added additional benefits in assisting with the decrease in the blood pressure, thereby facilitating a double decrease in the systolic blood pressure.

#### **5.9. COMPARISON OF PRE TEST DIASTOLIC READINGS OVER THE 4 DAYS**

Basal diastolic readings pre intervention are depicted on Figure 4.10, the figure shows how the reduction in diastolic blood pressure pre intervention over the four days is significant, Table 4.10 has statistically significant ( $p=0.018$ ).

These results reflect as would be expected physiologically (Guyton and Hall, 1997; Boon *et al.*, 2006), as a result of the pattern of response seen in the systolic blood pressure readings in Section 5.8.

#### **5.10. CHANGE IN MEAN SYSTOLIC READING (5 min post – pre)**

Figure 4.11 shows the change in systolic blood pressure five minutes post manipulation on all four days, there is reduction in systolic blood pressure five minutes post intervention on all four days. Table 4.11, calculated by repeated ANOVA testing, shows the significant difference between all four days ( $p=0.056$ ), systolic blood pressure decreases significantly on Day 2 and Day 3, five minutes post intervention.

See discussion after 5.12.

### **5.11. CHANGE IN MEAN SYSTOLIC READING (15 min post – pre)**

Figure 4.12 depicts the systolic blood pressure readings 15 minutes post manipulation on all four days. Day 2 and Day 4 show a reduction in systolic blood pressure 15 minutes post intervention. Table 4.12, calculated by repeated ANOVA testing, shows that the overall effect of the intervention over the four days were effective, with ( $p=0.003$ ). On Day 2 ( $p=0.002$ ), Day 3 ( $p=0.004$ ), Day 4 ( $p<0.001$ ) when compared to the sham laser on Day 1. The greatest reduction in systolic blood pressure occurred at 15 minutes post intervention.

See discussion after 5.12.

### **5.12. CHANGE IN MEAN SYSTOLIC READING (30 min post – pre)**

Figure 4.13, shows the decline in systolic blood pressure 30 minutes post intervention over all four days. The graph depicts a decline in systolic blood pressure over all four days. Table 4.13, calculated by repeated ANOVA testing, shows the reduction in systolic blood pressure 30 minutes post intervention is not statistically significant with an overall p value of ( $p= 0.388$ ).

No individual treatment demonstrated to be significantly beneficial than the sham intervention on Day 1. Day 4 intervention was slightly more beneficial with a ( $p=0.093$ ) value 30 minutes post intervention, however it was still not significant.

From the foregoing changes in the mean systolic blood pressure readings, it is evident that at that the most significant post – pre intervention changes occurred at the Day 2 readings, indicating that the cervical spine manipulation of the subjects caused the greatest degree of change when compared to the sham intervention ( $p=0.007$ ). Similarly, the thoracic manipulation ( $p=0.021$ ) (Day 3) as well as the combined cervical and thoracic manipulations ( $p=0.073$ ) (Day 3) achieved results that were better than the sham intervention (Day 1). Therefore it could be stated that these interventions all attained decreases in the systolic blood pressure that are better than a placebo or sham treatment. This also supports the results from research by Brown, Goodwin and Matthew (1969), Eldred, Hutton and Smith (1976), Poppele and Quick (1981), Morgan, Prochazka and Proske (1984), Shambaugh

(1987), Edin and Vallbo (1988), Bergmann (1992), Sato (1992), Slosberg (1998), Murphy (1999), Pickar (2002), Fryer (2003) and Welch and Boone (2008).

Furthermore, the above results supports the discussion in Tables 5.1, 5.2 and 5.3 in terms of the mechanisms of action employed to achieve these results and also indicates that cervical spine manipulation combined with thoracic spine manipulation may not be associated in significantly reducing systolic blood pressure as there seems to be an antagonistic effect when both are applied within one intervention setting.

These results and conclusions however need to be accepted with caution as the sample size in this study was small and as a result of the fact that the cumulative effect of successive days of intervention were unknown. Therefore it is recommended that future studies look at increasing the sample size and look at four groups – all receiving sham on Day 1 and then receiving either cervical spine manipulation or thoracic spine manipulation or a combination of cervical spine and thoracic spine manipulation or repeat sham on Day 2; to more exactly measure individual intervention effects.

### **5.13. CHANGE IN MEAN DIASTOLIC READING (5 min post – pre)**

Figure 4.14 shows a reduction in diastolic blood pressure over all four days of intervention, Day 3 showed the greatest decline in diastolic blood pressure five minutes post intervention. Table 4.14, calculated by the repeated ANOVA testing, shows all the relevant values at five minutes post intervention over the four days, highlighting a overall insignificant ( $p=0.744$ ) value.

See discussion after 5.15.

#### **5.14. CHANGE IN MEAN DIASTOLIC READING (15 min post – pre)**

The Figure 4.15 shows a reduction in diastolic blood pressure 15 minutes post intervention over the four days, indicating a greatest decline on Day 4, however the  $p$  value calculated by repeated ANOVA testing in Table 4.15 has no significant value with a ( $p=0.812$ ).

See discussion after 5.15.

#### **5.15. CHANGE IN MEAN DIASTOLIC READING (30 min post – pre)**

Figure 4.16 shows the diastolic blood pressure fluctuations over all four days, 30 minutes post intervention. The figure shows a reduction in diastolic blood pressure 30 minutes post intervention, however results in Table 4.16 calculated by repeated ANOVA testing, indicates that they are not statistically significant ( $p=0.362$ ).

From the results it would seem to be evident that at the five minute reading all interventions resulted in a reflex increase in the diastolic blood pressure post intervention indicating that the concomitant decrease in the systolic blood pressure readings (see Tables 5.1, 5.2 and 5.3) elicited a strong baroreceptor response (Sharma, 1992; Guyton and Hall, 1997; Boon *et al.*, 2006). This results in an increase in peripheral vasoconstriction which allowed blood flow to increase into the atria resulting in an increased diastolic blood pressure (Guyton and Hall, 1997; Boon *et al.*, 2006;) at the five minute reading. No significant difference between the interventions was noted other than the fact that all interventions increased the post intervention diastolic blood pressure to a level higher than that of the sham intervention on Day 1.

This is different to the 15 minute readings in that the cervical spine manipulation group had a post intervention diastolic blood pressure reading that was less than the pre intervention reading. This is significant and may be related to the fact that the manipulation of the cervical spine has the greatest influence on the parasympathetic system (Knutson, 2001; Dimmick *et al.*, 2006; Bakris, 2007) and therefore has the ability to assist with the decrease of the diastolic blood pressure more quickly over time through both neurological and physiological mechanisms (Guyton and Hall, 1997; Kumar *et al.*, 2003; Boon *et al.*, 2006),

than any of the other interventions. This compared to the 30 minute reading indicates that this may be possible as the thoracic spine manipulation lagged slightly behind as a result of the fact that the same neurological and physiological mechanisms were stimulated (Guyton and Hall, 1997; Kumar *et al.*, 2003; Boon *et al.*, 2006), but it did not have the same input from the parasympathetic stimulation as seen in the cervical spine manipulation (Knutson, 2001; Dimmick *et al.*, 2006; Bakris, 2007).

In addition the results obtained from the combination group, indicated that the previously suggested antagonistic effect of the two procedures seems to have its most significant effect on the diastolic blood pressure (see Section 5.12).

These results and conclusions however need to be accepted with caution as the sample size in this study was small and as a result of the fact that the cumulative effect of successive days of intervention were unknown. Therefore it is recommended that future studies look at increasing the sample size and look at 4 groups – all receiving sham on Day 1 and then receiving either cervical spine manipulation or thoracic spine manipulation or a combination of cervical spine and thoracic spine manipulation or repeat sham on Day 2; to more exactly measure individual intervention effects.

## 5.16 CONCLUSION

Therefore to conclude the objectives and hypotheses outlined in Chapter One were reviewed in terms of whether or not the stated hypotheses were accepted or rejected.

The **first objective** was to determine the effect of sham laser on blood pressure in normotensive males. Blood pressure was measured five, 15 and 30 minutes post sham laser treatment.

Hypothesis One:

There would be no change in the blood pressure of the normotensive males with the sham intervention.

This hypothesis was **accepted** based on the foregoing results pertaining to systolic blood pressure; however the hypothesis was **rejected** pertaining to diastolic blood pressure.

The **second objective** was to determine the effect of cervical manipulation on blood pressure in normotensive males. Blood pressure was measured five, 15 and 30 minutes post cervical manipulation.

Hypothesis Two:

There would be no change in the blood pressure of the normotensive males with the active intervention (cervical spine manipulation).

This hypothesis was **rejected** based on the foregoing results pertaining to systolic blood pressure; however the hypothesis was **accepted** pertaining to diastolic blood pressure.

The **third objective** was to determine the effect of thoracic manipulation on blood pressure in normotensive males. Blood pressure was measured five, 15 and 30 minutes post thoracic manipulation.

Hypothesis Three:

There would be no change in the blood pressure of the normotensive males with the active intervention (thoracic spine manipulation).

This hypothesis was **rejected** based on the foregoing results pertaining to systolic blood pressure however it was **accepted** pertaining to diastolic blood pressure.

The **fourth objective** was to determine the effect of a combination of a cervical manipulation and thoracic manipulation on blood pressure. Blood pressure was measured five, 15 and 30 minutes post manipulation.

Hypothesis Four:

There would be no change in the blood pressure of the normotensive males with the active intervention (cervical and thoracic spine manipulation).

This hypothesis was ***rejected*** based on the foregoing results pertaining to systolic blood pressure; however the hypothesis was ***accepted*** pertaining to diastolic blood pressure.

The **fifth objective** was to determine the effect on basal blood pressure, following each day of intervention.

Hypothesis Five:

There would be no change in the basal blood pressure of the normotensive males with the active interventions (sham laser, cervical and thoracic manipulation).

This hypothesis was ***accepted*** based on the foregoing results for both systolic and diastolic blood pressure readings.



## **CHAPTER 6**

### **RECOMMENDATIONS AND CONCLUSION**

#### **6.1. CONCLUSION**

The purpose of this study was to determine two main results, whether a combination of an upper cervical spine manipulation and a upper thoracic spine manipulation had a cumulative effect on reducing blood pressure in males between the ages of 20 – 35, as well as determining whether there was an overall reduction in blood pressure following four consecutive days of treatment.

The sample size consisted of forty individuals, recruited on a voluntary basis. All individuals in the study underwent all four days of intervention.

Day 1 : Intervention A, sham laser.

Day 2 : Intervention B, cervical manipulation.

Day 3 : Intervention C, thoracic manipulation.

Day 4 : Intervention D, a combination of intervention B and Intervention C.

The results of this study suggest that blood pressure decreases following a cervical spine manipulation or thoracic spine manipulation; and that a combination of the cervical spine and thoracic spine manipulations did not have a cumulative effect on the reduction of blood pressure.

The results also suggest that the effect of the manipulation is short-term and at 15 minutes the results were still evident, however by 30 minutes they were no longer present.

The treatment over four consecutive days also showed no effect on blood pressure, thus possibly implying the effects are short-term, perhaps this warrants further testing on mild to moderate hypertensive individuals into the long-term effects of manipulation.

## **6.2. RECOMMENDATIONS**

It is recommended that any future studies consider the following.

### **6.2.1. PLACEBO TREATMENT**

The sham laser, intervention A, on Day 1, was to determine the effects of 'treatment anxiety' on blood pressure. This sham treatment should have rather been a sham manipulation in order to establish the subject's response to a manipulative type treatment, rather than a modality. This is particularly true in that the effect of the therapists touch may well have had a beneficial effect (Richardson, 2007) on the patients receiving manipulation, thereby making the results obtained in this study artificially higher than would be expected if the sham had incorporated physically touching the subjects in some manner.

### **6.2.2. SAMPLING**

A larger sample size would have presented with a more reliable representation of the population (Mouton, 1996). In future a comparison between different racial groups as well as females could be incorporated into a study.

### **6.2.3. ACCURACY OF MEASUREMENTS**

All readings of blood pressure and heart rate were recorded using a fully automated digital blood pressure monitor (Microlife BP 3AG1). This unit has been tested according to the British Hypertension Society (2008) and has the highest possible grading for systolic blood pressure and diastolic blood pressure readings. See Chapter Three for the unit's specifications.

The weight and height of each individual was recorded utilizing the scale and height measuring unit at the Durban University of Technology. Body Mass Index was then calculated by the researcher; these measurements are thus subjective in nature and

an automated system (digital weight and height measures) would have been more accurate and are therefore recommended.

#### **6.2.4 FURTHER RESEARCH**

- The effect of mobilization needs to be investigated to establish whether it will have a similar affect to manipulation, as it is perceived to have similar effects on the paraspinal tissues and muscle spindles which were being activated in the same manner as the mechanical stimulus during a manipulation (Korr, 1979; Bergman, 1992; Pickar, 2002).
- As somatovisceral reflexes are stimulated by mechanical input and muscle activation, therefore massage should have an autonomic response and affect blood pressure similar to that of manipulation (Korr, 1979; Bergman, 1992; Pickar, 2002).
- Inclusion of mild to moderate hypertensive patients could show a greater significance in the reduction of blood pressure following a manipulation. The statistical decrease in blood pressure in normotensive patients in this research warrants further research on mild to moderate hypertensive individuals. Future researchers must take into consideration that the subject may be on anti-hypertensive medication and allowance must be made for this during the trial, as the anti-hypertensive medication along with the manipulation may cause a severe reduction in blood pressure (Masarsky *et al.*, 2001).
- A screening visit prior to any intervention treatments is suggested, in order obtain more accurate intervention readings, by educating the subjects on the research requirements, such as avoiding blood pressure stimulates, such as caffeine (Guyton and Hall, 1997; Kumar *et al.*, 2003), that may have an effect on the blood pressure readings at the initial visit.

- A reduction in blood pressure occurred over the four days, thus research into the intermediate and long term effects of manipulation on blood pressure would be justified. This could perhaps monitor different manipulations over several hours to weeks.
- A suggestion of further research is to have separate groups of subjects receiving individual interventions and monitoring the results over several hours, days and weeks (e.g.: only sham laser, only cervical spine manipulations, or only thoracic spine manipulations) therefore monitoring the effects of these individual interventions on blood pressure.

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## **APPENDIX A**

Come in for your  
**FREE**  
spinal assessment  
and blood  
pressure check.

Research on blood pressure is  
currently being carried out at the  
Durban University of Technology  
Chiropractic Day Clinic.

If you are male, non-smoker,  
between the age of 20 - 35,  
please contact

**Angela**  
on  
**(031) 373 2205.**



## **APPENDIX B**

## **Letter of Information and Consent**

### **Title of the Research Study:**

**The effect of cervical and thoracic manipulations on blood pressure in normotensive males.**

### **Principle Investigator:**

Angela Pastellides (031) 373 2205

### **Co-Investigator/s:**

Supervisor: Dr A Docrat. M.Tech: Chiro., CCFC, PG-DIP.U.T.Med.(UWC)  
(031) 373 2589

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

Chiropractic has been proven to lower blood pressure when a neck manipulation is administered. Upper back manipulations have also been shown to reduce blood pressure, thus the purpose of this study is to determine whether a combination of a neck and upper back manipulation has a combined effect on reducing blood pressure.

### **Outline of the Procedures:**

You are requested to sign the informed consent form once you have read the letter of information and are willing to participate in this study

Patients fitting the research criteria will come into the DUT Chiropractic Day Clinic to have a consultation, a patient file will be created and the data stored, once the research data is collected the information will be shredded, the patient's file will remain open for five years. Patients are required to come for treatment over four consecutive days. Patients will have a full physical and neck and upper back orthopedic examination. Before any treatment, an initial blood pressure and heart rate will be recorded in the right arm of the patient; the arm will be relaxed on the desk and taken with a Microlife (Model number: BP- 3AG1) electronic blood pressure machine. On each day of treatment blood pressure will be recorded five, 15 and 30 minutes after treatment.

### **Inclusion criteria:**

Individuals with a systolic reading between 120-140 mmHg and a diastolic reading between 75-90 mmHg.

Healthy males between 20-35 years of age.

Individuals with a resting heart rate of 60-100 beats per minute.

Exclusion criteria:

Individuals on anti-hypertensive medication.

Individuals with a Body Mass Index above 27 kg/m<sup>2</sup>.

Individuals on medication that can affect blood pressure.

Individuals that smoke cigarettes.

Females.

Individuals with a high Body Mass Index

Individuals with contra-indications to manipulation, such as, vertebral artery insufficiency, spondylolisthesis, vertebral malignancy, vertebral disc prolapse causing nerve root entrapment, rheumatoid arthritis, severe osteoporosis, acute vertebral fracture, osteomyelitis, tuberculosis, infective arthritis.

**Risks or Discomforts to the Subject:**

Possible temporary muscle stiffness post manipulation.

Neck and upper back spinal manipulation is a safe treatment however there are risk factors that may predispose you to an adverse reaction. An assessment of your risk profile would be done during the consultation and discussed with you individually. A clinical decision would be made whether treatment would be contra-indicated. If you any further questions, please do not hesitate to ask.

**Benefits:**

Participants will receive a free spinal assessment and physical on initial visit.

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

You are free to withdraw at any stage without any adverse consequences and your future health care will not be compromised.

### Remuneration and Costs of the Study:

Treatment for the duration of the research process will be free of charge. Subjects taking part in the study will not be offered any other form of remuneration for taking part in the study. Normal cost of consultations will be charged for those patients wanting further treatment after completion of the research. All patient information is confidential and the results of the study will be made available in the Durban University of Technology library in the form of a mini-dissertation.

### Confidentiality:

Personal details will be kept in a research file and only the supervisor will have access to them. Should you be interested, the results of the study will be made available in the Durban University of Technology library in the form of a mini-dissertation.

### Research-related Injury:

Contact myself or my supervisor, should you have any further questions please contact Mr Vikesh Singh (031) 373 2701.

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

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

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

Subject's signature:.....

Date:.....

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

Researcher's signature:.....

Date:.....

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

Witness signature: .....

Date:.....

## **APPENDIX C**


**Faculty of Health Sciences**
**ETHICS CLEARANCE CERTIFICATE**

Student Name	Angela Pastellides	Student No	20000664
Ethics Reference Number	FHSEC 051/08	Date of FRC Approval	17/12/2008
Research Title:	<b>The effect of cervical and thoracic spinal manipulations on blood pressure in normotensive males.</b>		

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

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

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

  
 SIGNATURE OF STUDENT/RESEARCHER

04/12/08

DATE

  
 SIGNATURE OF SUPERVISOR/S

04/12/08

DATE

  
 SIGNATURE OF HEAD OF DEPARTMENT

9/12/08

DATE

  
 SIGNATURE: CHAIRPERSON OF RESEARCH ETHICS COMMITTEE

11/12/2008

DATE



## **APPENDIX D**

**DURBAN UNIVERSITY OF TECHNOLOGY**

**CHIROPRACTIC DAY CLINIC**  
**CASE HISTORY**

Patient: \_\_\_\_\_ Date: \_\_\_\_\_  
File # : \_\_\_\_\_ Age: \_\_\_\_\_  
Sex : \_\_\_\_\_ Occupation: \_\_\_\_\_  
Intern : \_\_\_\_\_ Signature: \_\_\_\_\_

**FOR CLINICIANS USE ONLY:**

Initial visit:

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

**Case History:**

Examination:

Previous:

Current:

X-Ray Studies:

Previous:

Current:

Clinical Path. lab:

Previous:

Current:

**CASE STATUS:**

PTT: _____	Signature: _____	Date: _____
------------	------------------	-------------

**CONDITIONAL:**

Reason for Conditional:

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

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

Conditions met in Visit No: \_\_\_\_\_

Signed into PTT: \_\_\_\_\_

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

Case Summary signed off: \_\_\_\_\_

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

### Intern's Case History:

**1. Source of History:**

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

**3. Present Illness:**

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

**4. Other Complaints:**

**5. Past Medical History:**

- ☐ General Health Status
- ☐ Childhood Illnesses
- ☐ Adult Illnesses
- ☐ Psychiatric Illnesses
- ☐ Accidents/Injuries
- ☐ Surgery
- ☐ Hospitalizations

**6. Current health status and life-style:**

- ☐ Allergies
- ☐ Immunizations
- ☐ Screening Tests incl. x-rays
- ☐ Environmental Hazards (Home, School, Work)
- ☐ Exercise and Leisure
- ☐ Sleep Patterns
- ☐ Diet
- ☐ Current Medication
- ☐ Analgesics/week:
- ☐ Tobacco
- ☐ Alcohol
- ☐ Social Drugs

**7. Immediate Family Medical History:**

- ☐ Age
- ☐ Health
- ☐ Cause of Death
- ☐ DM
- ☐ Heart Disease
- ☐ TB
- ☐ Stroke
- ☐ Kidney Disease
- ☐ CA
- ☐ Arthritis
- ☐ Anaemia
- ☐ Headaches
- ☐ Thyroid Disease
- ☐ Epilepsy
- ☐ Mental Illness
- ☐ Alcoholism
- ☐ Drug Addiction
- ☐ Other

**8. Psychosocial history:**

- ☐ Home Situation and daily life
- ☐ Important experiences
- ☐ Religious Beliefs

## **9. Review of Systems:**

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

## **APPENDIX E**

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

**Patient Name :** \_\_\_\_\_ **File no :** \_\_\_\_\_ **Date :** \_\_\_\_\_

**Student :** \_\_\_\_\_ **Signature :** \_\_\_\_\_

**VITALS:**

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

**GENERAL EXAMINATION:**

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

**SYSTEM SPECIFIC EXAMINATION:**

CARDIOVASCULAR EXAMINATION
RESPIRATORY EXAMINATION
ABDOMINAL EXAMINATION
NEUROLOGICAL EXAMINATION
COMMENTS

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

## **APPENDIX F**



**DURBAN UNIVERSITY OF TECHNOLOGY**  
**REGIONAL EXAMINATION - CERVICAL SPINE**

Patient: ..... File No: .....

Date: ..... Student: .....

Clinician: ..... Sign: .....

**OBSERVATION:**

Posture  
 Swellings  
 Scars, discolouration  
 Hair line  
 Body and soft tissue contours

Shoulder position

Left :

Right :

Shoulder dominance (hand):

Facial expression:

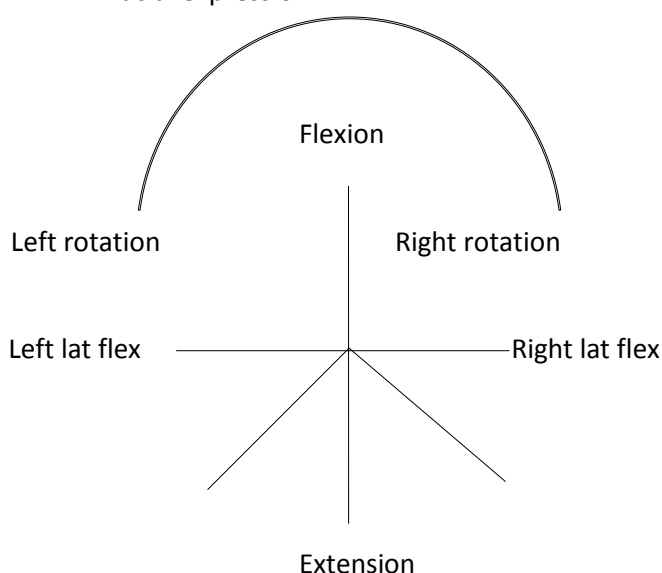
**RANGE OF MOTION:**

Extension ( 70°):

L/R Rotation ( 70°):

L/R Lat flex (45°):

Flexion ( 45°)



**PALPATION:**

Lymph nodes  
 Thyroid Gland  
 Trachea

**ORTHOPAEDIC EXAMINATION:**

Tenderness		Right	Left
Trigger Points:	SCM		
	Scalenii		
	Post Cervicals		
	Trapezius		
	Lev scapular		

	Right	Left		Right	Left
Doorbell sign			Cervical compression		
Kemp's test			Lateral compression		
Cervical distraction			Adson's test		
Halstead's test			Costoclavicular test		
Hyper-abduction test			Eden's test		
Shoulder abduction test			Shoulder compression test		
Dizziness rotation test			Lhermitte's sign		
Brachial plexus test					

**NEUROLOGICAL EXAMINATION:**

Dermatomes	Left	Right	Myotomes	Left	Right	Reflexes	Left	Right
C2			C1			C5		
C3			C2			C6		
C4			C3			C7		
C5			C4					
C6			C5					
C7			C6					
C8			C7					
T1			C8					
			T1					
<b>Cerebellar tests:</b>		Left		Right				
Disdiadochokinesis								

<b>VASCULAR:</b>	Left	Right		Left	Right
Blood pressure			Subclavian arts.		
Carotid arts.			Wallenberg's test		

**MOTION PALPATION & JOINT PLAY:**

Left: Motion Palpation:

Joint Play:

Right: Motion Palpation:

Joint Play:

**BASIC EXAM: SHOULDER:**

Case History:

ROM: Active:

Passive:

RIM:

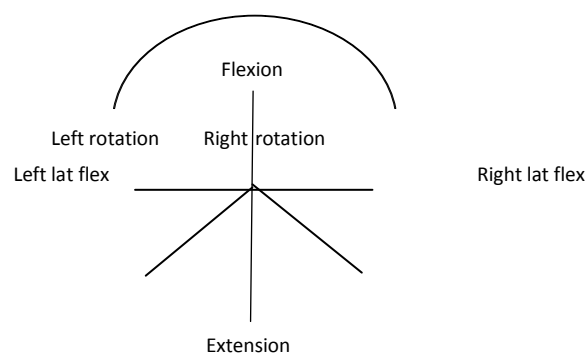
Orthopaedic:

Neuro:

Vascular:

**BASIC EXAM: THORACIC SPINE:**

Case History:



Motion Palpation:	
Orthopaedic:	
Neuro:	
Vascular:	
Observ/Palpation:	
Joint Play:	

## **APPENDIX G**

## THORACIC SPINE REGIONAL EXAMINATION

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

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

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

### **STANDING:**

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

Scars  
Chest deformity

Skyline view – Scoliosis

(pigeon, funnel, barrel)

Spinous Percussion

Breathing (quality, rate, rhythm, effort)

Deep Inspiration

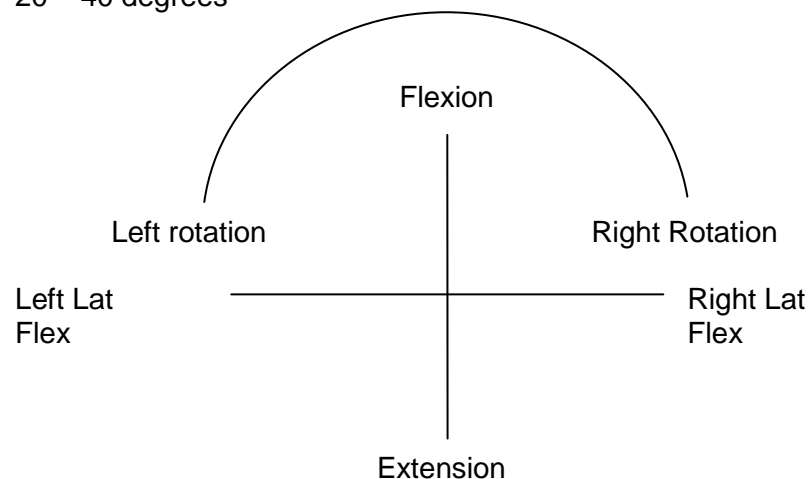
### **RANGE OF MOTION:**

Forward Flexion 20 – 45 degrees (15cm from floor)

Extention 25 – 45 degrees

L/R Rotation 35 – 50 degrees

L/R Lat Flex 20 – 40 degrees



### **RESISTED ISOMETRIC MOVEMENTS:** (in neutral)

Forward Flexion

Extension

L/R Rotation

L/R Lateral Flexion

### **SEATED:**

Palpate Auxillary Lymph Nodes

Palpate Ant/Post Chest Wall

Costo vertebral Expansion (3 – 7cm diff. at 4<sup>th</sup> intercostal space)

Slump Test (Dural Stretch Test)

### **SUPINE:**

Rib Motion (Costo Chondral joints)

SLR

Soto Hall Test (#, Sprains)

Palpate abdomen

**PRONE:**

Passive Scapular Approximation

Facet Joint Challenge

Vertebral Pressure (P-A central unilateral, transverse)

Active myofascial trigger points:

	Latent	Active	Radiation Pattern		Latent	Active	Radiation Pattern
Rhomboid Major				Rhomboid Minor			
Lower Trapezius				Spinalis Thoracic			
Serratus Posterior				Serratus Superior			
Pectoralis Major				Pectoralis Minor			
Quadratus Lumborum							

COMMENTS: \_\_\_\_\_

**NEUROLOGICAL EXAMINATION:**

DERMATOMES												
	T 1	T 2	T 3	T 4	T 5	T 6	T 7	T 8	T 9	T 10	T 11	T 12
Left												
Right												

**Basic LOWER LIMB neuro:**

Myotomes	
Dermatomes	
Reflexes	

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

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

BASIC EXAM	History	ROM	Neuro/Ortho
LUMBAR			
CERVICAL			

## **APPENDIX H**

**Next appointment:**

## **APPENDIX I**



I, Gregory Thiel, give Angela Niky Pastellides permission to utilize the photographs of me in her dissertation.

Sign:\_\_\_\_\_

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