The effect of cranial-sacral membrane and fluid balance technique on the autonomic nervous system using pulse rate, breathing frequency and blood pressure as indicators.

Master thesis by Dr.med. Roman Schneider

March 2006

Abstract

The effect of cranial-sacral membrane and fluid balance technique on the autonomic nervous system using pulse rate, breathing frequency and blood pressure as indicators.

Roman Schneider

Objectives: The hypothesis of this study intends to show that one osteopathic technique is able to balance the ANS, supporting a shift of the sympathetic control into a parasympathetic control. The influence of Osteopathy on the autonomic nervous system (ANS) gives an explanation of its influence on health and disease. Today science as well as the common sense are aware of the role of stress in upsetting the balance of health. Osteopathy should open the skill to directly interpret and influence autonomic activity using perceptual and palpatory skills.

Design: There is no method to measure the activity of the ANS. Therefore the three most common physiological indices, the heart rate, respiratory frequency and blood pressure, are used to show changes in the activity of the ANS.

An osteopathic treatment group and a control group in the state of rest were measured in supine position on arrival (0 minutes), after 5 minutes of rest, after 10 minutes of rest, after the osteopathic treatment or rest of another 5 minutes (after 15 minutes) and after 20 minutes.

The osteopathic treatment was applied only between the 10th and 15th minute of the experiment. Comparison of the results between the group of osteopathic treatment and the group of rest in this time should demonstrate the effect of the cranial-sacral membrane and fluid balance technique.

Subjects: 70 people volunteered as subjects for the following study. The group consisted of 27 men and 43 women between the age of 20 and 67 without clinical pathology of heart, lung and blood pressure. The results of 50 individuals with the impact of osteopathic treatment and 20 subjects observed in the state of rest as control group are presented.

Results: A significant decrease of -18,8% in the mean value of the **heart rate** was caused by the osteopathic treatment compared to a decrease of -5,6% mean in the control group with a difference of 13,2% within 20 minutes. The most significant decrease could be observed within the five minutes of treatment.

The decrease of the **breathing frequency** during twenty minutes in the group of osteopathic treatment is with -24,7% double as high as the decrease of the breathing frequency in the group of rest with

-11,9%. In comparison with the results of the rest group slightly increasing about 2,3% between the 10th and 15th minute the osteopathic treatment group significantly decreases their breathing frequency during the treatment about -15,2% with a total difference of 17,5%.

The results of **blood pressure regulation** shows a decrease of the systolic mean value of the osteopathic treatment subjects with a difference of 9,8% and a decrease of the diastolic mean value with a difference of 5,8% in comparison with the group of rest within 20 minutes. Between the 10th and 15th minute the systolic and diastolic mean values of the rest group raise while the ones of the osteopathic treatment group decrease resulting in a significant difference of 16,8% in the systolic mean value and of 10,3% in the diastolic mean value.

Conclusions: The studies findings point out clearly that there is an effect of the applied osteopathic technique balancing the ANS indicating a shift from the sympathetic to parasympathetic control.

Key words: ANS, heart rate, breathing frequency, blood pressure regulation, cranial membrane and fluid balance technique

Content

1	Introduction1
1.1	Aims and purpose1
1.1.1	Personal interest 1
1.1.2	Context1
1.1.3	Utilization2
1.2	Hypothesis2
2	Fundamentals 4
2.1	Cranial Osteopathy history and development4
2.2	Definitions
2.2.1	CRI Cranial Rhythmic Impulse6
2.2.2	Primary Respiratory Mechanism6
2.3	Anatomical basics of the PRM7
2.3.1	The relevant bone structures7
2.3.2	The connecting membranes9
2.3.3	The cerebrospinal fluid 10
2.4	Physiological basics11
2.4.1	Blood pressure regulation and heart rate11
2.4.1.1	Determinants of the cardiac output12
2.4.1.2	Parasympathetic Activity 12
2.4.1.3	Sympathetic Activity 12
2.4.1.4	Stroke Volume
2.4.1.5	Determinants of the Total Peripheral Resistance (TPR)
2.4.1.6	Baroreceptor Reflex 14
2.4.1.7	Long Term Regulation 16
2.4.1.8	Chemoreceptors

2.4.1.9	9 Antidiuretic Hormone and Atrial Natiuretic Peptide	. 19
2.4.1.′	10 Hypertension and Hypotension	. 19
2.4.2	The Autonomic Nervous System and intrinsic rhythms	. 20
2.4.2.	1 Low-Frequency Oscillations in Blood Pressure	. 20
2.4.2.2	2 Heart rate variability	. 21
2.5	The concept of Cranial Osteopathy	. 21
2.5.1	The inherent mobility of the brain and the spinal cord	. 21
2.5.2	The fluctuation of the cerebrospinal fluid	. 21
2.5.3	The mobility of the intracranial and intraspinal membranes	. 22
2.5.4	The articular mobility of the cranial bones	. 22
2.5.5	Involuntary mobility of the sacrum between the ilia	. 22
3	Models and hypothesis of the Cranial Osteopathy	. 24
3.1	The Sutherland and Magoun model	. 24
3.2	The pressurestat model	. 24
3.3	The muscle reaction model	. 25
3.4	The tissue pressure model	. 25
3.5	The entrainment hypothesis	. 25
4	Methods of treatment in Cranial Osteopathy	. 26
5	The studies technique of treatment	. 27
5.1	Membrane balance technique	. 28
5.2	Fluid balance technique	. 28
6	Empirical study	. 29
6.1	The study design	. 29
6.2	The study methodology	. 32
6.3	The study realisation	. 33
6.4	The study findings	. 35
6.4.1	Individual Rest example	. 35 IV

6.4.2	Individual Osteopathic Treatment example	. 38
6.4.3	Rest Group results	. 41
6.4.4	Osteopathic Treatment Group results	43
6.4.5	Rest and Osteopathic Treatment Group results compared	. 44
7	Discussion and Summary	. 51
7.1	Findings of the study in the context to other Cranial Osteopathy studies	. 51
7.2	Verification of the initial hypothesis	. 54
7.3	Limitations of the study and ideas for future studies	
	in the field of Cranial Osteopathy	55
8	Bibliography	. 57
9	Appendix	60
9.1	Index of tables	60
9.2	Index of figures	61
9.3	Unfiltered data for the analysis	62
9.3.1	Studies single case report	62
9.3.2	Raw data group of Rest (R)	. 63

1 Introduction

1.1 Aims and purpose

1.1.1 Personal interest

My interest in the field of Cranial Osteopathy is strongly driven by my personal practical experiences with this system of gentle treatment. Using cranial techniques my patients respond with great acceptance and interest. I am often asked, how it works and what the principles of osteopathy are. As a therapist I like to explain what I am doing during the treatments.

Inspired from this conversations I was looking for a method, which will give me certain proof about the efficiency of one of my favourite cranial techniques.

Besides my education at the Vienna School of Osteopathy I also graduated as M.D. at the University of Vienna in 2000. Therefore I was always motivated closing the inherent gap of this two systems finding a synthesis.

So I took the chance with this master thesis not only bringing my Msc at the Donau Universität Krems and the D.O. at the Vienna School of Osteopathy to a close, but also researching my personal questions with a basic study on the effect of cranialsacral membrane and fluid balance technique on the autonomic nervous system using pulse rate, breathing frequency and blood pressure as indicators.

1.1.2 Context

In the AAO Journal/19 published winter 1999 James S. Jealous D.O. argues for "Accepting the death of osteopathy: A new beginning". "We all have lived in despair most of our professional lives, watching osteopathy be defiled, degraded, forgotten and turned more and more into an allopathic clone. My goal today is not to degrade but to state the facts that we all know are true. [...] Osteopathy has died, what remains are only an empty skeleton of the dynamic gift we were once given. The essence of osteopathy is gone extinguished. Today we are relating to a ghost, codependently and neurotically fixated upon imitating allopathic medicine. Many believe this illusion to be an evolution for the profession." (Jealous 1999, S.19)

Nevertheless of his concerns for "the clone" Osteopathy it must be possible especially with a holistic view to combine both directions, the traditional principles of Osteopathy and the measuring methods of orthodox medicine. At the Vienna School of Osteopathy many case studies were initiated to reach this goal. Further inspired by the work of Peter Sommerfeld, this study is directly referring to two clinical case studies published at the Vienna School of Osteopathy by Walter Krasser D.O. and Maria Schögler D.O.. Walter Krasser D.O. observed in his dissertation: "The Decrease of High Blood Pressure using a Lateral Fluid Drive Technique with Concentration on the Ossa Temporalia" an average decrease in blood pressure of 10% after his treatments. On the other hand Maria Schögler D.O. could not find a significant effect of her CV4 – technique, a cranial technique, upon blood pressure with threshold hypertensive patients as she mentions in her dissertation.

These different results made a basic study, which deals with healthy individuals having no acute complaints or signs of imbalance of the Autonomic Nervous System (ANS), even more necessary and interesting.

Many of today's complaints such as hypertension, chronic fatigue syndrome, neurasthenia and weakness of the immune system are caused by an imbalance of the ANS. Manual methods as Osteopathy are successfully used in treating such complaints. A part of the osteopathic approach, which deals with utmost subtle phenomena, is the Cranial Concept. It seems to be very effective in balancing the ANS. Not only osteopaths but also other therapeutic approaches recognise a system with clinical relevance in its theoretical and practical sense.

1.1.3 Utilization

This master thesis is written for therapists seeking a reference to the efficiency of Cranial Osteopathy in their professional work or their osteopathic research. This paper is not intending to prove the independent existence of the Cranial Rhythmic Impulse. This paper deals with the effect of one cranial-sacral membrane and fluid balance technique on the ANS. Referring to ethical criteria this master thesis does not claim to be a medical clinical study.

1.2 Hypothesis

The hypothesis of my study intends to show that one osteopathic technique is able to balance the ANS, supporting a shift of the sympathetic control into a parasympathetic control.

There is no method to measure the activity of the ANS. Therefore the three most common physiological indices, the heart rate, respiratory frequency and blood pressure, will be used to show changes in the activity of the ANS.

2 Fundamentals

This chapter will introduce Cranial Osteopathy, its history and development, the definitions of the terms, which are essential in the given study. Furthermore this chapter opens the theme with a short introduction to the anatomical basics, physiological basics and the concept of Cranial Osteopathy needed for the foundation of the studies treatment technique, the methodology and further arguments in this master thesis.

2.1 Cranial Osteopathy history and development

One part of the osteopathic approach, which deals with utmost subtle phenomena, is the Cranial Concept.

Osteopathy has been defined as "a comprehensive system of diagnosis and therapy, based on the interrelationship of anatomy and physiology, for the study, prevention and treatment of disease. The Cranial Concept is osteopathy in its finest ad most fundamental application." (Magoun 1976, p. 1)

The interest in Cranial Osteopathy is constantly rising, several groups of therapeutic approaches and schools have been established since the founder William Garner Sutherland has introduced his idea of the Cranial Concept.

Sutherland himself, the founder of cranial osteopathy, dates back the beginning of his research: "My activities in cranial articular mobility date back to 1899, while I was a student at the American School of Osteopathy, Kirksville, Missouri. The idea originated while I was viewing the disarticulated bones of a skull belonging to doctor Andrew Tailor Still. The articular surfaces of these bones seemed to me to indicate that they were designed for articular mobility.

I found that anatomical texts, while describing the bones thoroughly as to shape, as well as to their external and internal surfaces, had very little to tell concerning the surfaces, which to the student of Osteopathy were the most important of all – the articular surfaces. To gain that knowledge I began with the disarticulated bones, an intensive study on the articular surfaces, with final conclusion that they were truly designed for articular mobility." (Sutherland 1994, p. 17)

His ideas were not well accepted by his colleagues in the first place. "To them, the idea of cranial articular mobility was the dream of an erratic." (Sutherland 1994, p. 18).

Manuscripts were rejected and talks at district, state and even national convention of the American Osteopathic Association in 1932 fell on death ears. However no one can refute results obtained and more and more requests came for treatment of patients and instructions of doctors. From 1940 on the acceptance became much more general. A technical manual was published. In 1944 even one of the osteopathic colleges started a postgraduate teaching program under his direction. which was followed by courses at other osteopathic institutes and elsewhere over the country. Recognition and progress advanced slowly and steadily. In 1946 an official organization was formed, called the Osteopathic Cranial Association. This name was changed to the Cranial Academy in 1960. This is a component society of the Academy of Applied Osteopathy, which is an affiliate of the American Osteopathic Association. It laid down standards for instruction, set up educational and research projects and seriously undertook the creation of a literature. (Magoun 1976, p. xi) Literature like "A Cranial Bowl" by Sutherland 1939, "A Manual of Cranial Technique" by Lippincott & Lippincott 1943, "Osteopathy in the Cranial Field" by Magoun 1966 and "Contributions of Thought. Collective Writings of William Sutherland, D.O." covering the years 1914 – 1954 edited by his wife Adah Strand Sutherland 1971, which also provided a basis for the following study and the creation of this master thesis.

2.2 Definitions

2.2.1 CRI Cranial Rhythmic Impulse

Seven years after Sutherland died, the term CRI was coined by John and Rachel Woods in 1961. Another name for the CRI is the Sutherland wave, named after its discoverer (craniosacral fluctuation was not discovered by Upledger). Empirical studies have found a palpable rate of 6 to 12 cycles per minute in healthy humans, independent of cardiac and diaphragmatic rhythms.

2.2.2 Primary Respiratory Mechanism

About 1924, Sutherland began describing a "primary respiratory mechanism". He proposed that the brain involuntary and rhythmically moves within the skull, causing dilation and contraction of cerebral ventricles. This pumping action of the ventricles generates a pulse wave of cerebrospinal fluid (CSF) that transfers movement to the reciprocal membrane and dural meninges, causing movement from the cranium down to the sacrum. (McPartland 1997, p. 40 - 41)

Magoun describes the term as follows: It will be seen to involve the entire body as a unit of physiological function under the name of the Primary Respiratory Mechanism, which includes the phenomena below.

- a) The inherent motility of the brain and spinal cord.
- b) The fluctuation of the cerebrospinal fluid.
- c) The mobility of the intracranial and intraspinal membranes.
- d) The articular mobility of the cranial bones.
- e) The involuntary mobility of the sacrum between the ilia.

And one might well add, the effect of all these phenomena throughout the entire body by way of all possible channels.

(Magoun 1976, p. 23)

Further detailed explanations to the concept of PRM will be presented in the chapter hypothesis of the Cranial Osteopathy

2.3 Anatomical basics of the PRM

This chapter is meant to give you an overview of the anatomical basis for the so called Primary Respiratory Mechanism: the relevant bone structures, the connecting membranes and the cerebrospinal fluid.

2.3.1 The relevant bone structures

The Occipital Bone is situated at the back part and base of the cranium, is trapezoid in shape and is much curved on itself. It presents at its front and lower part a large oval aperture, foramen magnum (see Fig.01), by which the cranial cavity communicates with the spinal canal. The portion of bone behind this opening is flat and expanded and forms the tabula; the portion in front is a thick, elongated mass of bone, the basilar process; while on either side of the foramen are situated processes bearing the condyles (ibid., 43), by which the bone articulates with the atlas (see Fig.01), the 1st cervical vertebra. These processes are known as the condylar portions. (cp. Pickering & Howden 1988, p. 56)



Fig.01 Occipital Bone (Rohen, Yokochi 1988, p. 29)



Fig.02 Occipital Bone, Atlas and Axis (Rohen, Yokochi 1988, p. 184) The motion of the Occiput is essential in the cranial sacral mechanism. The exact explanation will follow later on.

The Spinal Column is formed by the junction of cervical, dorsal and lumar vertebrae and extends in the sacrum. The Sacrum is a large, triangular bone, situated at the lower part of the vertebral column, and at the upper and



back part of the pelvic cavity, where it is inserted like a wedge between the two innominate bones. Its upper part or base is articulating with the last lumbar vertebra, its apex with the coccyx. (cp. Pickering & Howden 1988, p. 51-52)

Fig.03 Sacrum and Spine (*Rohen, Yokochi 1988, p. 176, 179*)



2.3.2 The connecting membranes

The Dura Mater (lat.: Pachymeninx) is a thick and dense inelastic fibrous membrane which lines the interior of the skull. Its outer surface is rough and fibrillated, and adheres closely to the inner surface of the bones, forming their internal periosteum, this adhesion being most marked opposite the sutures and at the base of the skull. Its inner surface is smooth and lined by a layer of endothelium. It sends four processes inward, into the cavity of the skull, for the support and protection of the different parts of the brain. These four processes are: falx cerebri, the tentorium cerebelli, the falx cerebelli and the diaphragma sellae. (cp. Pickering & Howden 1988, p. 639 - 640)



Fig.04 Dura Mater (Rohen, Yokochi 1988, p. 85)



Fig.05 Falx cerebri and cerebelli (Pernkopf 1963, p. 50)

All this processes except of the diaphragma sellae are connected with the inner surface of the Occiput, forming the venous sinuses. This duplications of folds of the Dura Mater are of great significance to the cranial concept, expressed in the reciprocal tension membrane movement.



Fig.06 Occiput and Spine (Rohen, Yokochi 1988, p. 184) The Dura Mater extends into the spinal canal surrounding the cord like a loose sheath and is separated from the bony walls of the spinal canal by a quantity of loose areolar tissue and a plexus of veins. (cp. Pickering & Howden 1988, p. 707)

The dura is firmly attached at the foramen magnum and on the posterior aspect of the bodies of the second and third cervical vertebrae, but then only lightly until it reaches the level of the second sacral segment. The attachment is here and below again firm.

2.3.3 The cerebrospinal fluid

The cerebrospinal fluid, also called liquor cerbrospinalis, is a transparent, slightly yellowish, watery fluid found in the vetricles, the subarachnoid space and the central canal of the cord. It acts as a support and a buffer for the central nervous system and its vital to its metabolism. It is produced chiefly in the choroid plexus of the four ventricles. The fluid continuity is as follows: lateral ventricles, foramina of Monro, third ventricle, cerebral aqueduct, fourth ventricle, foramen of Magendie and foramina of Luschka, the subarachnoid space of the cord. It escapes by way of the Pacchionian bodies into the venous sinuses, out along the cranial and spinal perineural spaces,

by the way of perivascular spaces and also through the collagen fibres of the fascia into the lymphatic system. (cp. Magoun 1976, p. 19 - 20)



Fig.07 Ventricle System (Rohen, Yokochi 1988, p. 108) The fluctuation of the cerebrospinal fluid is fundamental for the concept of Cranial Osteopathy.

2.4 Physiological basics

In order to explain the impact of the applied technique in the study of this master thesis a short physiological overview of the blood pressure regulation, heart rate and respiratory frequency regulation is needed. Due to the complexity of this topic, this chapter focuses on this three physiological indicators.

2.4.1 Blood pressure regulation and heart rate

The arterial blood pressure is perhaps the most important regulated cardiovascular variable. The blood pressure on the arterial side of the circulation must be maintained at an appropriate level to drive the blood flow through the various organs. The pressure must also be kept from exceeding normal limits to minimize the After load imposed upon the heart. The arterial blood pressure depends upon the contractile properties of the heart, the properties of the vasculature (compliance and vasculature tone) and the blood volume. (McDonagh 2005¹)

A simplified equation by constant blood volume and viscosity and presumed venous pressure ~ 0 mm Hg can be expressed in the formula:

MAP = CO x TPR

Mean arterial blood pressure (MAP) is governed by adjustments to the cardiac output (CO) and the total peripheral resistance (TPR).

2.4.1.1 Determinants of the cardiac output

The determinants of the cardiac output are the heart rate and the stroke volume. The stroke volume is the difference between the diastolic and end systolic volume.

The heart has an inherent rhythm due to the pacemaker activity of the cardiac conduction system. Superimposed on the inherent pacemaker activity are inputs from both arms of the autonomic nervous system, that is both parasympathetic and sympathetic fibres innervate the heart. Increased parasympathetic activity, via the vagus nerve, causes the heart rate to decrease. Increased sympathetic activity causes the rate to increase. (Mc Donagh 2005¹)

2.4.1.2 Parasympathetic Activity

The parasympathetic nerves innervate the heart via the vagus nerve. Parasympathetic fibres terminate in the SA node, the AV node and the atria. The neurotransmitter released by parasympathetic nerve endings is acetylcholine. The primary site of parasympathetic activity is the SA node. In the SA node, increased parasympathetic activity decreases the rate of spontaneous depolarization of the nodal cells to threshold. In the AV node, increased parasympathetic activity decreases nodal excitability, increasing the AV nodal delay. In the atria, increased parasympathetic activity decreases atrial contractility, decreasing atrial contractility. Thus, the effect of increased parasympathetic activity to the heart is to slow the heart rate and modestly decrease atrial contractility. (Mc Donagh 2005¹)

2.4.1.3 Sympathetic Activity

The sympathetic nerves innervate the heart via the left and right stellate ganglia. Fibres from the left stellate ganglion tend to innervate the left ventricle. Fibres from the right stellate ganglion tend to innervate the SA and AV nodes. The neurotransmitter released by the sympathetic cardiac fibres is norepinephrine. The cardiac receptors are known as ß1- Adrenergic receptors. In the SA node, increased sympathetic activity causes an increase in the spontaneous depolarization rate of the nodal cells. In the AV node, the nodal delay is decreased. In the ventricles, increased sympathetic activity tends to increase ventricular contractility. Thus, the effect of increased sympathetic activity to the heart is to increase the heart rate and increase ventricular contractility. (Mc Donagh 2005¹)

2.4.1.4 Stroke Volume

The stroke volume is also effected by the Autonomic Nervous System. An increase in atrial contractility due to sympathetic stimulation will increase diastolic filling. An increase in ventricular contractility due to sympathetic stimulation will increase the stroke volume by increasing the volume ejected which decreases the end systolic volume. (Mc Donagh 2005¹)

2.4.1.5 Determinants of the Total Peripheral Resistance (TPR)

The primary determinant of the TPR is the adjustable arteriolar radius. Local (intrinsic) control is primarily involved in matching organ blood flow to the metabolic needs of that organ. Extrinsic control, via the sympathetic nervous system, is primarily involved in regulation of the mean arterial blood pressure.

The effect of increased sympathetic activity on arteriolar tone depends upon the type of receptor innervated. Sympathetic activity to alpha 1 receptors causes vasoconstriction, but sympathetic activity to beta 2 receptors causes vasodilation. The overall effect of increased sympathetic activity to the peripheral circulation is vasoconstriction and an increase in TPR.

(Mc Donagh 2005¹)



Fig.08 Determinants of Mean Arterial Blood Pressure (Mc Donagh 2005²) Figure 08 shows approximately the complexity of the determinants concerning the regulation of the mean arterial blood pressure.

2.4.1.6 Baroreceptor Reflex

The principal "moment to moment" controller of MAP is the baroreceptor reflex. This reflex represents one of the most exquisite control systems of the body. Pressure sensors monitor arterial blood pressure on a beat to beat basis and feeds back information leading to appropriate changes in the CO and TPR to keep MAP within physiologic limits. The baroreceptor reflex is an example of a feedback control system. Control systems typically consist of a sensor, a feedback system, a central controller and effectors. (cp. Mc Donagh 2005²; Gangong 1993, p. 545; Schmidt et al. 1990, p. 540)

Physiologic control systems work in a similar manner, that is with a sensor, controller and effectors. For the baroreceptor reflex, blood pressure sensors (called baroreceptors) are strategically located in the arch of the aorta and in the carotid sinus. These sensors are mechanoreceptors, sensitive to stretch or distension of the arteries (as occurs when the blood pressure increases). When the arterial blood pressure increases, the receptors are stretched and neural output increases to the control centre in the brainstem. The baroreceptors are sense the mean arterial blood pressure, but are especially responsive to changes in arterial pressure. Afferents to the Cardiovascular Control Centre. The controller, located in the brainstem, is called the cardiovascular control centre. Afferent activity to the control centre, from the carotid sinus baroreceptors is carried via the carotid sinus nerve, which joins the glossopharyngeal nerve. Afferent activity to the brainstem from the aortic arch baroreceptors is carried via the vagus nerve. The control centres are located in the reticular formations of the medulla and in the lower third of the pons. The afferent input is integrated in the nucleus tractus solitarius (NTS), which directs activity to several cardiovascular centres. The specific cardiovascular brainstem centres are:

- The vasoconstrictor centre is located in the upper medulla and the lower pons.
 Efferent neurons are sympathetic and synapse in the spinal cord. The efferent fibres then travel to sympathetic ganglia and finally to the target organs, producing arteriolar and venous constriction.
- In the cardiac accelerator centre efferent sympathetic neurons also synapse in the cord, then go to ganglia, then to the heart (SA node, AV node and both ventricles). Increased activity causes an increase in heart rate and an increase in ventricular contractility.
- Parasympathetic fibres from the cardiac decelerator centre travel in the vagus to the heart. Increased efferent activity decreases heart rate and weakly reduces atrial contractility.

The effectors are the prime determinants of CO and TPR. Increased sympathetic activity tends to increase both the CO and the TPR by the mechanisms already described. Increased parasympathetic activity to the heart tends to decrease heart rate and atrial contractility, decreasing the CO.

(cp. Mc Donagh 2005²; Gangong 1993, p. 545; Schmidt et al. 1990, p. 540)



Fig.09 The baroreceptor reflex (Mc Donagh 2005²)

2.4.1.7 Long Term Regulation

In addition to moment to moment changes in the CO and TPR, the arterial blood pressure is chronically affected by the extracellular fluid (ECF) volume, which is in an equilibrium with blood volume. Increased ECF tends to increase blood pressure throughout the cardiovascular system. The blood volume is regulated by the reninangiotensin system. This system is normally activated by a decrease in MAP. (Mc Donagh 2005²)



Fig.10 Renin – Angiotensin II – Aldosterone System (Mc Donagh 2005²)

As shown in Figure 10 a decrease in MAP tends to decrease renal arterial pressure. The decrease in pressure is sensed by mechanoreceptors in the renal afferent arterioles causing prorenin to be converted to renin by the juxtaglomerular cells. Renin secretion is also increased by sympathetic stimulation and by Beta 2 agonists, such as isoproterenol. Renin secretion is decreased by Beta 2 antagonists, such as propranolol. Renin, released into the plasma, catalyzes the conversion of angiotensinogen to angiotensin I. In the lungs and kidneys, angiotensin I is converted to angiotensin II by angiotensin converting enzyme (ACE). (cp. Mc Donagh 2005²; Schmidt et al. 1990, p. 545-546, Gangong 1993, p. 416)

2.4.1.8 Chemoreceptors

Peripheral chemoreceptors, particularly sensitive to dissolved oxygen, are located in the carotid body, near the bifurcation of the common carotid arteries and in the aortic bodies located along the aortic arch. When the arterial concentration of oxygen decreases, information is transmitted to the central cardiovascular control centre in the medulla. The cardiovascular response is generally to increase CO and TPR in an attempt to improve oxygen delivery to the receptors.

Central chemoreceptors are also located within the medulla. These sensors are particularly responsive to changes in pH and PCO2. A decrease in medullary blood flow will lead to a build up of CO2 and H+, which will tend to cause a decrease in medullary pH. The response is an increase in sympathetic activity and parasympathetic activity. The sympathetic activity tends to increase the CO and TPR, while the parasympathetic activity tends to lower heart rate.

(cp. Mc Donagh 2005²; Schmidt et al. 1990, p. 544)

Ganong (1993) notes that chemoreceptor in the carotid and aortic bodies discharge may also contribute to the production of Mayer waves. These should not be confused with Traube-Hering waves, which are fluctuations in blood pressure synchronized with respiration. The Mayer waves are slow regular oscillations in arterial pressure that occur at the rate of about one per 20-40 seconds during hypotension. Under these conditions hypoxia stimulates the chemoreceptors. The stimulation rises the blood pressure, which improves the blood flow in the receptor organs and eliminates the stimulus to the chemoreceptors, so that the pressure falls and a new cycle is initiated. However, Mayer waves are reduced but not abolished by chemoreceptor denervation and are sometimes present in spinal animals, so oscillation in spinal vasopressor reflexes is also involved. (p. 548) Schmidt et al. (1990) presume that Mayer waves are due to fluctuations of the peripheral vessel tonus with a period duration of 6-20 seconds with a most frequent middle duration of 10 seconds (p. 555) supported by the an article of Karemaker (1999).

2.4.1.9 Antidiuretic Hormone and Atrial Natiuretic Peptide

Antidiuretic Hormone (ADH) is a hormone produced in the pituitary gland. ADH is involved in regulation of extracellular fluid osmolarity and blood volume regulation. ADH secretion is stimulated by an increase in plasma osmolarity or a decrease in blood pressure. There are two ADH receptors, V1 and V2. The V1 receptors are located in vascular smooth muscle cells. Activation of the V1 receptor causes smooth muscle contraction, vasoconstriction and increased TPR. Activation of the V2 receptor, located in the renal collecting ducts, causes reabsorption of water (tending to increase extracellular water and "dilute" the increase in osmolarity that caused V2 receptor activation. (Mc Donagh 2005²; Gangong 1993, p. 218 et seqq.)

Atrial Natiuretic Peptide (ANP) is secreted by the atria in response to an increase in ECF volume. The response is vasodilation and an increase in sodium and water excretion. (Mc Donagh 2005²)

2.4.1.10 Hypertension and Hypotension

Blood pressure is a continuous variable, and risks of various adverse outcomes rise with it. Normal blood pressure is 120/80 mmHg. Hypertension is usually diagnosed on finding blood pressure above 140/90 mmHg measured on both arms on three occasions over a few weeks. Recently, the JNC VII (The Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure) has defined blood pressure over 120/80 mmHg and below 140/90 mmHg as "pre-hypertension". "Prehypertension is not a disease category. Rather, it is a designation chosen to identify individuals at high risk of developing hypertension (JNC VII)." (Farlex 2005).

For most individuals, a healthy blood pressure lies between 90/60 mmHg to 130/80 mmHg. A small drop in blood pressure, even as little as 20 mmHg, can result in transient hypotension. Orthostatic hypotension is a common cause of low blood pressure, resulting from a sudden change in body position. Reduced blood volume, called hypovolemia, can also cause hypotension and often results from the use of diuretics or vasodilators such as nitric oxide or furosemide. Another - albeit rare - form, is "post prandial hypotension", which occurs 30-75 minutes after eating. It is not well understood. (Wikipedia 2005)

2.4.2 The Autonomic Nervous System and intrinsic rhythms

Blood pressure (BP) and heart rate (HR) are continually varying. When Fourier analysis (a branch of mathematics which studies the representation of functions or signals as the superposition of basic waves) was applied to analysis of BP variability (BPV) and HR variability (HRV), two frequency peaks stood out: one around the respiratory frequency and one around 0.1 Hz, or one oscillation in 10 s. (Karemaker 1999)

These frequencies had been observed in blood pressure recordings before, actually over 130 years ago: Traube-Hering waves (coupled to respiration) and Mayer waves as also mentioned by Ganong in the chapter chemoreceptors. In the earlier research it had been established that oscillating sympathetic activity causes the Mayer waves in blood pressure. The respiration-coupled blood pressure oscillations were partly explained by mechanical effects of respiration and possibly by the vagally induced heart period oscillations coupled to respiration, known as respiratory sinus arrhythmia (RSA). (cp. Karemaker 1999)

Due to the easy availability of ECG recordings, heart rate oscillations have extensively been studied, much more so than blood pressure oscillations. In this field of research the idea was put forward that the 0.1 Hz oscillation in HRV, also called low frequency or LF, might be used as indicator of sympathetic activity. The respiratory (high frequency or HF) frequency in HR must be due to vagal activity, the vagus nerve being the only one that can make HRV follow at the respiratory rate. (Karemaker 1999)

2.4.2.1 Low-Frequency Oscillations in Blood Pressure

Arterial pressure Mayer waves, occurring at an interval of about 10 seconds or 0.10 Hz in humans, are presumed to result from rhythmic sympathetic vasomotor activity. RR interval oscillations at this same frequency are mediated by both cardiac sympathetic an cardiac vagal outflows and are thought to represent arterial barorflex responses to pressure oscillations. Data demonstrate that in supine humans, low-frequency RR interval oscillations follow but do not dampen arterial pressure oscillations; elimination of RR interval variability does not increase low-frequency arterial pressure oscillations. When vascular sympathetic outflow was increased by 40° tilt, elimination of low-frequency RR interval variability augmented the diastolic pressure Mayer waves. (Tayler et al. 1996, p. 1530)

2.4.2.2 Heart rate variability

Low-Frequency (LF) and High-Frequency (HF) can increase under different conditions. An increased LF is observed during 90° tilt, standing, mental stress, and moderate exercise in healthy subjects. (...) Conversely an increase in HF is induced by controlled respiration, cold stimulation of the face, and rotational stimuli. Vagal activity is the major contributor to the HF component. Disagreement exists in respect to the LF component. Some studies suggest that LF, when expressed in normalized units, is a quantitative marker of sympathetic modulations; other studies view LF as reflecting both sympathetic activity and vagal activity. Consequently, the LF/HF ratio is considered by some investigators to mirror sympathovagal balance or to reflect the sympathetic modulations. (American Heart Association 1996, p. 1053)

2.5 The concept of Cranial Osteopathy

On the basis of the definition of Primary Respiratory Mechanism by Magoun this chapter intends to give a comprehensive overview to the concept of Cranial Osteopathy.

2.5.1 The inherent mobility of the brain and the spinal cord

Magoun states, that the inherent mobility of the cranial bones is a pulsation which is synchronous with the cardiac contraction. Further it is a pulsation which coincides with respiratory pressure changes associated with inhalation and exhalation. Thirdly he assumes, it is a wave not related to either heart rate or respiration but one which constantly maintains its own cycle. And it is still an undulating pulsation which has not been identified. (cp. Magoun, 1976, p. 23-24)

2.5.2 The fluctuation of the cerebrospinal fluid

He also assumes that the fluctuation of the cereprospinal fluid is due to the rhythmical contraction of the oligodendroglial cells of the neuroglia in the central nervous system referring to the findings of Woolley and Shaw of the Rockefeller Institute for Medical research and many other scientific studies of his time. (cp. 1976, p. 24)

2.5.3 The mobility of the intracranial and intraspinal membranes

The detailed anatomy has been described in the chapter anatomical basics of the PRM. Magoun mentions also the connection between the falx cerebelli and the intraspinal membrane below, which forms the all-important link between the membranous articular mechanism of the cranium and the sacrum. It is in the form of a tube surrounding the spinal cord. This is a possible explanation for the continuation of the Cranial Rythmic Impulse form the brain to the sacrum. (cp. Magoun 1976, p. 29)

In the context of mobility of the the intracranial and intraspinal membranes Magoun points at the importance of the connection between the membranous structures in the skull.

"The falx cerebri and tentorium cerebelli should be thought of as three sickle-shaped agencies, all of which arise from a common origin at the straight sinus, named "The Sutherland Fulcrum" in honour of its discoverer, and have their secondary insertion into the various bones of the cranium." (Magoun 1976, p. 27)

Due to the Sutherland Fulcrum it is possible to balance the membranes of the brain by using the occiput as a lever, like in the technique applied in the studies treatment.

2.5.4 The articular mobility of the cranial bones

Throughout life a very light degree of mobility can be found in the sutures of the skull, which are serrated or interdigitated with an intervening connective tissue allowing motion. (cp. Magoun 1976, p. 32) According to McPartland referring to Retzlaff modern research corroborates Sutherlands hypothesis. Histological studies have shown that most cranial sutures do not ossify at any age but remain supple with ligaments, blood vessels and nerves; hence motion at sutures is possible. (1997, p. 41) This anatomic fact allows the palpation of the CRI and is the basis of cranial treatments.

2.5.5 Involuntary mobility of the sacrum between the ilia

The pelvic ligaments allow the sacrum to swing in an arc without materially changing their tension, the movement occurring synchronously with that of the rest of the craniosacral mechanism. (Magoun 1976, p. 34)



Fig.11 Reciprocal tension membrane movement in flexion (Magoun 1976, p. 37) The reciprocal tension membrane movement model summarizes the connections mentioned above. According to Magoun the cranial rhythmic impulse manifests as inhalation or flexion and exhalation or extension (cp. 1976, p. 38).

"All parts operate as unit of reciprocating function including the spinal reciprocal tension membrane. This is sometimes referred as the "core link" connecting the articular mechanism of the cranium with the sacrum to coordinate action." (Magoun 1976, p. 38)

3 Models and hypothesis of the Cranial Osteopathy

Starting from the concept of the primary respiratory mechanism by Sutherland and Magoun other explanations have been discussed.

3.1 The Sutherland and Magoun model

Sutherland and Magoun proposed that the cranial rhythmic impulse is due to the rhythmical contraction of the oligodendroglial cells of the neuroglia in the central nervous system. The PRM is considered to include the innate motility of the central nervous system manifesting a circularly coiling and uncoiling of the neural tube. Furthermore it is assumed that this movement is like a dynamo due to the collecting and discharging electromagnetic field of the cells. (cp. Magoun 1976, p. 35; McPartland 1997, p. 41)

3.2 The pressurestat model

Upledger doubts the concept of Sutherland – the brain is the driving force of the CRI – because for him the brain is not able to form a hydraulic pump strong enough to rise and lower the hydrostatic pressure within this partly closed system. Further he states that the rhythmic movement of the oligodendroglial cells in vitro is not even one tenth of the frequency we are able to observe in the PRM. Although he mentions the different conditions for oligodendroglial cells in vitro he created another explanation – his pressurestat model.

The pressurestat model is based on the hypothesis that temporarily more cerebrospinal fluid is produced than can be absorbed by the glandulae pacchioni or arachnoid villi resulting in fluctuation of volume and pressure of the CSF. He assumes a regulatory mechanism which turns the production of liquor on when the pressure goes down and turns it off as soon as the pressure has reached its set point. He offers two explanations: firstly a neuro-vegetative reflex connecting the proprioceptors within the sutures with the plexus choroideus where one finds the main production of CSF, secondly a valve like mechanism within the straight sinus.

(cp. Upledger 1994, p. 22-23)

Despite experimental results that seem to support Upledger's model, the basic approach of the theory is inconsistent with the generally accepted Monroe-Kellie-Hypothesis. This hypothesis postulates that changes of intracranial pressure are mainly regulated by the exchange of fluids between three compartments: brain, CSF and blood. (Sommerfeld 2001, p. 26)

3.3 The muscle reaction model

The muscle reaction model, also called Beckers model, assumes that the CRI is due to a tonic reaction of the postural muscles to gravity. Either the muscles send a stimulus to the central-nervous system causing fluctuations in the liquor pressure or the postural muscles could affect the dural membrane directly via the continuation of the fascia changing the pressure rhythmically by altering their tension. This model was disproved by Upledger who found a stable CRI in patients who had injuries in the spine resulting in atonic paresis and therefore no activity of skeletal muscles. (cp. Upledger 1994, p. 24)

3.4 The tissue pressure model

A tissue pressure model was developed by Norton to provide a possible physiologic basis for the manifestation of the CRI. The model assumes that the CRI is related to activation of slowly adapting cutaneous mechanoreceptors by tissue pressures of both the examiner and the subject, and that the sources of change in these tissue pressures are the combined respiratory and cardiovascular rhythms of both examiner and subject. A significant correlation was found between frequencies calculated from the model and published values for CRI obtained by palpation. These comparisons suggest that the CRI may arise in soft tissues and represents a complex interaction of at least four different physiologic rhythms. (cp. Norton 1991, p. 975)

3.5 The entrainment hypothesis

Entrainment is a integration or harmonization of oscillators. All organisms pulsate with myriad electrical and mechanical rhythms. (McPartland 1997, p. 40) McPartland hypothesizes the CRI is the perception of entrainment, a palpable harmonic frequency of multiple biological oscillators. (1997, p. 42)

The oscillators include: cardiac pulse, heart rate variability, Traube-Hering modulation, diaphragmatic excursion, contractile lymphatic vessels, CSF production by the choroids plexus, pulsating glial cells, electrical fields generated by cortical

neurons, cortical oxidative metabolism and probably many other oscillators. This rhythms, except those by the brain, can be easily transformed into tissue movement and can be perceived as CRI. (McPartland 1997, p. 42)

It is a known factor that rhythms like pulse rate, heart rate variability, Traube-Hering wave are strongly influenced by the ANS as mentioned in the chapter before, the autonomic nervous system and intrinsic rhythms. Especially the oscillations in the heart rate variability describes a low frequency which might be used as indicator for the sympathetic activity and a high frequency in heart rate which is due to vagal activity.

McPartland assumes if there is balance within the autonomic nervous system than the bodies many rhythms harmonize into a strong, coordinated, sinusoidally fluctuating entrainment frequency, which can be palpated as strong healthy CRI. (1997, p. 42)

The entrainment hypothesis opens a framework which allows explanations to the phenomenon that scholars like Becker and Jealous found rhythms of different frequencies. Becker described the slow tide which oscillates with a frequency of 0.6 cycles per minute. Jealous calls Beckers rhythm long tide and his discovery 2 ½ cycles per minute rate. He describes the 2 ½ cycles per minute as an integration of the information of a patients whole system. (cp. McPartland 1997, p. 43)

In the entrainment hypothesis there is no contradiction of finding different cranial rhythms, because a variety of integrations of oscillators is possible.

4 Methods of treatment in Cranial Osteopathy

The methods of treatment in Cranial Osteopathy were widely criticized by scholars rooted in orthodox medicine lacking reproducibility of results by different osteopaths. Even the study to inter- and intraexaminer reliability in palpation of the PRM by Sommerfeld (2001) could not lead to a satisfying explanation.

He suggests what is now identified as the PRM, is a certain combination of rhythmic phenomena (referring to the entrainment hypothesis and heart rate variability) coming from the examiner and the patient. (cp. 2001, p. 83)

CRI could be seen as an indicator for the harmony of the autonomic nervous system and all oscillating systems in the body. The intersubjective exchange of rhythms and information between practitioner and patient – calling for "thinking, seeing, feeling, knowing fingers" (Magoun 1976, p. 87) – could be the basis allowing a self healing process to take place. The perception of a patient, the acceptance of his person as a whole and the recognition of his concerns on a subtle level like the CRI induces very often a relaxation on a profound level. This three attitudes are the basis of the technique described in the next chapters.

5 The studies technique of treatment

The technique used in the following study is called a cranial-sacral membrane and fluid balance technique.

In this technique the practitioner places one hand under the occiput and the other hand under the sacrum. The patient is in a supine position, laying on the practitioners hands. (cp. Upledger 1994, p. 86-87)



Fig.12 Position of the hands to balance sacrum and occiput in supine position (Upledger 1994, p. 97)

This technique was chosen, because it allows to balance osteopathic lesions in the spine and occiput and restore a continuous cranial flow.

5.1 Membrane balance technique

By placing one hand under the occiput the focus lies on:

- 1. loosening muscular tension between the occiput and the first and second cervical vertebra.
- 2. the free movement of the sutura lambdoidea and sutura occipito-mastoidea
- using the occiput to get in touch with the tentorium and the rest of the dural meninges according to the reciprocal tension membrane with special attention to the Sutherland fulcrum
- 4. following the reciprocal tension membrane to the sacrum where the dura is firmly attached
- 5. establishing a free movement between occiput and sacrum balancing possible distortions from one end of the system to the other.

Following this schedule the whole system seems to settle in a position of very relaxed balance. (cp. Upledger 1994, p. 97)

5.2 Fluid balance technique

From this state of relaxed balance, mentioned before, it is possible to enter another level almost like through a gate, called the fluid field. In this state of perception one does not attune to anatomy and applied physiology, but to the field surrounding and penetrating the body. By watching the field balancing a self-healing process can be initiated. Beyond the free flow of the CRI it is possible to enter underlying rhythms like the long tide of 2 ½ cycles per minute as mentioned by Jealous in "The long tide and the breath of life" 1995. Sometimes the attention can be focused on the potency in the cerebrospinal fluid, called by Sutherland "the breath of life, a fluid within a fluid" (cp. McPartland 1997, p. 43)

These terms explain subjective applied knowledge of high therapeutic value. Nevertheless a mystical plain is reached using these techniques. Only the entrainment hypothesis opens a possible explanation in the sense of scientific argumentation concerning these phenomena.

6 Empirical study

The empirical study intends to evaluate the hypothesis of this thesis. We want to find out if one osteopathic treatment is able to balance the ANS, supporting a shift from the sympathetic control to a parasympathetic control by using the three most common physiological indices, the heart rate, respiratory frequency and blood pressure.

6.1 The study design

70 people volunteered as subjects for the following study. The group consisted of 27 men and 43 women between the age of 20 and 67 without clinical pathology of heart, lung and blood pressure.





We present the results of 50 individuals with the impact of osteopathic treatment (OT) and 20 subjects observed in the state of rest (R) as control group.



Tab.02 Demographic Indicator Sex

18 men and 32 women participated in the experimental group and volunteered for the osteopathic treatment (OT). Nine men and eleven women formed the control group and volunteered for their observation in the state of rest (R).



Tab.03 Demographic Indicator Age

The youngest subject was 20 years old in the group of rest (R) and 22 years old in the group of osteopathic treatment (OT). The oldest subject was in the age of 67 in the group of rest (R) and 59 in the group of osteopathic treatment (OT). The mean
Diploma thesis

age between the R group and the OT group shows a difference of five years, the median only one and a half year. Most of the participating volunteers in both groups were in their mid thirties according to the median. Over all these numbers reflect a balanced spectrum in the demographic indicator age.



Tab.04 Demographic Indicators Age and Sex, R Group



Tab.05 Demographic Indicators Age and Sex, OT Group

Tables 04 and 05 show the equal distribution in both groups OT and R between male and female and their age. The following study results are based on this equal distribution.

6.2 The study methodology

There is no method to measure the activity of the ANS. Therefore we used heart rate, respiratory frequency and blood pressure which show measurable changes in the activity of the ANS.

The technique of measuring pulse rate involved the use of a digital pulse counter. The respiration rate was measured parallel by counting the breaths during one minute. The blood pressure was measured by modern oscillometric blood pressure technology.

Traditionally the arterial blood pressure in humans is routinely measured by the auscultatory method. An inflatable cuff (Riva-Rocci cuff) attached to a mercury manometer (sphygmomanometer) is wrapped around the arm and a stethoscope is placed over the brachial artery at the elbow. The cuff is rapidly inflated until the pressure in it is well above the expected systolic pressure in the brachial artery. The artery is occluded by the cuff, and no sound is heart with the stethoscope. The pressure in the cuff is then lowered slowly. At the point at which systolic pressure in the artery just exceeds the cuff pressure, a spurt of blood passes through with each heartbeat and synchronously with each beat a tapping sound is heard below the cuff. The cuff pressure at which the sounds are first heard is the systolic pressure. As the cuff pressure is lowered further the sounds become louder, then dull and muffled; finally, in most individuals they disappear. These are the sounds of Korotkow. The sounds of Korotkow are produced by turbulent flow in the brachial artery. (Gangong 1993, p. 531-532)

According to clinical experience the results of this measuring method vary significantly between examiners. A traditional measurement method using a cuff wrapped around the arm could also disturb the subjects relaxation, which is essential for the outcome of the study.

Both considerations leaded to the use of modern oscillometric blood pressure technology. A wrist blood pressure clock for mobile use by Hartmann was placed on the subjects wrist before starting the examination improving the reliability of the measurement and the comfort for the study subjects.



Fig.13 Tensoval Mobil - oscillometric blood pressure technology

The wrist blood pressure clock was clinically validated after the recognized test standard ANSI/AAMI SP10-1992 (Association for the Advancement of Medical Instrumentation) by an independent institute and fulfils the strict quality requirements to the measuring accuracy in the European norm EN 1060-3:1997. Therefore the manufacturer guarantees a systematic measuring accuracy of +/- 5 mmHg and an empirical standard deviation of 8 mmHg. (cp. Hartmann AG 2001)

This accuracy was considered as sufficient for the studies outcome because the absolute values were not as important as the chronological variations of the taken measurements.

6.3 The study realisation

The participants of the study were regular patients of osteopathy and the studies examination took place in the time-range of half an hour before the regular osteopathic treatment. So all volunteers knew about osteopathy and were asked in a previous therapy if the would like to support the study. Also all the explanations needed were given at that time.

The subjects were familiar with the place and the person involved in the experiments. This setup was chosen to avoid the well known white coat effect resulting in a hypertonic reaction because of excitement.

The time of the experiments was carefully chosen. The experiment never happened directly after a big meal to avoid postprandial hypo- and hypertension. The experiments were mainly done in the time from 4 to 6 p.m.

It was intended that all individuals felt comfortable during the procedure with special attention to temperature, light and noise. Talking was avoided during the time of measurement to gain constantly straight results uninfluenced from emotional reactions and changes in breathing.

The participants were asked to relax and to have pleasant thoughts meanwhile. They were not asked to concentrate and they were not asked not to concentrate on their breath and heart rate. Because it is a well known fact that avoiding special thoughts and intensions result in the opposite too. Some subjects of the OT group relaxed so well that they almost felt asleep.

The subjects of both groups, the osteopathic treatment as well as the rest group, laid on the treatment table in supine position having the oscillometric blood pressure clock positioned on their wrists.

Both groups were evaluated

- 1) on arrival (0 minutes),
- 2) after 5 minutes of rest,
- 3) after 10 minutes of rest,
- after the osteopathic treatment or rest of another 5 minutes (after 15 minutes) and
- 5) after 20 minutes.

The osteopathic treatment was applied only between the 10th and 15th minute of the experiment. Comparison of the results between the group of osteopathic treatment (OT) and the group of rest (R) in this time should demonstrate the effect of the osteopathic technique.

The applied cranial-sacral technique has been explained in chapter five. In this technique the practitioner places one hand under the occiput and the other hand under the sacrum. The patient remains in a supine position, laying on the practitioners hands. (cp. Upledger 1994, p. 86-87) This setup was held for five minutes independent of the treatments progression.

The measurement results were filled in a prepared form (see appendix unfiltered data). All results were digitally processed, tables were generated and the results were plotted on graphs showing differences in the three indices measured for each individual.

6.4 The study findings

The results and findings are presented in the following order: first we present two individual examples, one from the group of rest, one from the group of osteopathy treatment. Their data was made anonymous by reducing their names into initials. All the collected data of the seventy experiments are aggregated first in groups of 20 subjects in the group of rest and 50 subjects in the group of osteopathic treatment. Further on these two groups are compared in their chronological variations during rest and osteopathic treatment.

6.4.1 Individual Rest example Effect of Rest (R)

Subject N°	11
Initials	M.B.
Sex	m
Age	42
Date	11.01.06

Pulse	Measurement after minutes					
	0 5 10 15 20					
beats / min	76	71	72	71	68	

Breathing	Measurement after minutes					
	0 5 10 15 20					
breaths / min	13	11	11	11	11	

Blood pressure		Measurement after minutes				
mmHg	0	5	10	15	20	
systole	135	125	122	124	121	
diastole	72	68	65	67	64	

Tab.06 Data sheet subject 11 group of rest

The personal data, sex and age, and the date of the experiment as well as the sequence number was noted from each subject as shown in table 06. The measurement data was noted in the beginning, after 5, 10, 15 and 20 minutes of rest. Table 06 is one example randomly chosen out of twenty subjects.



Tab.07 Pulse subject 11 group of rest

In the case shown in table 07 the pulse significantly lowers between the beginning and the fifth minute of rest. After the fifth minute it stays almost constant.





After five minutes the breathing of subject eleven becomes constant at the same level. This reaction can not be found on each individual as shown later.



Tab.09 Blood pressure subject 11 group of rest

The blood pressure regulation shown in table 09 is quite typical for a person relaxing. Interesting is the fact that on many measurements one can find a slight increase in blood pressure after 10 minutes, which might be due to long term blood pressure regulation described in the chapter 2.4 Physiological basics.

6.4.2 Individual Osteopathic Treatment example

Effect of osteopathic treatment (OT)

Subject N°	23
Initials	D.V.
Sex	f
Age	33
Date	10.10.01

Pulse	Measurement after minutes					
	0 5 10 15 20					
beats / min	78	75	70	58	58	

Breathing	Measurement after minutes					
	0 5 10 15 20					
breaths / min	13	12	11	9	9	

Blood pressure		Measurement after minutes				
mmHg	0	5	10	15	20	
systole	123	106	100	90	91	
diastole	76	68	67	59	58	

Tab.10 Data sheet subject 23 group of osteopathic treatment

Table 10 represent an example of the group of osteopathic treatment also randomly chosen.



Tab.11 Pulse subject 23 group of osteopathic treatment

Table 11 points out the time range of osteopathic treatment between the 10th and 15th minute. During this time the pulse of subject 23 reduced significantly.



Tab.12 Breathing subject 23 group of osteopathic treatment

Table 12 shows a slight change in the breathing sequence during the treatment time in comparison with the ten minutes of rest before.





The last individual table 13 displays a significant effect of the osteopathic treatment on the blood pressure regulation of subject 23.

6.4.3 Rest Group results



Tab.14 Pulse beats/minute Rest Group

Table 14 displays the mean and median of all 20 volunteers in the group of rest. The mean value of the pulse measurement shows a slight increase between the fifteenth and the twentieth minute.



Tab.15 Breathing breaths/minute Rest Group

The breathing of the group of rest demonstrates that the lowest breathing frequency within the twenty minutes of measurement happens around the tenth minute. It also displays an increase of frequency from this point to the fifteenth minute. This might be caused by the breathing getting more superficial. The increase is even underlined by the median value in table 15.



Tab.16 Blood pressure mmHg Rest Group

Table 16 demonstrates what has been mentioned before in the single case number eleven. Between the tenth and fifteenth minute one can find a slight increase in blood pressure after 10 minutes, which might be due to long term blood pressure regulation.



6.4.4 Osteopathic Treatment Group results

Tab.17 Pulse beats/minute OT Group

Table 17 shows the impressive effect of the applied cranial technique on the fifty subjects which can be seen in the mean and median values.



Tab.18 Breathing breaths/minute OT Group

As shown in table 18 even the breathing frequency reacted strongly on the osteopathic technique.



Tab.19 Blood pressure mmHg OT Group

The blood pressure regulation could also be affected by the OT as pointed out in table 19. The mean and median are similar in their trends.



6.4.5 Rest and Osteopathic Treatment Group results compared

Tab.20 Pulse beats/minute Rest compared with the Osteopathic Treatment Group

Table 20 demonstrates the significant difference between the group of rest and the group of osteopathic treatment. Between the tenth and fifteenth minute the two curves start to deviate due to the OT.



Tab.21 Pulse change Rest compared with the Osteopathic Treatment Group in percentage over 20 minutes

-18,8 percent decrease of pulse rate in the group of OT compared to -5,6 percent overall decrease in the group of rest shown in table 21 is impressive, but has to be relativised because of the slightly higher starting values in the group of OT.



Tab.22Pulse change Rest compared with the Osteopathic Treatment Group in
percentage between the tenth and fifteenth minute (OT)

Table 22 highlights the critical time between the tenth and fifteenth minute. A decrease of -9,1 percent in the group of OT compared to an almost constant value of -0,4 percent in the group of rest underlines what has been shown in table 21 before.





In table 23 the two curves are crossing between the tenth and fifteenth minute. The osteopathic treatment caused a decrease of breathing frequency in this time. Meanwhile the subjects in the group of rest showed a slight increase of breathing frequency in the same time period.



Tab.24 Breathing change Rest compared with the Osteopathic Treatment Group in percentage over 20 minutes

The decrease of the breathing frequency during twenty minutes in the group of OT is with -24,7 percent double as high as the decrease of the breathing frequency in the group of rest with -11,9 percent as shown in table 24.



Tab.25Breathing change Rest compared with the Osteopathic Treatment Group in
percentage between the tenth and fifteenth minute (OT)

Table 25 focuses again on the five minutes of OT demonstrating that the major change happened in the time range between the 10th and 15th minute. It also demonstrates that the group of rest increases slightly their breathing frequency about 2,3 percent. In comparison with this result the OT group significantly decreases their breathing frequency about -15,2 percent.



Tab.26 Blood pressure mmHg Rest compared with the Osteopathic Treatment Group

Table 26 compares the mean systolic and diastolic blood pressure of the group of rest and the group of osteopathic treatment. The systolic mean value of both groups decreases till the tenth minute. Between the 10th and the 15th minute the two lines cross, because the systolic mean value of the rest subjects increases while the systolic mean value of the OT subjects decreases significantly. This fact is very interesting because it shows that a self regulating mechanism of blood pressure which is evident in the group of rest is not working in the group of OT. The lines of the diastolic mean values of the rest and OT volunteers are crossing two times, once shortly before the 15th minute and a second time between the 15th and the 20th minute. This might be due to the fact, that the beginning diastolic mean value of the OT group is 5,4 mmHg higher than the one of the rest group. This difference might be explained by the random sample of this studies design. Larger groups should lead to approximately similar initial values. In the following discussion of the results we assume that the tendencies of the regulation are of greater importance than the absolute values.



Tab.27Blood pressure change Rest compared with the Osteopathic Treatment
Group in percentage over 20 minutes

Table 27 displays the difference of the systolic and diastolic mean values between the OT group and the group of rest as the control group within 20 minutes. The systolic mean value of the OT subjects decreases with a difference of 9,8% much more than the diastolic mean value with a difference of 5,8% in comparison with the group of rest. This fact correlates with normal physiology. The diastolic value always changes much less, because it is more an expression of the systemic pressure whereas the systolic pressure shows the maximum of the blood pressure amplitude.



Tab.28Blood pressure change Rest compared with the Osteopathic TreatmentGroup in percentage between the tenth and fifteenth minute (OT)

As explained before in table 26 (p. 41) the systolic and diastolic mean values of the rest group raise while the ones of the OT group decrease. Table 28 demonstrates clearly the different tendencies in the time of treatment between the 10th and 15th minute with a difference of 16,8% in the systolic mean value and a difference of 10,3% in the diastolic mean value.

7 Discussion and Summary

The discussion of the studies findings described in detail in chapter 6.4 will lead from this research in the context of other studies in the field of osteopathy to the verification of the initial hypothesis and the findings summary as well as an reflection of the limitations of this study and needs for future studies in the field of Cranial Osteopathy.

7.1 Findings of the study in the context to other Cranial Osteopathy studies

This study is directly referring to two clinical case studies published at the Vienna School of Osteopathy by Walter Krasser D.O. and Maria Schögler D.O.

Krasser (2000) observed in his study "The Decrease of High Blood Pressure using a Lateral Fluid Drive Technique with Concentration on the Ossa Temporalia" with an examination of 13 hypertonic subjects an average decrease in blood pressure of 10% in systolic as well as in diastolic mean value after using the defined cranial osteopathic technique. His treatment group consisted of 4 women and 9 men aged between 40 and 65 years. All volunteers were measured once before and after the treatment. "*The probationers of group A [the treatment group] showed on average an improvement of 10%, i.e. decrease of the systolic pressure. The diastolic value could as well be decreased by 10%.*" (Krasser 2000, p. 31)

His control group consisted of 8 hypertonic women and 4 hypertonic men aged between 31 and 64 years, who were also measured twice, but those persons were not treated. They have only been touched for a few minutes at their legs and in the area of the forefoot. It was Krasser's intention not to receive any therapeutic effect on the subjects of the control group. *"The probationers of the control-group showed on average a variation of the systolic pressure value of 0,1%. The diastolic value - for the whole group B [control group] - varied by 0,7%." (Krasser 2000, p. 41)*

The setup of this current study was chosen differently compared to Krasser's study by following reasons: Before finding out, if there is an therapeutic effect of a cranial technique, it would be interesting to find out whether there was a physiological effect. Therefore it was chosen to examine healthy individuals and not hypertensive subjects. Following the principle first trying to understand physiology before getting into pathophysiology. Experience from clinical practice shows, that independent by which means high blood pressure is decreased, the systolic and diastolic values hardly ever change about the same percentage as described by Krasser. This fact in Krasser's thesis might be due to the studies samples sizes, which are quite small by measuring 13 probationers in the treatment group and 12 in the control group.

Another distinction between the current study and the study of Krasser originates in the assumption that "you cannot not communicate" (Watzlawick et al. 2000), especially when you are touched physically by somebody. According to the entrainment theory cranial osteopathy is a resonance phenomenon of multiple biological oscillators between the therapist and the patient. Even when the therapist decides not to get into resonance with the patient, this is a clear information to the patient causing a reaction. Lets imagine one is in a waiting room and there is nothing else to do than waiting. Somebody else enters this room. It is a common need to get into to react to this behaviour? By avoiding any therapeutic effect Krasser might have influenced a normal relaxation of his control group.

Therefore the control group in our study was not touched at all to have a clear reaction of normal rest.

In this context it has to be mentioned that Krasser's first measurement in both groups happened after ten minutes of rest. The second time he measured after another 10 minutes in the control group and after about 10 minutes in the group he intentionally treated. In Krasser's control group the mean systolic value varied only by 0,1% and the mean diastolic value by 0,7%. In his paper it does not come out clearly, if he is taking about an increase ore decrease of blood pressure. He only mentions the terms variation and modification in this context in his chapter Results in Group B (control group). (cp. Krasser 2000, p. 40-49)

If one compares Krasser's findings with the corresponding measurements of the blood pressure mean values in the control group in this thesis between the 10th and the 20th minute of rest, the values even increase from 107,5 mmHg mean systolic at the 10th minute to 114,6mmHg at the 20th minute and from 67,9mmHg mean diastolic to 68,3mmHg at the 20th minute. In percentage this is an increase of 6.6% in the systolic mean value and an increase of 0,6% in the diastolic mean value between the 10th and the 20th minute.

Nevertheless Krasser's and our findings demonstrate that there are no big changes in blood pressure after 10 minutes of rest.

This difference also lines out the importance of the development of the blood pressure in time, what has been discussed before. Blood pressure regulation is due to so many regulating systems as shown in the chapter 2.4 physiological basics and is constantly adopting to multiple environmental influences. It is difficult to describe the complex regulation of blood pressure with only five measurements as done in this thesis - with two measurements it is impossible.

Comparing the results of the treatment groups in both studies Krasser finds a decrease of -10% in the systolic and diastolic mean values between the 10th and 20th minute according to the design of this study. In the current study one can find a decrease of -9,3% in the systolic mean value and a decrease of -8,4% in the diastolic mean value between the 10th and 20th minute. Additionally this study points out clearly the trend of a slight increase of blood pressure between the stop of the treatment at the 15th minute and the last measurement at the 20th minute.

Overall the outcome of these two studies confirm each other. There are measurable effects on healthy as well as on hypertonic subjects using cranial fluid techniques.

On the other hand Schögler (2000) could not find a significant effect of her applied cranial CV4-technique upon blood pressure in a group of threshold hypertensive patients as she mentions in her paper.

Schögler's groups consisted of 10 male subjects in the treatment group and 10 male individuals in the placebo group in the age between 30 and 45. The blood pressure was measured before and after the treatment in a sitting position and four times in between during treatment or placebo treatment in the control group.

The positioning of the hands were identical between the two groups, just the therapists intention was not present in the placebo group. We encounter the same problem as mentioned above in Krasser's thesis. According to the entrainment theory it is not possible for a therapist not to treat as soon as the therapist is laying his or her hands on the patient. Especially a therapeutic setup and the patient in belief of being treated makes it impossible to avoid a resonance phenomenon. Therefore her results of treatment are quite in the same range of Krasser's and our findings with the difference that she treated both groups according to our understanding. Once consciously in the treatment group and once unconsciously in the placebo group.

53

Schögler described a decrease of the mean systolic blood pressure by -8,81% in a group of ten threshold hypertensive patients within 15 minutes in comparison to a decrease of -5,27% in the placebo group of ten subjects within 15 minutes. The diastolic mean value did not change according to her measurements (cp. 2000, p. 42). This might be due to the fact, that she had the subjects sit in upright position to take the last measurement. In this position the short term blood pressure regulation has to function, otherwise the subjects would collapse due to hypostatic dysfunction. To our understanding the body's position has such big influence on blood pressure regulation, that one gets clearer results measuring always in the same position.

7.2 Verification of the initial hypothesis

The studies findings point out clearly that there is an effect of one osteopathic technique balancing the ANS indicating a shift from the sympathetic to parasympathetic control.

A significant decrease of -18,8% in the mean value of the heart rate was caused by the osteopathic treatment compared to a decrease of -5,6% mean in the control group with a difference of 13,2% within 20 minutes. In the time range between the 10th and 15th minute -9,1% decrease in the mean value of the heart rate in the treatment group in comparison to -0,4% mean in the group of rest shows a difference of 8,7%. The most significant decrease could be observed within the five minutes of treatment. Such a change in pulse rate demonstrates a clear shift into parasympathetic control.

The decrease of the breathing frequency during twenty minutes in the group of osteopathic treatment is with -24,7 percent double as high as the decrease of the breathing frequency in the group of rest with -11,9 percent. The group of rest increases slightly their breathing frequency about 2,3 percent between the 10th and 15th minute. In comparison with this result the osteopathic treatment group significantly decreases their breathing frequency during the treatment about -15,2 percent. This difference of 17,5% points out the parasympathetic reaction of the ANS to the osteopathic treatment.

Significant results can also be found in the blood pressure measurements. The systolic mean value of the osteopathic treatment subjects decreases with a difference of 9,8% much more than the diastolic mean value with a difference of 5,8% in comparison with the group of rest within 20 minutes.

Between the 10th and 15th minute the systolic and diastolic mean values of the rest group raise while the ones of the OT group decrease resulting in a significant difference of 16,8% in the systolic mean value and of 10,3% in the diastolic mean value.

Blood pressure regulation is controlled by multi-functional regulating systems. The ANS as a superior control system also seems to be influenced by the applied cranial membrane and fluid balance technique decreasing blood pressure.

According to these findings the entrainment hypothesis seems to open the framework which allows explanations to these phenomena, changes in pulse rate, breathing frequency and blood pressure regulation. McPartland states it in his words, "if there is balance within the autonomic nervous system than the bodies many rhythms harmonize into a strong, coordinated, sinusoidally fluctuating entrainment frequency, which can be palpated as strong healthy CRI" (1997, p. 42).

7.3 Limitations of the study and ideas for future studies in the field of Cranial Osteopathy

In this study as well as in the studies of Krasser and Schlögl cranial fluid techniques were applied to effect the blood pressure regulation and additionally in our case heart rate and breathing frequency. The reason for the preference of cranial fluid techniques to effect blood pressure regulation could be a following topic for further research in the field of Cranial Osteopathy.

A strong limitation in studies like these is the sample size. Compared with studies in the field of orthodox medicine the number of the studies cases, which have been quoted in this context, are of no real statistical relevance. For instance in the given study one could criticise the different starting mean values in blood pressure regulation between the treatment and the rest (control) group with 2,6% difference in the systolic mean starting value and 6,7% difference in the diastolic mean starting value. This values should be approximately similar in a larger group of subjects.

Therefore it would be useful to introduce team oriented master thesis projects to gain more significant results. In the context of this topic one joint thesis on blood pressure regulation due to a specific osteopathic technique would be of much greater relevance than three limited small sample studies. Even the studies design could be improved by collective versus individual ideas. The most restrictive limitation for further scientific development in the field of osteopathy was encountered during the six years research. By starting this osteopathic thesis in 2000 all articles for example in the journal of the American Academy of Osteopathy were accessible for everybody over the Internet free of charge. Today the access is restricted only to registered members paying for their access. This profit orientation is getting common in the so called knowledge economy of the Internet and e-commerce age. This policy change makes research in the osteopathic field more difficult and the acknowledgement by other scientific traditions almost impossible. Hardly any medical doctor would pay a fee to read an osteopathic article which might concern a topic of his interest, but through free access to the developing knowledge base other disciplines could build bridges into the field of osteopathy by encountering inspiring ideas.

We started this master thesis and our own six years journey of research inspired by James S. Jealous. Therefore we want to close the discussion in this thesis quoting Jealous again: "Osteopathy is about finding the "health" in the patient. This is a direct perceptual skill; it is not just the idea of making the person healthy. Finding the health in the patient is the learned art of directly perceiving something other than disease in the patient, a skill that therapeutically engages laws of healing not recognized by orthodox medicine. [...] Osteopathy awoke us to the role of the autonomic nervous system (ANS) in health and disease. This tremendous insight was profound. As science has matured, it has noticed the relationship of stress to disease. Today most Americans are aware of the role of stress in upsetting the balance of health. Osteopathy, however, was way ahead of even today's common medical knowledge. It had the skill to directly interpret and influence autonomic activity using perceptual and palpatory skills [...]." (Jealous 1999, S.20)

So it lies in our hearts and hands to prevent the death of osteopathy as a living tradition keeping the osteopathic spirit alive.

8 Bibliography

American Heart Association (1996). Special Report: Heart Rate Variability. Standards of Measurement, Physiological Interpretation, and Clinical Use. Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology. In. Circulation Vol. 93, No. 5, March 1, 1996. p. 1043 – 1065.

Farlex (2005). The Free Dictionary. Hypertension. Definition. In: <u>http://encyclopedia.thefreedictionary.com/hypertension</u> (visited 20th November 2005)

Ganong, W. F. (1993). Review of Medical Physiology (16th ed.). London, et.al.: Prentice Hall International, Appleton & Lange.

Hartmann AG (2001). Klinische Validierung TENSOVAL® Blutdruckmessgeräte. In: <u>http://de.hartmann.info/active/PDF/DE/sonstiges/validierung_mobil.pdf</u> (visited 20th November 2005)

Jealous, J. S. (1999). Accepting the death of osteopathy: A new beginning. In: The AAO Journal Vol.9 No.4 / Winter 1999, S. 19 – 22

Karemaker, J. M. (1999). Autonomic integration: the physiological basis of cardiovascular variability. In: The Journal of Physiology (1999), 517.2, p. 316-316 <u>http://jp.physoc.org/cgi/content/full/517/2/316</u> (visited 20th November 2005).

Krasser, W. (2000). The Decrease of High Blood Pressure Using a Lateral Fluid Drive Technique with Concentration on the Ossa Temporalia. Dissertation: Wiener Schule für Osteopathie.

Lippincott, R. C.; Lippincott, H. A. (1948). A Manual of Cranial Technique (2nd ed.). Ann Arbor, Michigan: Edwards Brothers Inc.

Magoun, H. I. (Ed.) (1976). Osteopathy in the Cranial Field (3rd). Kirksville, Missouri: The Journal Printing Company

McDonagh, P. (2005¹). Determinates of Mean Arterial Blood Pressure. In: <u>http://human.physiol.arizona.edu/SCHED/CV/McDo/McDo18/McDo.L18.pdf</u> (visited 19th November 2005) McDonagh, P. (2005²). Regulation of Mean Arterial Blood Pressure. In: <u>http://human.physiol.arizona.edu/SCHED/CV/McDo/McDo19/McDo.L19.pdf</u> (visited 19th November 2005)

McPartland, J. M., D.O., M.S.; Mein, E. A., M.D. (1997). Entrainment and the cranial rhythmic impulse. In: Alternative Therapies; Jan. 1997, VOL. 3, No. 1, p. 40 - 45

Norton, JM (1991). A tissue pressure model for palpatory perception of the cranial rhythmic impulse. In: Journal of the American Osteopathic Association, Vol. 91, Issue 10, p. 975-975.

Pernkopf, E. (1963). Atlas der topografischen und angewandten Anatomie des Menschen. München und Berlin: Urban & Schwarzenberg.

Pickering Pick, T., F.R.C.S.; Howden, R., M.A., M.B., C.M. (Eds.) (1988). Grays Anatomy. Anatomy descriptive and surgical (15th). New York:Galley Press

Rohen, J. W.; Yokochi, Ch., M.D. (1988). Anatomie des Menschen. Photografischer Atlas der systematischen und topographischen Anatomie (2. Aufl.). Stuttgart, N.Y.: Schattauer.

Schmidt, R. F.; Thews, G. (Hg.) (1990). Physiologie des Menschen (24. Aufl.). Berlin, u.a.: Springer-Verlag.

Schögler, M. (2000). Observation: Does the CV4-Technique Have a Measurable Effect Upon Blood Pressure with Threshold Hypertensive Patients? Are there Common Osteopathic Dysfunctions which May Have an Explainable Reason for the Development of Hypertension? Wiener Schule für Osteopathie, Diploma thesis.

Sommerfeld, P. (2001). Inter- and intraexaminer reliability in palpation of the Primary Respiratory Mechanism as well as possible correlations with examiners' and subjects' respiratory rates: Discussion of the fundamental problems and experimental research. Int. School of Osteopathy Vienna, Diploma thesis.

Sutherland, A. S.; Wales, A. L. (Ed.) (1998). Contributions of Thought. The Collected Writings of William Garner Sutherland, D.O. (2nd ed.). Forth Worth, Texas: Sutherland Cranial Teaching Foundation, Inc.

Sutherland, W. G. (1994). The Cranial Bowl. (Reprint). Mankato, Minnesota: Free Press Company.

Taylor, A. J.; Eckberg, D. L. (1996). Fundamental Relations Between Short-term RR Interval and Arterial Pressure Oscillations in Humans. In. Circulation Vol. 93, No. 8, April 15, 1996. p. 1527-1532.

Upledger, J. E.; Vredevoogd, J. D. (1994). Lehrbuch der Kraniosakral-Therapie (2. Aufl.). Heidelberg: Haug.

Watzlawick, P.; Beavin, J. H.; Jackson, D. D. (2000). Menschliche Kommunikation. Formen, Störungen, Paradoxien. Bern: Huber.

Wikipedia (2005). The Free Encyclopedia. Hypotension. Causes and Indicators. In: http://en.wikipedia.org/wiki/Hypotension (visited 20th November 2005)

9 Appendix

9.1 Index of tables

Tab.01	Number of Volunteers29
Tab.02	Demographic Indicator Sex
Tab.03	Demographic Indicator Age
Tab.04	Demographic Indicators Age and Sex, R Group
Tab.05	Demographic Indicators Age and Sex, OT Group
Tab.06	Data sheet subject 11 group of rest35
Tab.07	Pulse subject 11 group of rest
Tab.08	Breathing subject 11 group of rest
Tab.09	Blood pressure subject 11 group of rest
Tab.10	Data sheet subject 23 group of osteopathic treatment
Tab.11	Pulse subject 23 group of osteopathic treatment
Tab.12	Breathing subject 23 group of osteopathic treatment
Tab.13	Blood pressure subject 23 group of osteopathic treatment40
Tab.14	Pulse beats/minute Rest Group41
Tab.15	Breathing breaths/minute Rest Group41
Tab.16	Blood pressure mmHg Rest Group42
Tab.17	Pulse beats/minute OT Group43
Tab.18	Breathing breaths/minute OT Group43
Tab.19	Blood pressure mmHg OT Group44
Tab.20	Pulse beats/minute Rest compared with the Osteopathic Treatment Group44
Tab.21	Pulse change Rest compared with the Osteopathic Treatment Group in percentage over 20 minutes45
Tab.22	Pulse change Rest compared with the Osteopathic Treatment Group in percentage between the tenth and fifteenth minute (OT)45
Tab.23	Breathing breaths/minute Rest compared with the Osteopathic Treatment Group
Tab.24	Breathing change Rest compared with the Osteopathic Treatment Group in percentage over 20 minutes46
Tab.25	Breathing change Rest compared with the Osteopathic Treatment Group in percentage between the tenth and fifteenth minute (OT)47
Tab.26	Blood pressure mmHg Rest compared with the Osteopathic Treatment Group
Tab.27	Blood pressure change Rest compared with the Osteopathic Treatment Group in percentage over 20 minutes
Tab.28	Blood pressure change Rest compared with the Osteopathic Treatment Group in percentage between the tenth and fifteenth minute (OT)50 60

9.2 Index of figures

Fig.01	Occipital Bone (Rohen, Yokochi 1988, p. 29)	.7
Fig.02	Occipital Bone, Atlas and Axis (Rohen, Yokochi 1988, p. 184)	.8
Fig.03	Sacrum and Spine (Rohen, Yokochi 1988, p. 176, 179)	.8
Fig.04	Dura Mater (Rohen, Yokochi 1988, p. 85)	.9
Fig.05 F	alx cerebri and cerebelli (Pernkopf 1963, p. 50)	.9
Fig.06	Occiput and Spine (Rohen, Yokochi 1988, p. 184)	10
Fig.07	Ventricle System (Rohen, Yokochi 1988, p. 108)	11
Fig.08	Determinants of Mean Arterial Blood Pressure (Mc Donagh 2005 ²)	14
Fig.09	The baroreceptor reflex (Mc Donagh 2005 ²)	16
Fig.10	Renin – Angiotensin II – Aldosterone System (Mc Donagh 2005 ²)	17
Fig.11	Reciprocal tension membrane movement in flexion (Magoun 1976, p. 37).2	23
Fig.12	Position of the hands to balance sacrum and occiput in supine position (Upledger 1994, p. 97)	27
Fig.13	Tensoval Mobil - oscillometric blood pressure technology	33

9.3 Unfiltered data for the analysis

9.3.1 Studies single case report

Effect of osteopathic treatment (OT)

Subject N°:

Initials:

Sex:

Age:

Date:

Pulse:

Measurements after minutes

Beats/ min

0	5	10	15	20

Breathing:

Measurement after minutes

Breaths/ min

0	5	10	15	20

Blood Pressure: Measurements after minutes

mmhg

0	5	10	15	20

9.3.2 Raw data group of Rest (R)

Date of Study	28.02.01 - 30.1.06

Demographic Indicators	Sex	Age
V 01	f	59
V 02	f	29
V 03	f	20
V 04	f	30
V 05	m	30
V 06	m	32
V 07	f	57
V 08	f	35
V 09	m	31
V 10	m	36
V 11	m	42
V 12	f	61
V 13	m	36
V 14	m	19
V 15	m	56
V 16	f	31
V 17	f	42
V 18	m	46
V 19	f	67
V 20	f	24

Diploma thesis

Physiological					
Indicators					
Pulse	0 min.	5 min.	10 min.	15 min.	20 min.
V 01	80	75	60	60	60
V 02	75	70	65	66	67
V 03	75	70	71	72	73
V 04	67	81	89	79	84
V 05	69	63	70	65	68
V 06	67	60	56	62	62
V 07	78	73	62	63	65
V 08	73	69	70	71	75
V 09	70	65	68	69	68
V 10	69	60	58	61	62
V 11	76	71	72	71	68
V 12	81	78	81	80	78
V 13	68	69	70	68	70
V 14	65	68	66	65	66
V 15	82	75	72	72	73
V 16	68	65	62	64	64
V 17	76	72	69	69	70
V 18	74	70	68	68	66
V 19	86	81	81	79	80
V 20	59	58	60	60	58

Breathing	0 min.	5 min.	10 min.	15 min.	20 min.
V 01	14	12	9	9	9
V 02	13	10	10	10	11
V 03	16	14	13	14	14
V 04	14	15	15	15	16
V 05	9	10	10	10	10
V 06	11	10	10	11	11
V 07	13	12	11	11	11
V 08	15	12	12	12	13
V 09	11	10	10	10	10
V 10	12	11	10	11	11
V 11	13	11	11	11	11
V 12	15	13	11	12	11
V 13	10	11	9	11	10
V 14	11	11	10	10	10
V 15	12	10	11	10	10
V 16	13	11	10	9	10
V 17	11	11	10	10	10
V 18	14	13	13	13	12
V 19	15	13	13	14	13
V 20	10	9	9	9	9

Blood pressure	sy 0 min.	di 0 min.	sy 5 min.	di 5 min.	sy 10 min.	di 10 min.	sy 15 min.	di 15 min.	sy 20 min.	di 20 min.
V 01	145	95	127	92	127	92	128	93	127	92
V 02	141	70	118	71	115	71	116	72	117	70
V 03	112	75	97	64	97	63	101	63	103	65
V 04	127	79	110	75	111	74	110	75	112	75
V 05	107	71	104	68	100	62	99	61	100	65
V 06	101	69	107	70	101	67	109	71	106	70
V 07	145	89	126	85	125	85	127	86	125	85
V 08	111	74	97	64	95	63	100	63	103	68
V 09	108	70	105	69	103	61	104	62	103	64
V 10	110	70	105	69	101	67	108	70	106	70
V 11	135	72	125	68	122	65	124	67	121	64
V 12	141	82	115	66	118	63	115	64	116	62
V 13	126	70	110	62	112	56	110	54	109	52
V 14	118	68	115	62	113	57	115	57	115	56
V 15	148	82	153	78	132	80	130	78	131	79
V 16	126	76	120	70	122	68	119	68	120	66
V 17	136	71	128	70	122	66	120	67	118	65
V 18	135	68	121	68	123	67	122	68	121	68
V 19	147	80	135	76	131	72	132	74	130	76
------	-----	----	-----	----	-----	----	-----	----	-----	----
V 20	115	72	113	65	108	58	110	56	109	54

9.3.3 Raw data group of osteopathic treatment (OT)

 Date of Study
 26.02.01 - 26.06.02

Demographic	0	٨٥٥		
Indicators	Sex	Age		
V 01	f	43		
V 02	m	24		
V 03	m	27		
V 04	f	47		
V 05	f	27		
V 06	f	45		
V 07	m	43		
V 08	f	30		
V 09	m	30		
V 10	f	26		
V 11	f	25		
V 12	f	22		
V 13	f	43		
V 14	m	32		
V 15	f	53		
V 16	f	39		
V 17	m	29		
V 18	m	33		
V 19	f	43		
V 20	f	25		
V 21	f	38		
V 22	m	41		
V 23	f	33		
V 24	m	37		

V 25	f	28
V 26	f	25
V 27	f	43
V 28	f	34
V 29	m	33
V 30	f	56
V 31	f	39
V 32	m	27
V 33	m	28
V 34	f	43
V 35	f	29
V 36	f	48
V 37	m	44
V 38	f	30
V 39	m	34
V 40	f	24
V 41	f	29
V 42	f	25
V 43	f	56
V 44	m	50
V 45	f	59
V 46	f	45
V 47	f	38
V 48	m	49
V 49	m	26
V 50	m	31

Physiological					
Indicators					
Pulse	0 min.	5 min.	10 min.	15 min.	20 min.
V 01	75	60	60	60	60
V 02	70	61	61	60	60
V 03	123	105	101	99	99
V 04	68	66	66	63	63
V 05	60	59	59	51	51
V 06	65	63	59	61	60
V 07	80	75	75	73	73
V 08	80	74	62	55	56
V 09	61	53	51	52	52
V 10	74	60	52	52	52
V 11	61	52	54	51	52
V 12	77	70	70	53	52
V 13	71	61	59	58	58
V 14	54	52	52	51	51
V 15	61	53	52	51	52
V 16	71	59	59	59	59
V 17	73	63	63	62	62
V 18	111	103	101	96	96
V 19	73	68	67	64	64
V 20	70	62	60	51	51
V 21	66	62	60	61	60
V 22	78	74	74	71	71
V 23	78	75	70	58	58
V 24	68	56	55	52	52
V 25	76	63	57	53	53
V 26	67	61	57	52	52

V 27	78	73	72	54	53
V 28	73	65	66	59	58
V 29	64	64	64	53	53
V 30	67	56	56	52	52
V 31	78	65	66	61	62
V 32	72	65	64	60	60
V 33	119	105	103	85	87
V 34	75	68	68	62	63
V 35	60	61	58	56	58
V 36	67	65	65	61	60
V 37	81	76	77	72	73
V 38	81	77	69	56	57
V 39	75	69	68	56	57
V 40	73	63	64	58	55
V 41	73	64	65	53	52
V 42	77	72	71	53	54
V 43	81	76	78	65	70
V 44	75	73	72	63	64
V 45	63	55	54	51	52
V 46	75	69	68	61	60
V 47	69	65	64	57	58
V 48	77	75	75	71	70
V 49	98	95	98	71	75
V 50	72	69	65	65	61

Breathing	0 min.	5 min.	10 min.	15 min.	20 min.
V 01	13	12	12	10	10
V 02	13	10	10	9	9
V 03	12	11	11	10	10
V 04	13	12	12	10	10
V 05	13	12	12	11	11
V 06	13	12	11	10	10
V 07	13	12	12	11	11
V 08	13	11	11	11	11
V 09	16	15	14	13	14
V 10	13	12	11	11	12
V 11	11	9	9	8	8
V 12	15	14	14	11	11
V 13	13	11	11	9	9
V 14	16	14	14	12	13
V 15	15	15	14	12	12
V 16	13	12	11	10	10
V 17	14	12	12	11	11
V 18	12	11	11	9	9
V 19	13	12	12	10	10
V 20	14	12	11	10	10
V 21	12	12	11	9	9
V 22	14	12	12	10	10
V 23	13	12	11	9	9
V 24	16	15	14	11	10
V 25	14	12	12	11	11
V 26	12	10	10	9	9
V 27	15	14	13	9	9

V 28	13	12	12	9	9
V 29	15	14	14	11	11
V 30	14	13	14	12	12
V 31	14	12	12	9	10
V 32	13	11	11	9	9
V 33	14	12	12	10	10
V 34	15	13	13	10	10
V 35	13	11	12	11	11
V 36	13	12	12	9	10
V 37	15	13	13	11	11
V 38	15	13	12	9	9
V 39	15	14	14	11	11
V 40	13	12	11	10	10
V 41	14	11	11	9	9
V 42	14	13	13	10	10
V 43	15	14	14	12	12
V 44	16	14	14	11	12
V 45	14	12	12	11	11
V 46	13	12	12	10	10
V 47	13	11	11	9	9
V 48	13	12	12	11	11
V 49	15	14	14	11	11
V 50	14	13	12	12	11

Blood pressure	sy 0 min.	di 0 min.	sy 5 min.	di 5 min.	sy 10 min.	di 10 min.	sy 15 min.	di 15 min.	sy 20 min.	di 20 min.
V 01	140	85	135	85	132	85	115	85	115	85
V 02	140	80	121	77	121	76	104	65	105	65
V 03	131	84	134	82	128	78	117	74	118	75
V 04	103	66	102	67	101	66	95	62	98	63
V 05	118	77	113	76	112	74	102	67	104	68
V 06	124	85	122	82	120	83	112	75	113	75
V 07	145	85	122	82	120	81	118	78	119	78
V 08	121	75	101	64	93	61	90	58	91	58
V 09	141	89	132	83	125	81	115	74	117	75
V 10	115	73	105	71	102	70	100	67	100	68
V 11	116	73	105	70	95	66	88	56	89	58
V 12	140	88	125	78	122	78	100	65	102	66
V 13	102	68	89	60	89	59	87	55	87	56
V 14	100	67	95	66	94	64	89	57	89	58
V 15	138	88	125	87	116	74	100	67	102	69
V 16	135	85	130	85	135	85	110	78	110	78
V 17	142	82	123	79	123	78	106	66	107	66

Abstract

V 18	129	83	125	82	125	78	116	74	117	75
V 19	115	72	110	70	110	69	96	63	97	63
V 20	122	78	115	76	113	75	103	66	104	67
V 21	126	83	121	82	118	80	114	74	113	75
V 22	140	85	125	80	122	80	117	77	118	77
V 23	123	76	106	68	100	67	90	59	91	58
V 24	145	90	124	82	120	80	114	73	115	72
V 25	117	75	105	70	104	70	101	66	100	68
V 26	125	75	112	73	111	69	91	58	89	58
V 27	142	88	128	87	125	78	101	66	102	66
V 28	115	71	109	67	105	63	87	55	88	56
V 29	118	71	105	68	102	67	90	57	89	58
V 30	135	85	119	79	117	76	101	66	102	68
V 31	141	84	133	83	125	81	113	79	113	78
V 32	143	81	122	78	117	76	103	66	104	65
V 33	136	84	127	81	124	78	116	73	118	74
V 34	117	72	109	69	105	67	96	63	98	63
V 35	120	77	114	75	113	74	103	68	106	70
V 36	126	86	121	80	120	78	111	74	113	75

Abstract

V 37	149	86	126	83	124	82	114	77	114	- 78
V 38	135	79	118	73	115	71	92	62	91	62
V 39	146	88	126	81	127	83	113	71	113	72
V 40	128	73	105	71	103	71	102	67	101	68
V 41	132	78	105	71	103	67	92	59	90	59
V 42	137	88	119	85	115	77	100	66	101	66
V 43	149	88	132	83	127	81	117	77	119	78
V 44	142	91	136	88	132	85	117	62	119	64
V 45	133	79	125	78	122	76	107	67	106	69
V 46	136	82	120	80	120	76	110	73	111	75
V 47	115	76	112	72	113	72	92	63	92	64
V 48	143	86	125	81	123	81	109	77	110	78
V 49	133	82	134	82	131	79	115	74	115	75
V 50	139	89	123	83	120	81	115	74	116	75