

The influence of an Osteopathic treatment on the performance of a high - performance athlete

Master Thesis in order to obtain the academic title

Master of science in osteopathy

at Donau university Krems

set down

at Vienna School for Osteopathy

from

Elke Böhmüller

translated by Mag. Jaqueline Priour

supervised by Mag. Katharina Musil

Oberalm, May 2007

DECLARATION IN LIEU OF AN OATH

I HEREBY DECLARE THAT I HAVE WRITTEN THE SUBMITTED MASTER THESIS ON MY OWN. ALL PASSAGES THAT HAVE BEEN TAKEN OVER LITERALLY OR ROUGHLY FROM OTHER PERSONS' PUBLISHED OR UNPUBLISHED WORK HAVE BEEN MARKED AS SUCH. ALL SOURCES AND AIDS I HAVE USED FOR THE THESIS ARE MENTIONED. THE THESIS OF THE SAME CONTENT HAS NOT YET BEEN PRESENTED TO ANOTHER EXAMINATION AUTHORITY.

SIGNATURE

Foreword

To what extent osteopathic treatments would affect my patients has been occupying me for years. Working as an osteopath, now and again I treat top athletes. Their main concern is, to either restore their moving dysfunctions completely or to improve their reduced functional disorder to a certain extent.

To reach a maximum of physical performance the body depends on a perfect interplay of all physical functional processes (Weibl 2004, Ruddy 1996, De Haag 2005).

One day the coach of a tri athlete asked me what I had done to his trainee because he achieved excellent results and unexpected best time. This happened one day after my osteopathic treatment. As a result I was asking myself, to what extent indeed my treatment did trigger off an increase in performance.

If the irritating physical dysfunction factors, restricting the body's harmonious functional flow, could be eliminated by osteopathic treatment, the body's specific energy compensating the "physical restriction" before the treatment, would underlie positive changes and could be finally released to strengthen the entire organism. Therefore, I advanced the hypothesis that osteopathic treatment would lead to an increase in physical performance.

As an osteopath I made it to my task to observe and understand how the interplay of those restricting dysfunctions would react and tried to choose the appropriate and most efficient therapy involving structural, chemical, and neurological plains, all fluids of the body and also approaching the psychosocial background. My big challenge and goal in osteopathic treatments is to optimise and harmonise all physical body functions.

The question I put to myself was, how I could estimate to what extent my treatment would support the patient in optimising his body functions.

The more energy a human being is emitting the fitter and physically active and ready for high performance it is, was the basic idea of my hypothesis and the beginning of my studies and scientific researches.

The point was now, to find an appropriate and technically already certified **measuring device**, that measures and indicates the human vitality functions in specific parameters and data. From the measured data I then would try to draw conclusions about how physically vital and energy active my test persons are.

If my hypothesis was right that the total energy of the organism could be significantly increased through osteopathic treatments, the energy measuring data delivered by the measuring device would prove it.

One can easily find measuring instruments constructed with the purpose to measure the energy of an organism, the market is large – but getting one that already has been in use for scientific studies and from which output data is available and published in scientific journals – was not so easy.

Going through the **field of bio photons** I found an alternative. Bio photons are light particles that are emitted by the DNA of every cell nucleus. This electromagnetic cell emission can be measured through light quantum. The cell emission is given off by every living organism and represents an essential reference size to measure the body's regulation capability. In a further aspect it is possible to draw conclusions about the energy status of cells. This light holds a high degree of organisation. It is a kind of laser light capable of transporting information throughout the body (Bischof 1995).

After doing research in the very particular behaviour of the cell emission, scientists are convinced now, that the light measured through high tech light amplifiers, represents the energy power of every inner cell and as a further consequence represents the total energy field of the entire body. Such is the energy field which controls and regulates life processes inside our bodies (Bischof 1995).

This statement results in new perspectives, where cells communicate interlinked and their functions can be controlled and influenced as well. Besides mechanical, chemical and physical processes it is now evident that also electromagnetic processes play an important role in the interaction of the body's functions.

Due to the fact that the highly costs of such a measuring device, a single person can not afford, I found myself compelled to look for other financially more reasonable alternatives.

It seemed to me now that the **GDV measuring device (Gas Discharge Visualisation)** would be an appropriate alternative.

The light emission of cells is measured with this device too, but here the emission is induced through high frequency electromagnetic fields.

Paul Dobson and Elena O’Keeffe (2001) stated, that *„the GDV image can be reasoned to reflect in a general manner both body function and mental states.“*

Beyond doubts there is evidence of connection between the GDV measurement data and specific symptoms. *„Independent clinical observations in different countries revealed the diagnostic validity of the GDV-technique...“* (GDV-Technology 2006)

Unfortunately I could not find any convincing information material or detailed documents, which scientifically approved or certified the evidence of connection between the measurement data and man’s performance capability.

Therefore, I also rejected the idea to find an answer to my question with this measuring device.

Treating top athletes and cooperating with them, I finally discovered another possibility to investigate how osteopathic treatment and physical increase of performance are connected. – This time I relied on **HRV (Heart rate variability)** measures.

I finally decided to do my measuring with HRV measurements, as according to scientific studies from Werdan (2006) and Hoos (2006), the measuring method is not just scientifically approved and certified but also the ideal application possibility to determine the physical performance capability particularly of top athletes.

Furthermore, the purchase of this measuring device would not be too expensive and measuring easy to handle.

Content

DECLARATION IN LIEU OF AN OATH	II
Foreword	III
Content	VI
1 Introduction	1
1.1.1 Autoregulation	2
1.1.2 Flexibility	3
1.1.3 Economy of Energy	4
1.2 Objectives of the Thesis	5
1.2.1 Research Question	5
1.3 Hypothesis	5
2 Fundamentals	6
2.1 Historical Overview	6
2.2 The Vegetative Nervous System	7
2.2.1 Introduction	7
2.2.2 Parasympathetic Nervous System	10
2.2.3 Sympathetic Nervous System	11
2.2.4 Interaction between Sympathetic and Parasympathetic Nervous System	13
2.3 The Influence of Respiration	14
2.3.1 Respiratory Sinus Arrhythmia	14
2.4 Interaction VNS ↔ Heart	16
2.4.1 Regulation of the Heart Rate	18
2.5 Heart Rate Variability (HRV): Analysis Method	19
2.5.1 Basic Principles	20
2.5.2 HRV Analysis Methods	23
2.5.3 Time Domain Methods	24
2.5.4 Frequency Domain Methods	26
2.5.5 Significance of HRV Measures	29
2.5.6 HRV and Performance	31
2.5.7 Influence of Age and Gender	33
2.5.8 Influence of Mental Stress	33
2.5.9 Influence of Diseases	34
2.5.10 Validity	35
2.5.11 Reliability	36

2.5.12	Limitations	36
2.6	Osteopathic Approach	37
3	Methods	40
3.1	Study Design	40
3.2	Inclusion criteria	41
3.3	Exclusion criteria	41
3.4	HRV Measurements	41
3.5	HRV Analysis	42
4	Results	44
4.1	Results Pilot study	44
4.2	Results Central study	47
5	Discussion	57
5.1	Single Subject Design	57
5.2	Discussion of Results	57
5.2.1	Discussion of Pilot study	57
5.2.2	Discussion of Central study	59
5.3	Conclusion	60
5.4	Clinical Relevance	61
5.5	Recommendations for further research	61
6	Summary	62
7	Acknowledgements	64
8	Abstract	65
9	References	68
	List of Figures	78

1 Introduction

To what extent osteopathic treatments effects the human organism and how far reaching the consequences in the body itself are, this question is not just of great significance to the therapist but also for the patient himself.

Treating top athletes is a very special case. For this group of patients it is not just essential to recover quickly from pains and injuries but also to what extent the osteopathic treatment affects the physical performance capability.

As a consequence I come up with the question, what influence osteopathic treatment has to the increase in physical performance and how exactly the latter can be technically measured.

Going through a lot of scientific literature I hardly found any data or documents that described or profoundly examined the connection between sports and osteopathy.

It is exactly in the field of extreme sports where not only physical long - term changes are relevant but also the short- term effects of a certain treatment are decisive. Not only the increasing performance factors need to be considered carefully but also the decreasing reactions after a certain treatment demand detailed examination.

The HRV device is an ideal alternative to examine the connection between the physical performance capability and osteopathic treatment, in a certain period of time.

The scientific study from Garet et al (2004) shows with great significance the evident connection between the increase of HRV-measuring data and the improvement of physical sports performance.

HRV measuring is easy to handle and can be done at anytime. Short-term effects on the vegetative nervous system (VNS) after osteopathic treatment can be measured in an uncomplicated way (Mück-Weymann and Beise, 2005).

After Gutenbrunner (2005) the vegetative nervous system is the most extensive regulation system of the body. The VNS regulates and modulates under others all functions of the heart and blood circulation, digestion, water balance and brain activities. For that reason, far-reaching conclusions can be drawn thanks to this measuring method.

I treated the following three important preconditions as a consequence: Regulation capability,

flexibility and energy need. These 3 factors are indispensable for maximum physical performance and fitness.

1.1.1 Autoregulation

Just like every living organism, also a human being has its own specific inner body networks and self regulating systems. Such delicate systems and networks help to maintain the body's **homeostasis** and work after a certain physical economic-principle, which keeps the physical functions with the smallest loss of energy in balance (Pischinger 1990).

Here I would like to point out that after the latest research reports the idea of the homeostasis, means the organism tries to keep the body's parameters at the same level, is questioned and replaced by the concept of **homodynamic**. Such regulation capabilities are essential and basic preconditions for the self-healing properties and for regeneration of the body (Moser 2004).

Going through chapter 2.2 "The Vegetative Nervous System" (VNS), it becomes clear that the regulation processes of the vegetative nervous system is not a static process but a dynamic one.

Using regulation systems, VNS is one of them, the body is constantly occupied to balance out irritations and their further taking effects on the body to achieve homodynamic balance. Irritations which are disturbing the inner balance are caused by external or internal influences and changes.

For such adaptation processes a complex interacting regulation mechanism is indispensable (Wühr 2007).

Constant and intensive irritations result in **adaptations and compensations**, what equally represents the regulation mechanism, which tries to balance out the disturbing influences and changes and in a further consequence re-establishes a homodynamic condition. If not immediately balanced out on the disturbing spot, permanent compensation can lead to a kind of physical chain reaction, which are named compensation chains.

Adaptation and compensation cause physical deviations in form and function (such as deviations of joint structures or organic dysfunction) and, as a further consequence, can lead to acute or chronic illness (Wühr 2007). Osteopathic treatments try to alleviate or even eliminate such form and functional deviations to contribute to homodynamic processes.

It is not enough to rely on an ordinary cause and effect reasoning to completely understand the complex physical processes, due to the fact that the physical regulation mechanism of the organism is very complicated and interacting in itself (Pischinger 1990). Physical feedback processes come into play and return the effect to the original cause - which can also change the original cause itself (Bischof 1995).

If more than three compensation chains cross each other, the consequences of their reactions can neither be estimated, foreseen nor understood anymore. (Wühr 2007)

To mention the irreversible thermodynamics from Prigogine, I would like to point out that all systems are more or less open systems (including biological systems, such as human being), which are capable to exchange energy, information and material with their surrounding environment (Pischinger 1990). The systems theory and cybernetics also emphasise that parts of a unity can not be seen as separated or isolated – the interacting unity in itself should definitely be taken under consideration (Bischof, 1995). “*Man is more than the sum of his parts and their interactions,*” so Irving Korr (1963, p.27)

As soon as disturbing factors affect the body, regulation mechanisms start to balance out irritating deviations with the least expenditure of energy. (Pischinger 1990)

Regulation dysfunctions play a decisive role in the origin of illness. Pischinger states in 1990, that with all acute and chronic illness, regulation dysfunctions are found.

The vegetative nervous system is the most complex and extensive regulation system of the body (Gutenbrunner 2005), which also regulates and modulates under others the functions of the heart and blood circulation, digestion, brain, water balance and controls movement. It is through constant adaptations within those body systems that physical performances as well as regeneration processes are ensured. (De Haas 2005)

1.1.2 Flexibility

The vegetative nervous system is a very important control system for all ongoing physical regulation processes of the body. It enables the body to react flexibly to changes and activates adaptation towards occurring changing conditions in the surroundings. (Weeber 2007, Gutenbrunner 2005)

This flexibility of the vegetative nervous system can be measured by the HRV device (Werdan et al. 2006, Hoos 2006).

To balance out disturbing factors from inside as well as from outside the body, the body firstly reacts with adaptation and compensation and then tries to achieve and restore its ideal functional balance (Wühr 2007).

Adaptation and compensation demand very supple body **flexibility** so that the body can react promptly on external as well as internal changes. Body functions are adjusted as a consequence to changing conditions. Adaptations to these changes also mean a higher expenditure of energy. (Weibl 2004, Wühr 2007).

Isn't it one aim of an osteopathic treatment to alleviate or if possible eliminate adaptations and compensations, in order to reduce the energy need for body regulation? (see 2.6, Osteopathic Approach).

Flexibility and adaptation capability is indispensable for living and for the survival of organism. This can be applied not just in the fauna and flora but also in the world of human being (Bischof 1995).

1.1.3 Economy of Energy

If disturbing factors affect the body, the body firstly experiences stress, what means more or less loss of energy. As soon as the stress evoking situation or cause is eliminated or outbalanced, the body regenerates again. (Weibl 2004, Wühr 2007).

This means that the body needs more energy for adaptation and compensation than would be necessary for the same function without the influence of disturbing factors.

Therefore the conclusion is drawn: the more disturbing influences which restrict the harmonious interplay of the physical functions the body needs to balance out, the more energy is needed to establish physical homodynamic balance. (Weibl 2004, Wühr 2007)

Adaptation reactions are also absolutely necessary if it comes to physical maximum performance as it is the case in extreme sports. To achieve high athletic performance, an adequate adaptation to the increasing need of energy is indispensable. Adaptation to the increasing need of energy occurs through the activation of the sympathetic nervous system which increases the heart rate as well as the contraction power of the heart to provide body cells with an optimum of oxygen and nutrient. (De Haag 3005, p.43)

A smooth interplay between all ongoing regulatory mechanism processes of the body is indispensable to achieve high physical performance in the field of high-performance athletes. After Guttenbrunner (2005) the vegetative nervous system represents the most complex regulation system of the body. Changes within the vegetative nervous system can be measured by the HRV measuring device (Mück-Weymann and Beise 2005).

1.2 Objectives of the Thesis

This single subject study is investigating if an Osteopathic treatment can increase athletic performance.

An Osteopathic treatment usually aims to enhance or restore health in the patient (Haberl 2006). It also aims to restore homodynamic properties. If the dynamics of all body systems (see Auto regulation, 1.1.1) is well-balanced, it permits a continuous adjustment to inner and outer influences, and therefore increases global body regulations and peak performance. (This view was also put forward by J.P.Barall in a one-to-one interview on 16.2.07 in Gars/Kamp)

The vegetative nervous system (VNS) also known as the autonomic nervous system (ANS) is one of the most extensive regulatory systems in the human body and reflects global body regulations (Gutenbrunner 2005). As HRV represents one of the most promising quantitative markers of autonomic activity (Task force 1996), HRV was used to investigate this question.

1.2.1 Research Question

Is it possible to enhance the performance of athletes with Osteopathic treatments?

1.3 Hypothesis

Heart Rate Variability (HRV) can be increased in high-performance athletes by Osteopathic treatments.

2 Fundamentals

2.1 Historical Overview

Already 1700 years ago, Wang Shue, a Chinese physician realised the importance of heart rhythms to predict ill-health, when he stated: *“If the heart-beat becomes as regular as the knocking of a woodpecker, or the dripping of the rain on a roof, the patient will die within four days.”* (Mück-Weymann and Beise 2005)

Observing beat-to beat rhythm shifts of the heart became one of the central components of medical diagnostics in Traditional Chinese Medicine.

For many centuries, physicians associated these beat-to-beat rhythm shifts with their patients’ age, illness or psychological condition (Engel 2006).

“The clinical relevance of HRV was first appreciated in 1965 when Hon and Lee6 noted that foetal distress was preceded by alterations in interbeat intervals before any appreciable change occurred in heart rate itself.” (Malik et al 1996, Background)

Since Wolf et al. (1978) found out that there is a relation between a reduced HRV and a high risk of post-infarction mortality, a wide range of HRV analysis methods emerged. In 1996 a standard of measurements and physiological correlates of HRV and clinical applications was developed by a task force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology, which is now to be found in the list of references of many studies, where HRV-measures are applied.

In the last years, heart rate variability has been widely used as a measure of changes within the vegetative nervous system in physiological, psychological and clinical examinations. Only the development of technical measuring devices made possible research in detail.

2.2 The Vegetative Nervous System

2.2.1 Introduction

The peripheral nervous system is divided into the **somatic nervous system** and the **vegetative nervous system** (VNS). The somatic nervous system with its sensory and motor components is under voluntary control and most of the impulses reach our awareness.

The predominantly efferent VNS, also known as the visceral or autonomic nervous system, elucidates largely automatic or reflex responses, and transmits impulses from the central nervous system (CNS) to the peripheral organ system. These impulses often do not reach our consciousness and for the most part are not subject to voluntary control (Bakewell 1995).

Besides the mostly present efferent fibres of the VNS, there are also afferent fibres, regulating vasomotor and respiratory reflexes. These are, for example, the baroreceptors and chemoreceptors in the carotid sinus and aortic arch (Duus 1987). This information is carried to the CNS by afferent fibres of major autonomic nerves such as the vagus, splanchnic or pelvic nerves to the controlling centres of the brain (mainly the medulla, pons and hypothalamus). From these centres efferent impulses are conveyed to all parts of the body by the parasympathetic and sympathetic nerves, the two divisions of the VNS (Bakewell 1995).

*“The **parasympathetic** division protects the internal environment, that is, it is trophotropic because of its nourishment-providing function. The **sympathetic** division, by contrast, is ergotropic [work-preparing], influencing the performance of the whole body in response to the environment.”* (Frymann et al.1992, p.265).

Fig. 1 shows the effects of sympathetic and parasympathetic stimulation on different organs.

Responses of major organs to autonomic nerve impulses		
Organ	Sympathetic Stimulation	Parasympathetic Stimulation
Heart	Increased heart rate β_1 (& β_2)	Decreased heart rate
	Increased force of contraction β_1 (& β_2)	Decreased force of contraction

	Increased conduction velocity	Decreased conduction velocity
Arteries	Constriction (α_1)	Dilation
	Dilation (β_2)	
Veins	Constriction (α_1)	
	Dilation (β_2)	
Lungs	Bronchial muscle relaxation (β_2)	Bronchial muscle contraction
		Increased bronchial gland secretions
Gastro-intestinal tract	Decreased motility (β_2)	Increased motility
	Contraction of sphincters (α)	Relaxation of sphincters
Liver	Glycogenolysis (β_2 & α)	Glycogen synthesis
	Gluconeogenesis (β_2 & α)	
	Lipolysis (β_2 & α)	
Kidney	Renin secretion (β_2)	
Bladder	Detrusor relaxation (β_2)	Detrusor contraction
	Contraction of sphincter (α)	Relaxation of sphincter
Uterus	Contraction of pregnant uterus (α)	
	Relaxation of pregnant and non-pregnant uterus (β_2)	
Eye	Dilates pupil (α)	Constricts pupil
		Increased lacrimal gland secretions
Submandibular & parotid glands	Viscous salivary secretions (α)	Watery salivary secretions

Fig. 1 (Bakewell 1996, modified after Böhmüller 2007)

The CNS and the VNS play an important roll to serve as a major communication system in disease, as well as in health, in mediating influences among organs (Korr 1963).

The VNS is primarily responsible for the physiologic response to environmental change, which includes the regulation of heart rate, blood pressure, force of contraction and relaxation of smooth muscle in various organs, constriction and dilatation of blood vessels, visual accommodation, pupillary size, secretions from exocrine and endocrine glands, and other changes to which all individuals are exposed (Bakewell 1995).

Therefore, the VNS can be seen as a **regulatory system** for individual organ function and homeostasis, respectively homodynamic (see 1.1.1 Autoregulation).

All efferent nerve fibres, leaving the CNS, except for those, which innervate skeletal muscle, are constituted by vegetative nerves (Bakewell 1995).

That is why changes in the VNS, will have **responses in total body functions** (Beal 1985).

It is evident, that we are not constantly aware of the activity of the vegetative nervous system.

The normal functioning of the autonomic nervous system day and night, from heart-beat to heart-beat, plays a largely unconscious but vital role in our livelihood and the understanding of regulatory processes in training, as well as in Osteopathic treatments.

Changes within the VNS can be measured by HRV (Werdan 2006, Hoos 2006, Cerruti et al.1995)

The anatomical and physiological basis of the VNS will be described in short in the following chapters and a review will be given in Fig. 2.

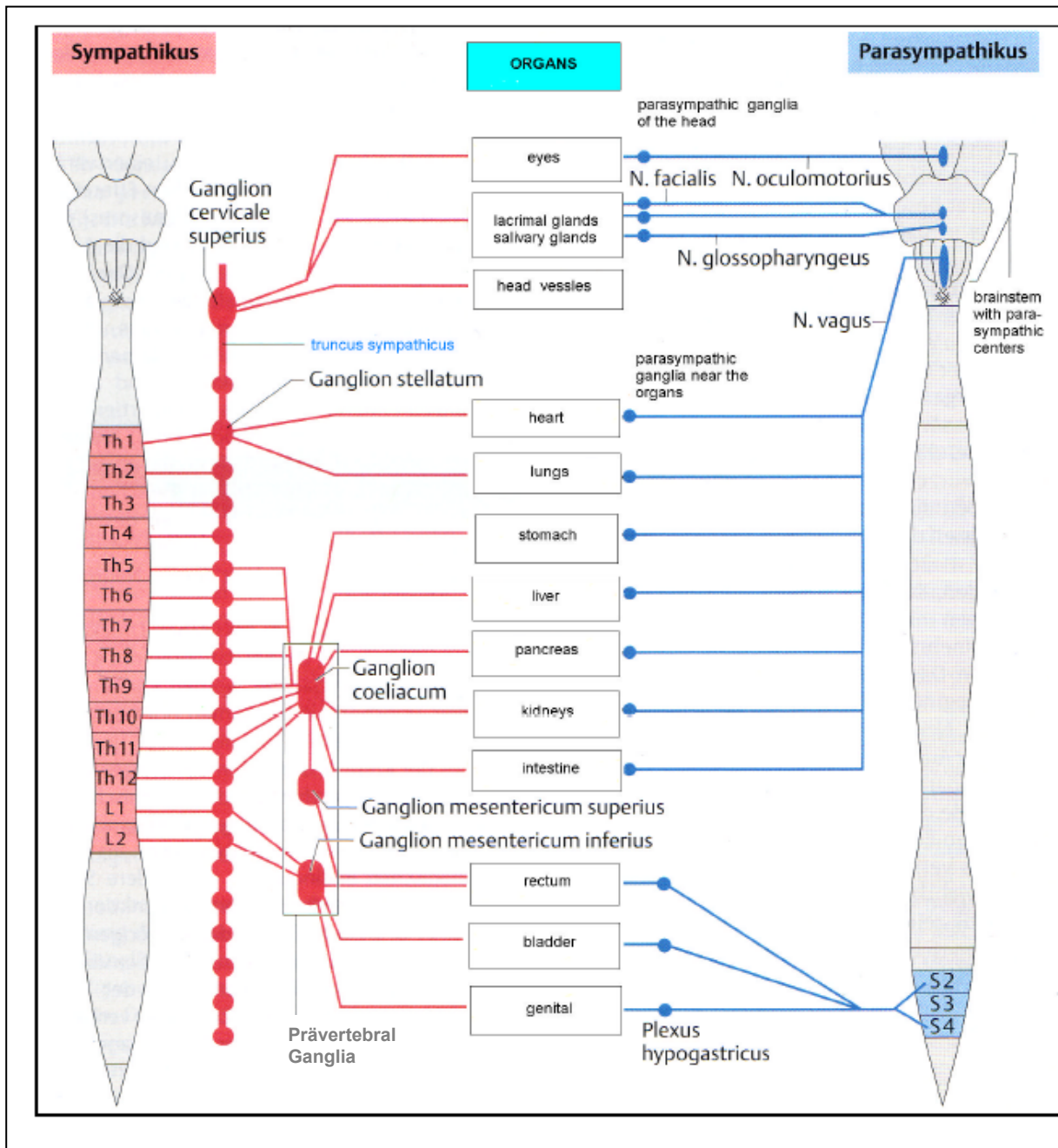


Fig. 2 Origin and service areas of the VNS (Slater 2000 after Hamill 1997, modified after Böhmüller

2.2.2 Parasympathetic Nervous System

The parasympathetic nervous system is, in physiological terms, mainly concerned with conservation and restoration of energy (Bakewell 1996). It causes a reduction in heart rate and blood pressure, decreases cardiac output, respiratory volume and basal metabolism, vascular dilatation, perspiration, reduces contraction of the urinary bladder, and facilitates digestion and absorption of nutrients, and consequently the excretion of waste products (Duus 1987).

The preganglionic neurons of the parasympathetic nervous system (PNS) arises from the cell bodies of the motor nuclei of the cranial nerves III (oculomotor nerve), VII (facial nerve), IX (glossopharyngeal nerve), which affect the pupil and salivary gland secretion, the vagus nerve (X) in the brain stem which carries fibres to the heart, lungs, stomach, upper intestine and ureter and from the second, third and fourth sacral segments of the spinal cord, which innervate the distal colon, rectum, bladder and reproductive organs (Duus 1987).

Therefore it is also known as the cranio-sacral outflow (Bakewell 1995).

The parasympathetic influence on heart rate is mediated via release of Acetylcholine (Ach) by the vagus nerve (X) (Duus 1987).

Ach is not only the neurotransmitter for parasympathetic activity but also for sympathetic preganglionic synapses, some sympathetic postganglionic synapses, the neuromuscular junction (somatic nervous system), and at some sites in the CNS. Nerve fibres that release Ach from their endings are described as cholinergic fibres (Bakewell 1995).

2.2.3 Sympathetic Nervous System

In contrast to the parasympathetic system, the sympathetic system enables the body to be prepared for fear, flight or fight. Sympathetic responses include an increase in heart rate, blood pressure and cardiac output, a diversion of blood flow from the skin and splanchnic vessels to those supplying skeletal muscle, increased pupil size, bronchiolar dilation, contraction of sphincters and metabolic changes such as the mobilisation of fat and glycogen (Bakewell 1995).

The cell bodies of the sympathetic preganglionic fibres are situated in the lateral horns of the spinal segments T1-L2, the so called thoraco-lumbar outflow. The myelinated preganglionic fibres enter the sympathetic ganglia, which are mainly arranged in two paravertebral chains, lie anterolateral to the vertebral bodies and extend from the cervical to the sacral region. They are called the sympathetic ganglionic chains.

The short preganglionic fibres which enter the chain make a synapse with a postsynaptic fibre either at the same or at a higher or lower dermatomal level. The longer unmyelinated postganglionic fibres usually return to the adjacent spinal nerve and travel to a large extent with the blood vessels to the effectors organ (Bakewell 1995).

“The sympathetic nervous system contributes postganglionic fibres to the vasculature of the body and is therefore the **vasomotor system** of the body.” (Stiles 1976, p.330)

Some preganglionic fibres do not synapse in the sympathetic chains but terminate in separate cervical or abdominal ganglia, or travel in the greater splanchnic nerve and directly synapse with chromaffin cells in the adrenal medulla.

The adrenal medulla responds to sympathetic nervous impulses by transforming the neural impulses into hormonal secretion, releasing **Adrenaline**. In situations involving physical or psychological stress, such as hypoglycemia, hypothermia, or fear, much larger quantities are released into the bloodstream. An increase of pulse rate and blood pressure, dilatation of blood vessels in skeletal muscles and a stimulated metabolism are a few reactions as a consequence of an increase of adrenaline in the blood stream (Bakewell 1995).

The combination of the sympathetic nervous system and the adrenal glands (also called the “sympathoadrenal” or “sympathico-adrenal” system) came to be understood as a single emergency system of the body like in “fight-or-flight” reactions.

As discussed above (see 2.2.2 Parasympathetic Nervous System), **Acetylcholine** (Ach) is the neurotransmitter which is used by the *parasympathetic nervous system*. It relays control signals from the preganglionic to the postganglionic cells in the ganglia by binding to a nicotinic receptor. Ach also supports the release of Adrenalin from the adrenal gland. One of its functions is the decrease of the pulse rate.

Noradrenalin (norepineprine) is the main neurotransmitter, used by the *sympathetic nervous system*, which is present in presynaptic nerve terminals as well as in the adrenal medulla. Nerve fibres that release noradrenalin from their endings are described as **adrenergic fibres**. Noradrenalin is released by sympathetic nerve terminals and acts locally on nearby cells. As an example, it is released from the sympathetic nerve terminals in the heart and acts on the nearby heart muscle cells.

(There is an exception, where sympathetic nerves also release acetylcholine. In sweat glands, sympathetic nerves release Ach as the signal for sweating.)

2.2.4 Interaction between Sympathetic and Parasympathetic Nervous System

The VNS is primarily involved in reflex arcs, involving autonomic or somatic afferent fibres, that may convey stimuli from pain receptors, mechanoreceptors or chemoreceptors in the heart, lungs, gastrointestinal tract etc., and efferent limbs, causing contraction of smooth muscle in certain organs (e.g. blood vessels, glands, bladder, gastrointestinal tract) and influencing the function of the heart and lungs (Bakewell 1995).

Most organs are innervated by fibres from both divisions of the VNS. The activation of either sympathetic or vagal outflow is usually accompanied by the inhibition of the other. Gellhorn regarded these two systems as strictly separated antagonists. This view has been more and more questioned during the last decade, as areas were discovered, where the sympathetic nervous system and the parasympathetic nervous system work rather complementary than antagonistically (Hugdahl 1996, cited after Engel 2006).

The fact that the two branches of the VNS do not always work reciprocally is shown in case of the “diving reflex”, where vagal activation causes bradycardia whereas sympathetic activation causes peripheral vasoconstriction at the same time (Malliani 1999).

The VNS does not only react on physical stimuli but also on emotional changes. Psychological and physical adjustment responses are inextricably connected in a complex manner. In which way, **behaviour reactions** lead to physical responses, is distinct (De Haas 2005).

The fact that the body does not always respond to changes in the same way, not even for one and the same person, will be shown in the following example. An increase in arterial blood pressure induced by a vasopressor drug usually provokes bradycardia during quiet resting conditions, whereas under an emotionally charged condition or under physical exercise a comparable increase in arterial pressure elicits tachycardia.

The short summary of the functioning of the VNS should remind of the importance of this regulatory system to coordinate the adaptation of the body to outer and inner changes, in daily life as well as in case of emergency.

2.3 The Influence of Respiration

2.3.1 Respiratory Sinus Arrhythmia

Blood pressure (BP) and heart rate (HR) are continually varying with thoracic respiration. With thoracic inspiration, heart rate accelerates, with expiration it slows down (Karemaker 1999). This matter of fact is shown in Fig. 3: “*Simultaneous recordings of instantaneous ECG (black) and ventilation, recorded as intra-peritoneal pressure (red; the upward spike indicates lung expiration). The interval tachogram (blue) describes the change in timing between heart beats (nominal scale), and shows that the heart beat is increased during lung inflation.*” (Campbell et al. 2006)

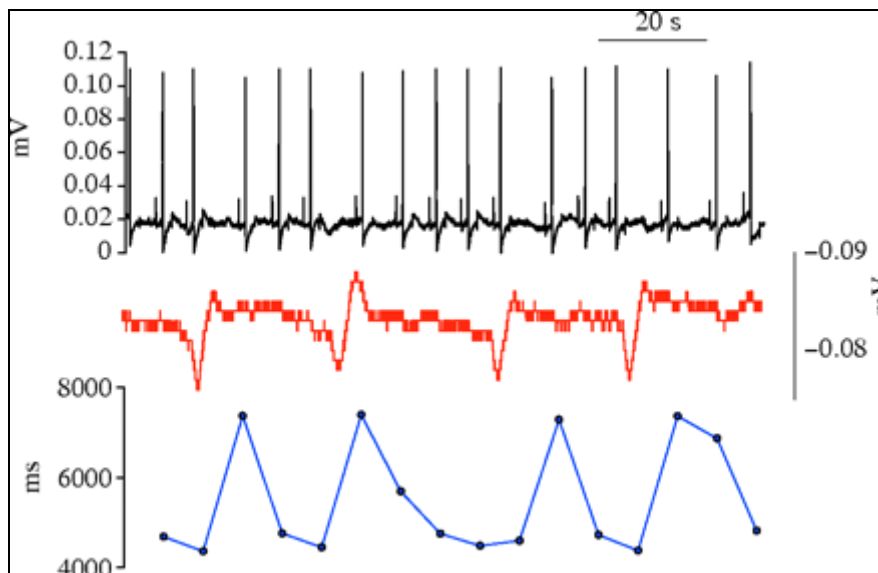


Fig. 3 (Campbell et al. 2006, Results)

Recently, heart rate oscillations have extensively been studied thanks to the easy availability of ECG recordings.

The fact, that inspiration is accompanied by an acceleration of the heart rate can be considered as well established. The mechanism, underlying this respiratory cardiac arrhythmia is still an unsettled matter (Anrep et al. 1936).

A widely spread view for the explanation of the respiratory sinus arrhythmia (RSA) is that the acceleration and deceleration of the heart rate is caused by arterial blood pressure fluctuations through the arterial baroreflex, induced by respiration. Another explanation emphasises the

origin of the RSA in the central nervous system, as the vagus nerve controls respiration as well as heart rate (Taylor and Eckberg 1996). Taylor and Eckberg concluded, after measures of RR-intervals were taken during 7 minutes of controlled-frequency breathing in both the supine and 40° passive head-up tilt positions, “*that respiratory sinus arrhythmia may be mediated by the baroreflex only when the mechanical effects of respiration on arterial pressure are greater than those in supine humans.*” (Taylor and Eckberg 1996, Conclusion)

Recent neurophysiologic research found that “*RSA is caused by interneural connections in the brainstem between the nuclei controlling the heart rate and the respiration.*” (Engel 2006 p.51)

Another interesting correlation between the heart rate and the respiration rate is the ratio between heart rate and respiratory rate.

During deep sleep, the ratio of heart rate and respiratory rate averages around 4:1 in healthy people, whereas during the day, it can alter between 2:1 and 7:1.

Moser (2004) describes this phenomenon as “Normalisierungseffekt”, which could be interpreted as an expression of the homodynamic behaviour of the cardio respiratory system. (See 1.1.1 Auto-regulation)

Fig. 4 demonstrates the ratio between heart rate and respiratory rate (QP/A) in 5 different subjects, out of a sample of 89 subjects. During daytime, the ratio ranges from about 2, 5 / 1 to about 6, 5 / 1, whereas during night time, especially during deep sleep from 3 to 6 o'clock, the quotient in all subjects converges to around 4:1.

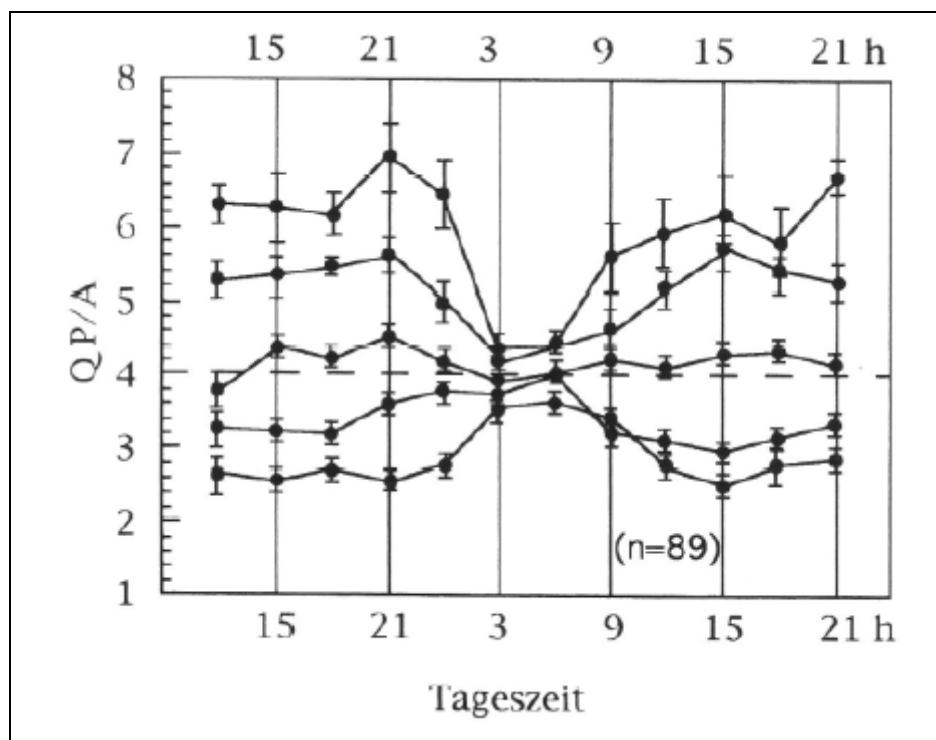


Fig. 4 (Moser 2004, p.30)

The connection between the heart rate and respiration rate gives rise to the assumption that RSA can be potentially influenced by thoracic respiration, which could be a disturbing factor in HRV measures. Researchers' views on the matter of respiration correlating with HRV measures are divergent. An interesting study on this issue was made by Hirsch and Bishop (1981), when they investigated the influence of controlled and spontaneous breathing on HRV. They “....compared HRV between spontaneous breathing and controlled breathing and found that the HR fluctuations during spontaneous breathing were within the 95% confidence limits of those measured during controlled breathing. Therefore they concluded that controlled breathing is not a prerequisite for correct HRV measurement.” (Engel 2006, p.53)

Out of this reason, respiration rate is not being taken into account in this study, as it was not in many other studies, using HRV measures.

2.4 Interaction VNS ↔ Heart

Traditionally, the interaction between the heart and the brain was seen from a rather one-sided perspective. Scientists were focusing primarily on the heart's responses to the brain's commands. Recent researches showed this interaction as a **two-way dialog**, a dynamic,

interaction between the heart and the brain. Furthermore, messages sent from the heart to the brain can also affect performance.

Research has shown that the heart communicates to the brain not only through neurological, biochemical and biophysical ways, but also energetically, through electromagnetic field interactions. The heart, as the most powerful generator of electromagnetic energy in the human body produces a rhythmic electromagnetic field, which can be measured in the form of an electrocardiogram (ECG) (McCraithy 2001).

ECG recordings are made for about 100 years. *“The beating of the heart produces a huge electric current that flows throughout the body, mainly because blood and extracellular fluids, with their high salt content, are extremely good conductors of electricity- and electricity is obviously a form of energy.”* This is why *“the electrocardiogram is a diagnostic tool rooted in energy medicine”*. (Oschman 2003, p.1and 2)

This electromagnetic field can be detected anywhere in the body and also a number of feet away from the body (McCraithy 2001), as it is displayed in Fig. 5.

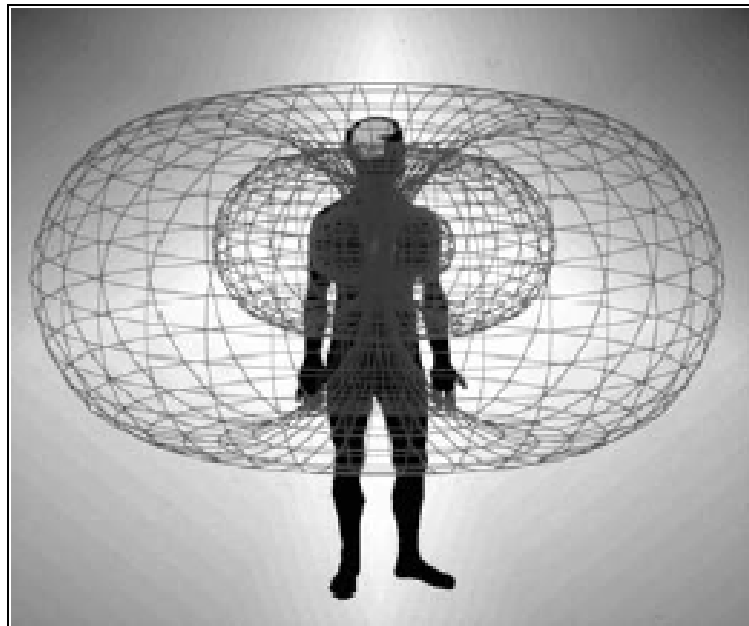


Fig. 5 The Heart's Electromagnetic Field (McCraithy 2001, p.20

McCraty et al (1996) found another interesting issue about the influences of the heart's electromagnetic field. They discovered, *“...that the **electromagnetic signals** generated by the heart have the capacity to **affect others around us**. Our data indicate that one person's heart signal can affect another's brainwaves, and that heart-brain synchronization can occur between two people when they interact.”* (Mc Craty et al. 2001, p.21)

The heart does not only influence the VNS by electromagnetic and homodynamic information, but also the central nervous system, respiration, thermal regulation, the renin-angiotensin system (Vestweber and Hottenrott 2001) and even people the people around us, as mentioned above.

By means of the VNS all of these systems are influencing the variability of the heart rate indirectly (Vestweber and Hottenrott 2001).

Therefore, the heart, as an organ located in the centre of the body, is predestined to display homodynamic processes (Moser 2004).

2.4.1 Regulation of the Heart Rate

The rhythm of the heart is largely under control of the VNS and can be altered by the VNS. (Task force 1996) (See 2.2, the Vegetative Nervous System)

Via stimulation of the cardiac beta-1 receptors, adrenaline and noradrenalin are being released through activation of **sympathetic** nerve fibres, which supply most of the muscle of the heart.

As a consequence, acceleration of diastolic depolarisation causes the heart rate to increase.

The influence of the **parasympathetic** nervous system on heart rate is based upon the release of Acetylcholine by the vagus nerve, which is found plentiful in the sinus node. The stimulation of muscarinergic receptors brings an increase of the conductivity of the heart's cell membranes. The deceleration of diastolic depolarisation makes the heart rate decrease (Schaefer 2006).

Short-term regulations are carried out by the fast-conductive fibres of the **vagal nerve**. The chemical reactions of the vagal stimulation on the sinus node are only short-lived, whereas **sympathetic impulses** are rather **long-lasting**, but do not cause reactions until 10 seconds. (Schaefer 2006)

This is why the heart reacts much faster on subtle vagal, than on sympathetic stimulations. Therefore, changes in vagal tone have an immediate impact in fine-tuning the variability of the heart rate. This explains the fact, that under normal conditions, the influence of the parasympathetic nervous system is the more important influence on the heart (Kestin 1993).

Heart rate can also be changed by circulatory substances. During stress, catecholamine, like adrenaline, is being released, which cause an increase in heart rate. Drugs can also be a cause for a change in heart rate (Kestin 1993).

Long-term mechanisms, regulating the heart rate include baroreflexes, chemoreflexes, thermoregulatory reflexes and the reflex control of blood pressure. (Schaefer 2006)

2.5 Heart Rate Variability (HRV): Analysis Method

Heart rate variability (HRV) is a measurement of the constant interaction between the sympathetic and parasympathetic activity in autonomic functioning (Task force 1996, Lau et al 2005). HRV measures describe variations of instantaneous heart rate (HR) and consecutive cardiac cycles (R-R intervals), (Task force 1996).

As shown in Fig. 6, HRV is influenced by different systems in the body. Malliani (1999) describes HRV “...as the sum of elementary oscillatory components, defined by their frequency and amplitude.” (Malliani 1999, p.4)

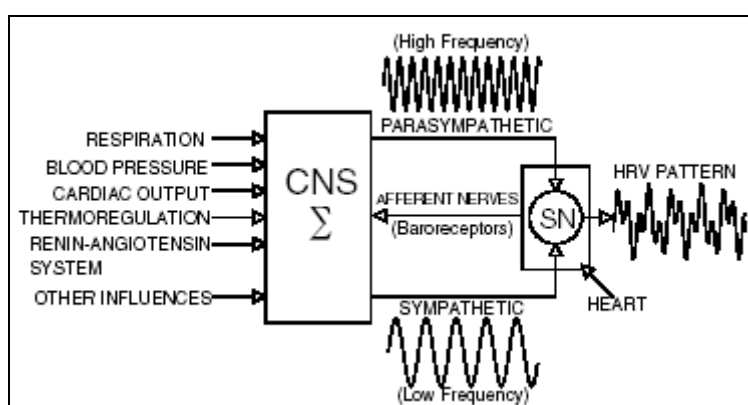


Fig. 6 (Mc Craty et al. 2001, p.14)

Figure 6 “...illustrates the nervous system links between the heart and brain. The sympathetic branch speeds heart rate while the parasympathetic slows it. Heart rate variability is due to the interaction between the two branches of the nervous system and the afferent signals sent from the heart to the brain (baroreceptor network).” (Mc Craty et al. 2001, p.14)

As HRV is created by organ systems like the VNS, the heart or the circulatory system, **dysfunctions of different organic systems can be seen in a change in HRV** (Mück-Weymann and Beise 2005).

In recent years, HRV gained increasing interest in sports and training sciences as well as in medicine.

In sports and training sciences, HRV is currently used for the non-invasive assessment of autonomic changes associated with short-term and long-term endurance exercise training in both leisure sport activities and high-performance training.

In medicine, HRV is an important tool especially for the risk stratification after myocardial infarction and the assessment of diabetic neuropathy (Task force, 1996).

In this research setting heart rate variability is used to assess autonomic function, as **HRV represents one of the most promising quantitative markers of autonomic activity** (Task force 1996) and presents a measure for the adaptability of the body to outer and inner changes. (Mück-Weymann and Beise 2005)

2.5.1 Basic Principles

Assuming that a healthy heart beats at regular intervals, it is surprising, that under resting conditions, the rhythm of the heart show irregular intervals (Mück-Weymann and Beise 2005, Mc Craty et al. 2001).

HRV derived from long time electrocardiogram (ECG) monitoring, where beat-to-beat changes in heart rate are measured. Compared to conventional ECG recordings, HRV measures provide more detailed information on changes in beat-to-beat variations between consecutive heart beats (Moser 2004). McCraty et al. (2001) describes HRV “...as a *dynamic window into autonomic function and balance.*” (McCraty et al. 2001, p.1)

The variability of beat-to-beat changes within consecutive cardiac cycles (R-R intervals) in the heart rate is shown in Fig. 7. Even without external demands, such as exercise, the heart rate (HR) continuously varies on a small scale. This is due to the sympathetic and parasympathetic activity of the autonomous nervous system.

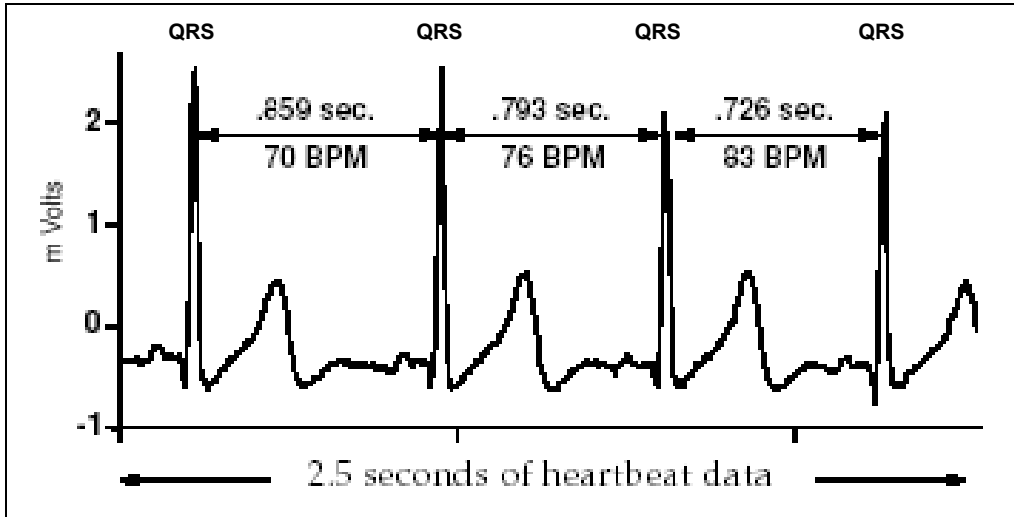


Fig. 7 (McCarty 2001, p.13, modified after Böhmüller 2007)

Fig. 8 represents the method used for spectral analysis of HRV measures. From the ECG, shown on the top left panel, the individual R-R intervals are computed (T1-T6). This results in the tachogramm (top right panel) from which power spectral density is computed (left bottom panel). The associated power of the spectral components, high frequency, low frequency and very low frequency are recognised and printed out (right bottom panel).

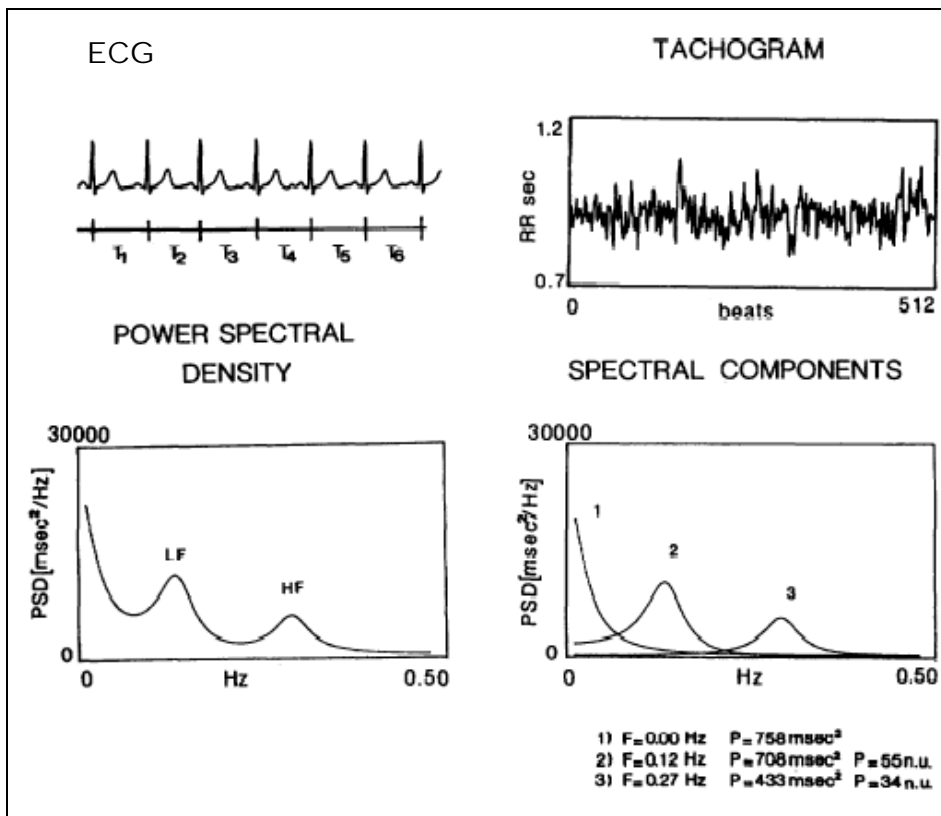


Fig. 8 Malliani (1991) p.483, modified after Böhmüller 2007

Heart rate constantly changes with respiration. It accelerates with inspiration and slows down with expiration, which is called respiratory sinus arrhythmia (see 2.2., The Influence of Respiration).

This irregularity represents a natural perturbation of the cardiovascular system, which is an example for the homodynamic processes within the body. This **variability provides stability** within the system.

The rhythm of the heart getting too monotonous is an indication of overstraining of the organism. This is being pointed out in HRV measures in a reduction of the variability between consecutive R-R intervals (De Haas 2005).

This issue is also interesting from a non physical point of view.

If rhythms in life become too monotonous, vitality is reduced. Inelasticity, inflexibility and rigidity are overtaking. The capacity of adaptation decreases.

One of the principles of Osteopathy emphasises: **“Life is Movement.”** In Osteopathy, the absence of movement (e.g. physically, bio chemically...) is seen as a cause of disease. This principle could also be interpreted figuratively. Life is indeed more than just movement. Life is movement within movement, life is flexibility. Conclusions of former studies using HRV measures, demonstrated that it is not movement alone which sustains health. It is the flexibility within this movement, which is essential for the body to adapt to changing influences (McCraty et al 2001).

It has become apparent, that too little variation in the sequence of beat-to-beat intervals can be pathological, whereas a large degree of variation is also unfavourable to efficient physiological functioning. *“The normal variability in heart rate is due to the synergistic action of the two branches of the ANS, which act in balance through neural, mechanical, humeral and other physiological mechanisms to maintain cardiovascular parameters in their optimal ranges and to permit appropriate reactions to changing external or internal conditions.”* (McCraty et al 2001, p.13)

According to the investigations made for this study (see 8, References), no absolute values to be interpreted as normal could be found in the literature.

Therefore, HRV measures should be seen on an individual level.

2.5.2 HRV Analysis Methods

The signal that is analysed in HRV measures is not the heart rate itself but the sequence of beat-to-beat intervals (R-R intervals) also called normal-to-normal (NN) intervals or the intervals between consecutive R waves of QRS complexes (Task Force 1996).

Chest electrodes are used to obtain a QRS complex to quantify HRV (see 2.5.2 Basic Principles Fig. 7.)

The intervals between the peaks of the sharp R waves (expressed in ms) are stored in a computer and can be visualised in the tachogram after analogue-to-digital conversion.

These intervals may vary from ~1200 ms at rest and ~300ms during maximal exercise, corresponding to heart rate from 50beats/min and 200beats/min. By the high performance athlete of this study, the intervals reached up to 2000 ms at rest. Heart rate variability ranges between ~50 ms at rest and ~6ms during extreme tachycardia.

In short term recordings, as used in this study, sections of several hundred beats, or recordings over a defined time, usually 5 min., are recorded. Simple descriptive statistics are computed, providing time-domain estimates of HRV (Malliani 1999). (See 2.5.3 Time Domain Methods)

After the HRV measures are digitised, the complete signal should be carefully edited by visual checks and manual corrections if the data are statistically analysed (Malik et al. 1996). Artefacts have to be identified and replaced by interpolation from the previous and following sinus intervals (Schaefer 2006).

Fig. 9 summarizes the individual steps which are necessarily used, when processing the ECG signal in order to obtain data for HRV analysis.

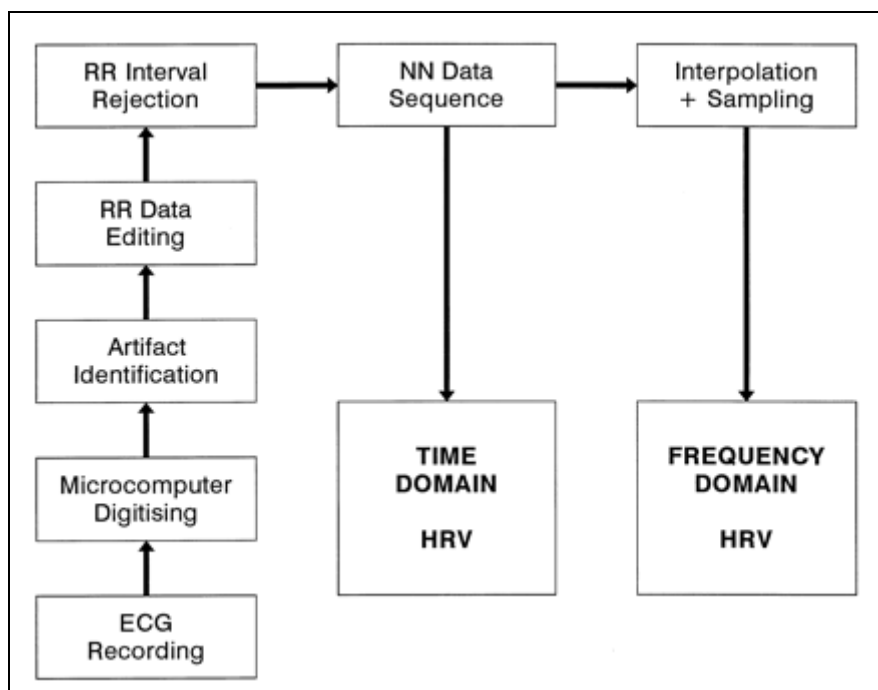


Fig. 9 (Task Force 1996, Standard measurement of HRV.)

To discriminate and quantify the relative power of the sympathetic and parasympathetic activity, HRV data have to be transformed into power spectral density, where the HRV signal is reduced into its constituent frequency components (McCarty et al. 2001). Different mathematical possibilities are available for this frequency analysis. Many studies have relied on the Fast Fourier Transform. This study uses the autoregressive auto regulation, as it is supported by Polar Precision Performance software 4.0 (Polar Electro Oy, Finland). (See 2.5.4., Frequency Domain Methods and 3.5 HRV Analyses)

Variations in heart rate may be evaluated by a number of methods.

The HRV methods, used in this study are the time domain measures and the frequency domain measures, which will be described in the following chapters.

2.5.3 Time Domain Methods

In time domain methods, either the intervals between successive QRS complexes, resulting from sinus node depolarisations, or the heart rate at any point of time are determined. The term used for intervals between successive QRS complexes is the so-called R-R intervals (interval between two R signals of the ECG) also called normal-to-normal (NN) intervals.

These time domain variables can be derived either from **direct measurements** of the R-R intervals or instantaneous heart rate on the one hand (e.g. mean heart rate, the mean R-R interval), or from the **differences between R-R intervals** on the other hand (e.g. difference between the longest and the shortest R-R interval, difference between night and day heart rate).

Selected time domain measures, derived from a series of instantaneous heart rates or cycle intervals can be calculated from long term, traditionally 24 hours, or short term 2 to 5 minute recordings, will be shown in Fig. 10. (Malik et al 1996).

Variable	Units	Description
Statistical Measures		
RR	ms	Mean of duration of total number of R-R intervals
NN	ms	Normal to Normal intervals, duration between consecutive R-intervals NN is another expression for R-R intervals
SDNN	ms	Standard deviation of all NN intervals
SDANN	ms	Standard deviation of the averages of NN intervals in all 5-minute segments of the entire recording
SD1	ms	standard deviation of the instantaneous beat-to-beat variability (RR-intervals) of data
SD2	ms	standard deviation of the continuous long-term (R-R intervals) variability
SDNN index	ms	Mean of the standard deviations of all NN intervals for all 5-minute segments of the entire recording
SDSD	ms	Standard deviation of differences between adjacent NN intervals
pNN50	%	NN50 count divided by the total number of all NN intervals
RMSSD	ms	The square root of the mean of the sum of the squares of differences between adjacent NN intervals

Fig. 10 Selected time domain heart rate variability indices (Malik et al. 1996 p.6, modified after Böhmüller 2007)

SD1 (standard deviation of orthogonal distances of RR_i/RR_{i+1} points to the oblique diameter of the confidence ellipse) is calculated by orthogonal regression analysis. It is an expression of the standard deviation of the instantaneous beat-to-beat variability of data. It characterises short-term variability (Hottenrott 2001)

SD1 was chosen for the analysis in this study to show the short-term variability of instantaneous R-R intervals, representing the regulatory capacity of the athlete.

SDNN reflects all the cyclic components responsible for variability in the period of recording, as variance is mathematically equal to total power of spectral analysis. This means that SDNN is an estimate of overall variability of the heart rate (HRV).

Because of its dependence on the length of recording period it is inappropriate to compare SDNN measures obtained through recordings of different durations. SDNN estimates shorter, as the period of monitoring decreases. This is why durations of recordings, determining SDNN values, should be standardized (Malik et al. 1996). As the length of HRV recordings in this study differ between 4 and about 7 minutes, calculations of SDNN were not carried out.

pNN50 and **RMSSD** measurements estimate high-frequency variations in heart rate and thus are highly correlated. They both reflect parasympathetic activity. Because of better statistical properties, the RMSSD method is preferred to pNN50 (Malik et al. 1996). For that reason RMSSD was chosen for the analysis of time domain measures.

There are some more variables that could be measured in time domain, (e.g. the pulse-respiratory-quotient) but have no relevance for this study.

2.5.4 Frequency Domain Methods

To discriminate and quantify sympathetic and parasympathetic activity and total autonomic nervous system activity, HRV data have to be subject to mathematical transformation. The HRV signal is transformed into its constituent **frequency components** and the relative power of these components is usually quantified in absolute values of power (milliseconds squared). This method is called Power spectral analysis (McCraty et al. 2001). Many studies have relied on the Fast Fourier Transform. The method used in this study is the autoregressive auto regulation, as it is supported by Polar Precision Performance software 4.0 (Polar Electro Oy, Finland).

Frequency measures are divided into three main frequency ranges: Very low frequency (VLF) from 0,003-0,4Hz, low frequency (LF) from 0,04-0,15 Hz, and high frequency, ranging from 0,15 to 0,4Hz which are shown in Fig. 11.

Variable	Units	Description	Frequency Range
Analysis of Short-term Recordings (5 min)			
5-min total power	ms ²	The variance of NN intervals over the temporal segment	0.003- 0.4 Hz
VLF	ms ² %	Power in VLF range VLF percentage on total power	0.003- 0.04 Hz
LF	ms ² %	Power in LF range LF percentage on total power	0.04-0.15 Hz
HF	ms ² %	Power in HF range HF percentage on total power	0.15-0.4 Hz
LF/HF	%	Ratio LF [ms ²]/HF[ms ²]	

Fig. 11 Selected frequency domain heart rate variability indices (Mallik et al 1996, modified after Böhmüller 2007)

Frequency measures can also be illustrated in a diagram, as shown in Fig. 12.

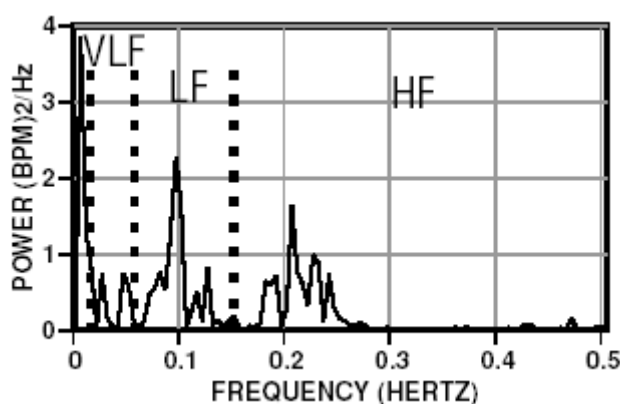


Fig. 12 (McCraty et al 2001, p.14)

The height of the peaks Fig. 12 points out the power of the HRV spectrum and reflects the activity in the different branches of the VNS.

In the following, detailed description of VLF, LF and HF:

The very low frequency (VLF) is an index of sympathetic activity, representing slower changes in heart rate (HR) (McCraty et al 2001). To interpret VLF from short-term recordings shorter than 5min., should be avoided, as it is a nonharmonic component, which does not have

coherent properties (Mallik et al 1996, Malliani 1999). But VLF was found to be a powerful predictor of a poor prognosis in patients with coronary heart disease (Pluim et al 1999).

The high frequency power (HF) is a primarily parasympathetic index (Malik et al 1996, Martinmaki et al 2005), representing quicker changes in heart rate (McCraty et al 2001) and corresponds to respiratory acts (Malliani 1999, Karemaker 1999).

The low frequency power (LF) is often interpreted as a marker of sympathetic activity. It was found out, that a large influence of the parasympathetic activity is also present in the low frequency component (Houle and Billman, 1999).

It reflects the blood pressure feedback signals sent from the heart back to the brain, which also has an effect on the HRV waveform (McCraty et al 2001, Malliani 1999).

“DeBoer et al. (1987) and Cooke et al. (1999) are inclined to put the origin of LF oscillations in some pacemaker, possibly in the CNS, rather than in the baroreflex.” (Cited after Karemaker 1999 p.316)

To investigate HF components of HRV, recordings of approximately 1 minute is needed, while approximately 2 minutes are needed to assess the LF component (Malik et al. 1996).

To investigate sympathetic power in this study, the percentage of VLF+LF on total power of the frequency spectrum was added (\sum VLF+LF). VLF and LF were not interpreted separately because of incoherent properties of VLF in less than 5 min. short term recordings. (See VLF above)

Interpretation of the absolute value of only one frequency spectrum reveals limited information, as it does not say anything about the percentage on overall power of the frequency spectrum (Ferscha et al. 1998).

Absolute values of HRV measures with different length of recordings may not be compared (Task Force 1996).

The ratio of low frequency to high frequency component (LF/HF) is a marker of the sympathovagal balance modulating sinus node pacemaker activity (Malliani 1999).

2.5.5 Significance of HRV Measures

The effects of internal and external changes on the vegetative nervous system (VNS) can be shown by HRV measures (Cerruti et al.1995). Changes include e.g. physical exercise, quality of sleep, and consumption of drugs.

An example for the changes in HRV values in time and frequency domain after new positioning of the body is demonstrated in Fig. 13.

Fig. 13 shows the change in R-R interval series (tachograms on the top) and the power spectra of high frequency (HF) at 0.33 Hz (corresponding to respiratory activity), low frequency (LF) at 0.09 Hz (usually corresponding to vasomotor waves), and very low frequency (VLF) around 0 Hz (on the bottom) of a 25-year-old healthy male subject in supine and upright (passive 90° tilt) positions (Malliani 1999).

“In the active upright position (or during passive tilt), in addition to an increase in heart rate and to small adjustments in blood pressure, a marked change occurs, as a rule, in the spectral profile; the LF component is increased, whereas the HF component is reduced. Variance usually decreases in the upright position, causing a reduction in the absolute value of both spectral components. Hence, in the upright position, LF tends to be decreased, in its absolute values, by the reduction of variance but also tends to be increased, in nu [normalized units], by the greater concentration of power in this part of the spectrum” (Malliani 1999)

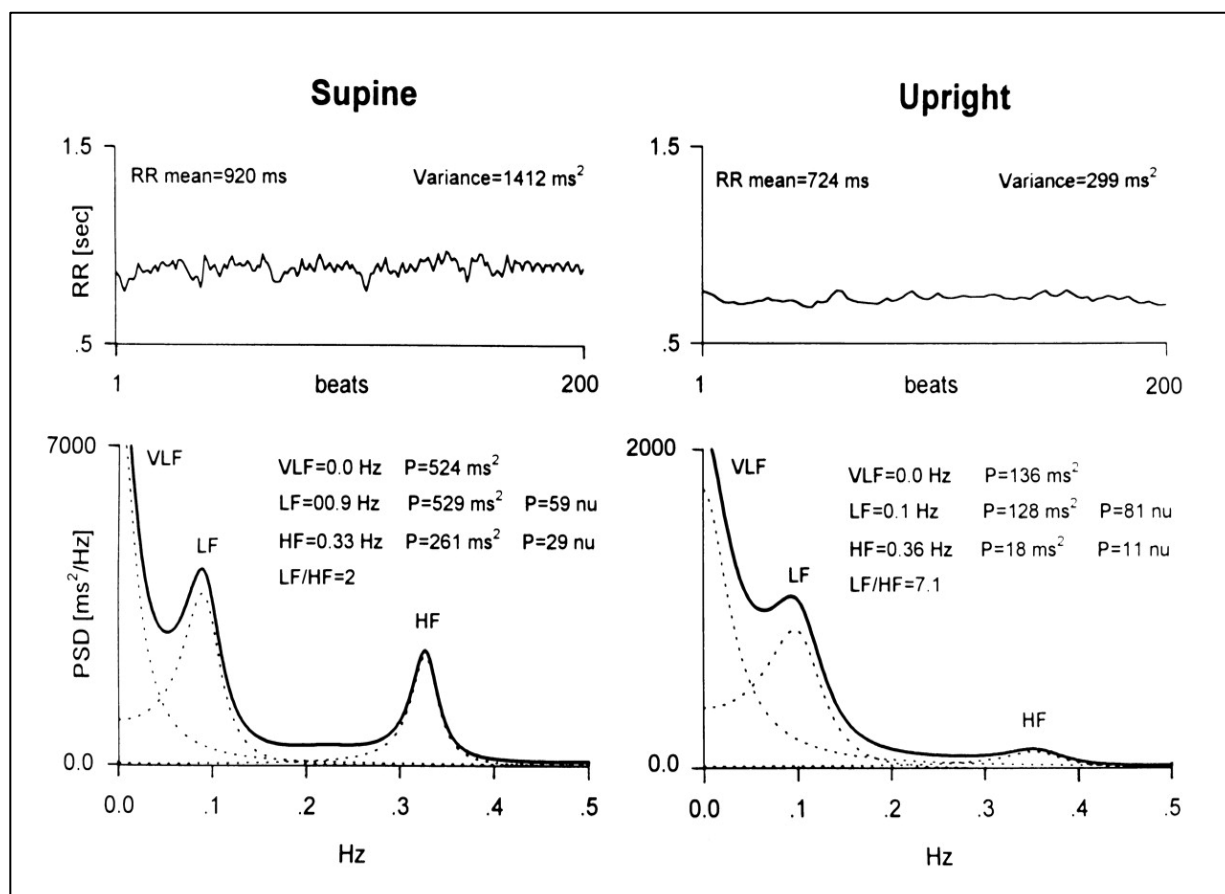


Fig. 13 (Malliani 1997 et al, Results)

Similar results can be found in Task force (1996).

Considering the previous chapters, it is evident that HRV measures can show **changes within the VNS**, the heart or the blood vessels (Mück-Weymann and Beise 2005).

HRV measures could also provide an opportunity to show the **regulatory capacity** of the body which is confirmed by the following statement.

“Systems-oriented models propose that HRV is an important indicator of both physiological resiliency and behavioural flexibility, reflecting the individual’s capacity to adapt effectively to stress and environmental demands.” (McCraty et al. 2001 p.1)

The **body ability to recover** can be tracked with parasympathically controlled parameters of HRV (e.g. HF power, RMSSD, pNN50) as an increase of parasympathic activation supports regeneration and recovery, an essential precondition for health. Recovery shows in a

reduction in sympathetic power (VLF and LF) and a simultaneous increase in high frequency parts, illustrating parasympathetic power (Tucek 2006).

McCraty et al. (2001) suggested that the **decrease** of the activity **in both branches** of the VNS can be interpreted as **autonomic exhaustion**. They found significantly lower sympathetic and parasympathetic activity in chronic fatigue patients, compared to healthy age and gender-matched controls.

In the following, the significance of HRV measures on high-performance athletes is discussed in more detail.

2.5.6 HRV and Performance

The impact of heavy training of high-performance athletes on the measuring results of HRV is a sensitive issue.

To gain an insight into the correlation of HRV measures and physical performance, here are short quotations of the conclusions of various studies:

7 male high-performance athletes were investigated about 3 months apart on 3 subsequent occasions by Iellamo et al. (2002).

From baseline up to 75% training load Iellamo et al. (2002) found “... *a progressive increase in the HF component and a decrease in the LF component of RR interval variability and in the LF/HF ratio.*” [...] “**At 100% training load, opposite changes were observed, with a relative increase in HR [heart rate] (...) accompanied by a marked and significant *decrease in the HF and an increase in the LF component of RR interval variability and in the LF/HF ratio.***”

Buchheit and Gindre (2006) investigated the relative association of cardio respiratory fitness and training load to different parameters of cardiovascular parasympathetic modulation and found out, “...*that vagal-related HRV indexes are significantly associated with VO₂max. Whatever the training loads, the fit subjects (FitLT and FitMT) [fit lowly-trained and fit-moderately trained] had the highest HRV indexes. This positive relationship between HRV and fitness is in agreement with a large number of previous studies.*” (Buchheit and Gindre 2006, p.12)

That vagal modulation can be a determinant of physical fitness, was also confirmed by Goldsmith et al. (1997). They found a close and highly **significant relationship between VO2 max and time domain measures** of vagal modulation such as RMSSD and pNN50 **as well as** a highly significant relationship between **VO2 max and HF power** (Malik et al., 1996).

Furlan et al.(1993) suggest, “... *that the **enhanced athletic performance** resulting from long term training may depend on an **increase of both vagal and sympathetic modulation.**” (Furlan et al. p.487)*

Du et al (2005) found a correlation between high levels of HRV and improved recovery. They investigated a group of 6 female marathon runners aged 32-40 years and a control group of 8 untrained, age-matched females and **found high levels of HRV** associated with rapid **heart rate recovery** after exercise.

Moser (2004) also brings up the close connection between high levels of HRV and the bodies capability to recover. He states, that the body recovers particularly fast, if all rhythms in the body are coordinated, which shows in a saving in power.

Garet et al. (2004) investigated the individual interdependence between VNS activity and performance in swimmers and found that variations in **VNS activity and variations in performances have correlated at an individual level.**

Due to the study of Goldsmith et al. (1997) “Physical fitness as a determinant of vagal modulation”, König et al. (2003) attested that **physical fitness can be predicted with HRV measures.**

The above-mentioned examples (see also 2.5.5 Significance of HRV measures) and those in the following three chapters point out that interpretations of HRV measures are not only heavily dependent on the training load (intensity and duration of the training), but also depend upon various circumstances, e.g. age, gender, physical health, physical fitness, emotional stress, quality of sleep, intake of drugs... (See 2.5.12. Limitations)

2.5.7 Influence of Age and Gender

HRV measures vary with age and gender in healthy individuals.

The ability for auto regulation decreases with increasing age and this is often noticed in a lowering of overall HRV (Malliani 1999). At the close of one's life, HRV is down to a minimum (Mück-Weymann and Beise 2005).

McCraty et al. (2001) investigated 260 healthy subjects from 10 to 99 years old. They found all investigated time domain measures decrease with aging. SDNN and SDANN decreased linearly across the entire lifespan, whereas pNN50 and rMSSD stabilised after age 59. Therefore SDNN could be a useful physiologic marker for aging.

By elderly men, Stein et al. (1997) found a reduction of global HRV and a loss of circadian variability. By elderly women, there was found a decline in short term indices of HRV, but no significant changes in circadian variability.

By men, HRV levels are significantly higher in time domain indexes of HRV than by age matched females, except those that reflect vagal modulation of heart rate (Stein et al. 1997). After the age of 50, gender related differences disappear (Stein et al. 1997, McCraty et al. 2001).

That there might be ethnic differences in HRV, African-Americans having greater HRV than European Americans was concluded by Wang et al. (2005).

There is evidence, that by women, regulation of the VNS is controlled more by the parasympathetic nervous system, whereas by men, to a greater extent by modulation of the sympathetic nervous system (König et al 2003).

2.5.8 Influence of Mental Stress

The influence of **mental stress** on HRV measures can be found in a decrease of total power of HRV (Moser et al. 2004, De Haas 2005), an increase in LF and a decrease in HF (Malliani et al. 1991).

How different **emotions** can affect autonomic nervous system function, which can be shown in HRV measures. During anger, there is a large increase in LF which represents the activity

of the sympathetic system, whereas during appreciation an increase in HF as an expression of an increased parasympathetic system can be seen.

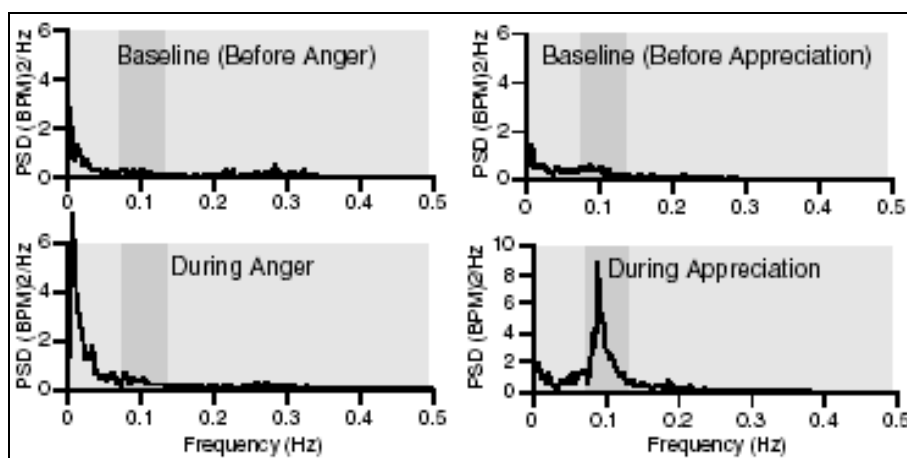


Fig. 14 (McCraty et al. 2001, p.18)

Fig. 14 shows increased LF power in the far left-hand region of the power spectrum with anger, and an increased parasympathetic activity, which helps protect the heart, around 0.1 Hz during appreciation (McCraty et al 2001).

Correlations between HRV measures and **biochemical changes** were also found out. Pluim et al. (1999) found high levels of HRV correlated with high myocardial energy phosphates (PCr/ATP), which contributes to the energy status of the heart.

2.5.9 Influence of Diseases

HRV is not only influenced by age and gender, but also by a number of **diseases**. The state of health is a decisive factor, influencing HRV measures, as Kleiger et al. (1991, p.626) confirm: *“Heart rate (HR) variability ... decreases with age and with certain disease states such as congestive heart failure, diabetic neuropathy, post myocardial infarction, and in some forms of inducible ventricular tachycardia or ventricular fibrillation”*.

Malliani et al. (1991) investigated the influence of high arterial blood pressure in frequency domain of HRV and found an increase of LF and a decrease of HF components compared to subjects with normal blood pressure under resting conditions.

The adaptability of the VNS is also reduced in patients with hypertension, which could be shown in a decrease of variability in circadian rhythm over 24 hours compared to normotensive test persons. (See Fig. 15.)

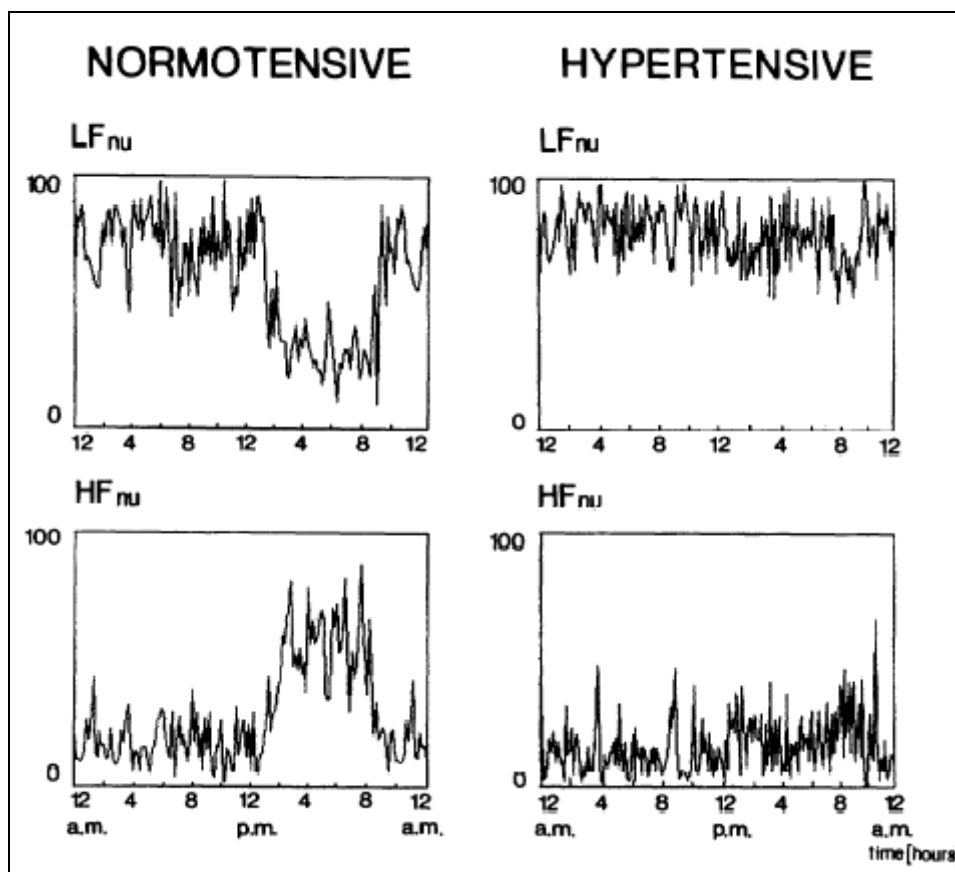


Fig. 15 (Malliani et a. 1991, p.487)

In Fig. 15 computer analysis plots of 24-hour RR interval variability a normotensive subject (110/70 mm Hg resting arterial pressure) is compared to a hypertensive subject (160/105 mm Hg resting arterial pressure). A clear circadian circle is present in both spectral components in the normotensive subject. The adaptability to circadian changes in the VNS is almost lost in the hypertensive subject.

The more hypertension increases, the more HRV is affected (Malliani et al. 1991)

2.5.10 Validity

In this study, HRV measures were taken with the polar S810 heart rate monitor. This measure device is scientifically proven, easily applied, non invasive, and at reasonable price.

The validity of the polar S810 heart rate monitor (HRM) was examined and compared with an electrocardiogram (ECG) (Physiotrace, Estaris, Lille, France) by Gamelin et al. (2006) during an orthostatic test. Time domain analysis, Pointcare plot analysis and fast Fourier transform

were performed. HRV parameters derived from HRM and ECG were not different and well correlated except root mean square of difference (RMSSD) and SD1 in standing position.

These results “...support the validity of the Polar S810 HRM to measure R-R intervals and make the subsequent HRV analysis in supine position.”(Gamelin et al. 2006, Conclusion). In standing position, caution must be taken for the parameters sensitive to the short-term variability (i.e., RMSSD and SD1).

A great degree of accuracy was also attested for the polar S810 heart rate monitor measures by Löllgen et al. (2003), for time domain as well as for frequency domain measures.

2.5.11 Reliability

The main quality criteria for scientific reasoning: reliability, validity and objectivity, were attested for the polar S810 heart rate monitor by Löllgen et al. (2003).

Kleiger et al. (1991) investigated the stability of HRV over time (3 to 65 days). They found variables strongly dependent on vagal tone as HF (high-frequency), LF (low-frequency), total power, RMSSD (root-mean-square successive difference), and pNN50 (percentage of differences between adjacent normal cycle intervals greater than 50 ms computed over the entire 24-hour electrocardiograph recording) were highly correlated, and concluded that measures of heart rate variability are stable over short periods of time.

2.5.12 Limitations

HRV can be influenced by a number of variables:

- Age and gender (see 2.5.7. Influence of Age and Gender)
- Level of physical fitness (see 2.5.6. HRV and Performance)
- The position, in which HRV measures are investigated (see 2.5.5., Significance of HRV measures)
- Mental stress (see 2.5.8)
- Diseases (see 2.5.9)
- Daytime of HRV measuring: ergo tropic (sympathetic) and trophotropic (parasympathetic) power changes over a period of 24h (see 2.5.9. Influence of diseases and 2.2 Vegetative nervous System). Therefore, HRV should be measured at the same time of the day.

- Respiratory rate (see 2.3., The Influence of Respiration)
- Noise
- Air temperature
- atmospheric pressure as during measures in higher altitudes or during diving
- Drugs as alcohol and nicotine but also neurotoxic substances and medications e.g. antiarrhythmica, β -adrenoblock and M-cholinoblock, Narkotica, Tranquilicer, Nifedipin, Diltiazem, Amiodaron, Amitriptilin, Fluoxetin, Clozapin, Propofol, ... (Arbeitsgemeinschaft der Wissenschaftlichen Medizinischen Fachgesellschaften 2006).

Heart rate can also be changed by circulatory substances. During stress, catecholamines, like adrenaline, are released, which cause an increase in heart rate (Kestin 1993).

2.6 Osteopathic Approach

Besides re-establishing structural and functional balance the objective of my Osteopathic treatments was to support all body systems to optimise their functioning in sense of a homodynamic balance to achieve an ideal performance state in the athlete.

These body systems include neurological, mechanical, membranous, electrophysiological, biochemical and fluid levels. They lay the foundations for auto regulation of the entire body functions. Optimising the functioning of all parts of the body and within these systems might help performing athletes to achieve their personal best. This could be a main precondition for a high-performance athlete to achieve peak performance.

Hoover (1956) assumed, that all parts of the body are so related that they adjust their functions to achieve **homeostatic balance** permitting the body to function optimally as a unit. However, the amount of energy the body needs to achieve and sustain homeostasis or homodynamic, (see Auto regulation, 1.1.1), depends on each individual body (Liem 2002, Wühr 2007).

Adaptations and compensations as well as diseases and injuries compromise the **regulatory circuitry** the body employs to maintain a high level of functioning for obtaining optimal

performance in any endeavour. Adaptations and compensations are the body's possibilities to sustain homeostatic balance, if impacts on the body can't be repaired to the full (Wühr 2007). That the body needs more energy to sustain adaptations and compensations, than under optimal conditions is a concept also supported by Wühr (2007) and Liem (2001). If distorted body functions could be resolved or alleviated with osteopathic treatments, it would eradicate or at least diminish the body's adaptations or compensations. This would result in a more economical functioning of the body needing less energy. By athletes, the husbanded / saved energy the body does not need for adaptations and compensations might be used to enhance body performance.

That structure and function are interdependent is one of the Osteopathic principles. A.T. Still's quotation: "*When perfect harmony is not found in form and function, lack of speed in motion exists*" could also be applied on athletes' speed in a figurative sense. The model of homeo-dynamics, which describes a well-balanced dynamics of all body systems, implies a harmonious interplay between form and function. A lack of form and function would decrease the body's homodynamics. Increased homeo-dynamics, permitting a continuous adjustment to inner and outer influences, would increase global body regulations. As a result the body's possibilities to achieve peak performance could be enhanced. (Also J.P.Barall promoted this view in a one-to-one interview on 16.2.07 in Gars/Kamp)

Osteopathic treatments are also founded on the principle that the body has the capacity to heal itself. Therefore a sensitive and highly reactive VNS could be an important prerequisite data for the body to self restore and to process therapeutic stimuli supporting the organism's self healing powers (see 2.2 The Vegetative Nervous System). It also fine tunes the body's musculoskeletal system, "*the primary machinery of life*", to meet the ever changing demands (Frymann et al. 1992). Therefore the treatments also focused in supporting the VNS.

Before every treatment, an accurate Osteopathic anamnesis was made. This is a precondition for a safe and effective treatment. According to the above mentioned findings and general principles of osteopathy, adequate structural, visceral and cranio-sacral techniques were chosen for the athlete to adapt the treatment to the patient.

As this study deals with a high-performance athlete and athletes often suffer from injuries I would like to mention the perception of James Oshman (2003), who promotes the view that

one of the most profound goals of all therapies is to resolve internalised memories e.g. traumatic experiences in a way that leaves the organism free to continue on its path toward its own perfection.

3 Methods

3.1 Study Design

A single subject design was used to investigate the question, if the performance of athletes can be enhanced with Osteopathic treatments.

A young (23 years of age) male, physically and mentally healthy high-performance tri-athlete was chosen for this study. The social environment was considered as stable. The athlete was informed about the procedure, participated voluntarily and did not receive any compensation. Circumstances which can influence the measurement results are known to the athlete.

A pilot study was done over a period of about five months (31.5.06 – 26.10.06) to investigate changes in HRV in the morning before and the day after 7 Osteopathic treatments. As HRV measures changed in part significantly, a long term study was planned over a period of 10 weeks to show the behaviour of measures on a longer passage of time.

In February 2007 we started a single subject design study over a period of 10 weeks. Four Osteopathic treatments with intervals of at least 2 weeks in between, were carried out. HRV short-term recordings, 4 to 7 minutes, were made by the athlete every morning immediately after waking up. For measurements, he was lying in supine position without speaking nor moving to ensure a physiologically comparable period of recording.

4 Osteopathic treatments with duration of 45min. were carried out between 11 a.m. and 1 p.m. in a private practice.

An Osteopathic anamnesis was done before every treatment to adjust the therapy procedure to the current situation of the athlete. According to findings, structural, cranial and visceral osteopathic techniques were applied. (See Osteopathic Approach 2.6)

The treatment took place always in the same quiet room with comfortable temperature, about 22°C.

To define adequate conditions for the Osteopathic treatment the appointments were timely spread in the middle of moderate training sequences lasting for about 10 days. It was made sure that the period of study was free of competitions.

HRV recordings in time and frequency domain were analysed 7 days before and 7 days after the treatment to show the behaviour of measures.

The athlete recorded daily, in addition to the HRV recordings, the feeling during training on a scale from 1 (very good feeling) to 5 (very bad feeling), the intensity of training on a scale from 1 (regenerative training) to 5 (competition) and the duration of the training sequences in minutes.

The 1 to 5 scale was chosen, instead of the usual 1 to 10, because the athlete had already been used to recording his feelings and training intensity over the past four years.

3.2 Inclusion criteria

A high-performance athlete, physically and mentally healthy was selected.

Nutritional supplements were allowed, as no changes in HRV were expected due to the intake.

3.3 Exclusion criteria

There was not any other treatment taking place within 7 days before and 7 days after the Osteopathic treatment.

The investigated subject is non smoking and did not take any drugs, intervening the VNS or heart rate (see 2.5.12: Limitations and 2.4.1: Regulation of the Heart Rate)

The intake of alcohol was prohibited the day before, the day of treatment and the day after the treatment but could not be restricted completely during the period of investigation.

3.4 HRV Measurements

After the ringing of the alarm clock in the morning, the electrodes of the transmitter belt (Polar T61 coded™ transmitter, Finland) were carefully wetted before the transmitter was sufficiently tightened to the chest to ensure good skin contact.

A heart rate monitor (S810i, Polar, Finland) was started to continuously monitor HRV for a period of 5 min., the athlete still lying in bed in supine position without speaking or making any movements.

Taking into account the results of Blasi et al. (2003), analysis of HRV records started one minute after the beginning of recording.

(Blasi et al. (2003) investigated 16 normal humans during acoustically induced arousals from sleep and found a rapid surge in overall magnitude of the RR interval spectrum, particularly in LF power, and HF lower than baseline in the post arousal period. The increase of LF power remained up to 40 seconds after the stimulus.)

3.5 HRV Analysis

The HRV recordings of the heart rate monitor (S810i,Polar, Finland) were statistically analysed by using Polar Precision Performance software 4.0 (Polar Electro Oy, Finland). The monitored tachogramms were visually checked for artefacts.

Occasional ectopic beats were identified and replaced with interpolated R-R intervals.

From the recorded R-R intervals, i.e. the time between the R peaks of consecutive QRS complexes, time domain parameters are calculated. (See 2.5.3 Time Domain Methods)

Frequency domain analyses are performed on the bases of autoregressive autocorrelation by the above software (Mahlke et al. 2001).

The **time domain analysis** includes common descriptive statistics. Therewith, the variability and standard deviation of consecutive R-R intervals are quantified.

In this study, two representative time domain parameters, RMSSD and SD1 have been chosen for HRV analysis.

RMSSD has proved to be a meaningful parameter for short-term measures as an indication for vagal influence on HRV.

SD1 is an expression of the standard deviation of the instantaneous beat-to-beat variability of data. It characterises short-term variability (Hottenrott 2001). (See 2.5.3: Time Domain Methods)

Following classification was used in this study:

-1	≤ r ≤	-0,7	...	very strong correlation
-0,7	≤ r ≤	-0,5	...	strong correlation
-0,5	≤ r ≤	0,5	...	moderate correlation
0,5	≤ r ≤	0,7	...	weak correlation
0,7	≤ r ≤	1	...	very weak correlation

Positive correlation was set at $r < -0,5$.

Besides HRV measures, the athlete recorded his **personal feelings** during training on a scale from 1 (very good feeling) to 5 (very bad feeling) every day.

Training intensity was recorded on a scale from 1 (regenerative training) to 5 (competition).

Duration of daily training was recorded in minutes every day.

Five recordings before and three recordings after the treatment out of 60 recorded days are missing.

The reason for the missing data was that the athlete either did not sleep well or overslept. He was therefore missing sleep or under stress. This case would obscure the measurements because of the stress potential. Another reason was that he did not push the start button on the measuring device correctly, so no measures were taken, or that he simply forgot to measure.

4 Results

Results are presented in two stages, first the pilot study, subsequently the central study. Changes in HRV measures are illustrated in bar diagrams **the day before and the day after the treatment** in the pilot study as well as in the central study.

The behaviour of the **longer-term measures** (4 periods) is shown in a graph below.

Mean values were calculated for the time before and the time after treatment for each parameter.

Pearson's correlation coefficient was calculated for time domain parameters (SD1 and RMSSD), frequency parameters (HF and \sum VLF + LF) and for all parameters together including the personal feeling during training periods in the central study.

In the following, changes in time domain and frequency domain parameters are compared one to one to underline differences before and after the Osteopathic treatment.

4.1 Results Pilot study

An overview of all measures before and after seven treatments is presented in Fig. 16.

The following figures display changes before and after the treatment for each parameter calculated in time domain (SD1 and RMSSD) and frequency domain analysis (HF and \sum VLF+LF).

2006	31.5.	1.6.	20.6.	21.6.	28.6.	29.6.	30.8.	31.8.
SD1	86,30	90,00	68,60	79,20	73,40	119,40	10,60	50,80
RMSSD	121,70	126,50	96,00	111,40	104,70	163,40	15,00	72,00
HF	20 %	10 %	19 %	29 %	37 %	16 %	8 %	18 %
\sum VLF+LF	80 %	90 %	81 %	71 %	63 %	84 %	92 %	82 %

2006	13.9.	14.9.	9.10.	10.10.	25.10.	26.10.
SD1	26,00	59,10	44,70	80,20	44,90	79,00
RMSSD	36,70	83,40	63,10	113,10	63,50	111,50
HF	27 %	20 %	10 %	13 %	25 %	37 %
\sum VLF+HF	73 %	80 %	90 %	87 %	75 %	63 %

Fig. 16 Measures of time domain and frequency domain indices before and after 7 Osteopathic treatments

Results in time domain measures

In time domain SD1 and RMSSD all measures increased the day after all seven treatments. SD1 increased from 4, 29% after the first treatment and over 379% after the fourth treatment (see Fig. 17).

RMSSD increased 3, 94% after the first and 380% after the fourth treatment (See Fig. 19).

Mean increase of SD1 was 106,33% and 105,45% for RMSSD the day after 7 treatments.

The increase of SD1 and RMSSD after the operation is considerably larger (75, 95% to 379, 25%) than before the operation (4, 29% to 62, 67%).

The two treatments after the operation showed with 379% and 127% the largest increase.

date	before	after	Diff. [%]
31.5./1.6.	86,3	90	4,29%
20.6./21.6.	68,6	79,2	15,45%
28.6./29.6.	73,4	119,4	62,67%
30.8./31.8.	10,6	50,8	379,25%
13.9./14.9.	26	59,1	127,31%
9.10./10.10.	44,7	80,2	79,42%
25.10./26.10.	44,9	79	75,95%

Fig. 17 Measures in SD1 [ms] before and after treatment

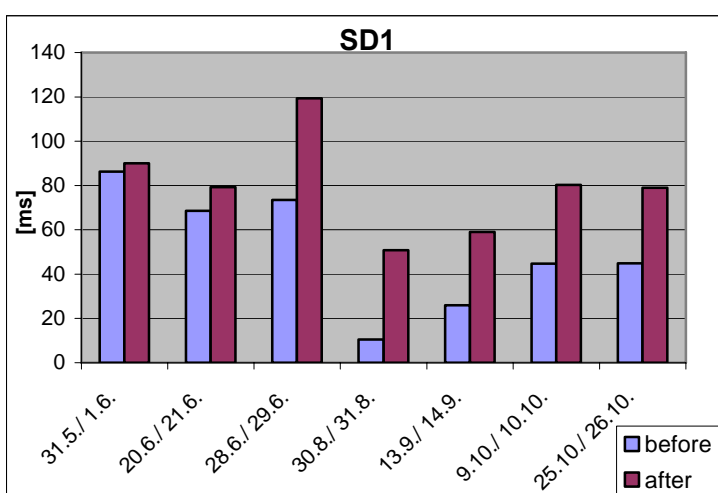


Fig. 18 Measures in SD1 [ms] before and after treatment

date	before	after	Diff. [%]
31.5./1.6.	121,7	126,5	3,94%
20.6./21.6.	96	111,4	16,04%
28.6./29.6.	104,7	163,4	56,06%
30.8./31.8.	15	72,0	380,00%
13.9./14.9.	36,7	83,4	127,25%
9.10./10.10.	63,1	113,1	79,24%
25.10./26.10.	63,5	111,5	75,59%

Fig. 19 Measures in RMSSD [ms] before and after treatment

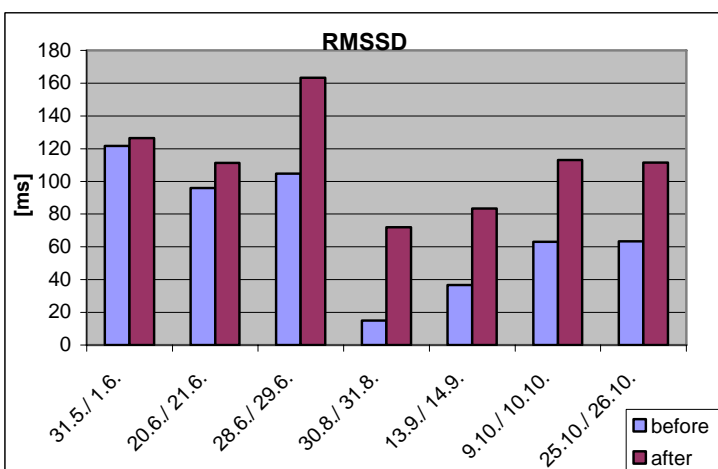


Fig. 20 Measures in RMSSD [ms] before and after treatment

The impact on HRV, left by an ENT operation the athlete had to undergo on 6.August 2006, can clearly be observed.

The rapid decrease in time domain measures (see 6, Discussion) can clearly be observed. Measures made on 30.8. and 31.8. clearly decreased compared to the measures before. This fact can be seen more clearly in Fig. 18 and Fig. 20. where measures after the operation remained lower than measures before the operation (blue bars indicate the measures before the treatment).

Results in frequency domain measures

Frequency domain measures did not show the same results as time domain measures, whereas all measures showed an increase the day after the treatment.

In frequency domain, measures before and after the treatment showed an irregular behaviour.

In frequency domain, measures in the morning before and measures in the morning after the treatment vary in value from -50% to +125% in HF and from -16% to 33, 3% in \sum VLF + LF (see Fig. 21 and Fig. 23).

date	before	after	Diff. [%]
31.5./ 1.6.	20,00	10,00	-50,0%
20.6./ 21.6.	19,00	29,00	52,6%
28.6./ 29.6.	37,00	16,00	-56,8%
30.8./ 31.8.	8,00	18,00	125,0%
13.9./ 14.9.	27,00	20,00	-25,9%
9.10./ 10.10.	10,00	13,00	30,0%
25.10./ 26.10.	25,00	37,00	48,0%

Fig. 21 Measures in HF [%] before and after treatment

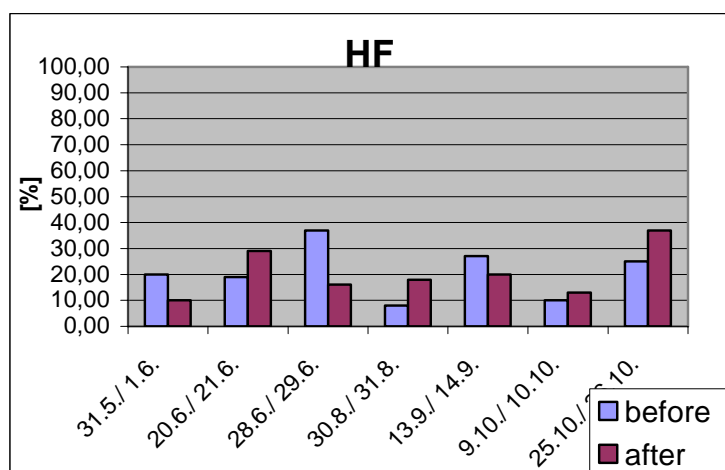


Fig. 22 Measures in HF [%] before and after treatment

The effects of the treatments on HF show an increase after the second, fourth, sixth and seventh treatments and a decrease after the first, third and fifth treatments. (Fig. 22)

For \sum VLF + LF, the opposite applies. (Fig. 24)

date	before	after	Diff. [%]
31.5./ 1.6.	80	90,000	12,5%
20.6./ 21.6.	81	71,000	-12,3%
28.6./ 29.6.	63	84,000	33,3%
30.8./ 31.8.	92	82,000	-10,9%
13.9./ 14.9.	73	80,000	9,6%
9.10./ 10.10.	90	87,000	-3,3%
25.10./ 26.10.	75	63,000	-16,0%

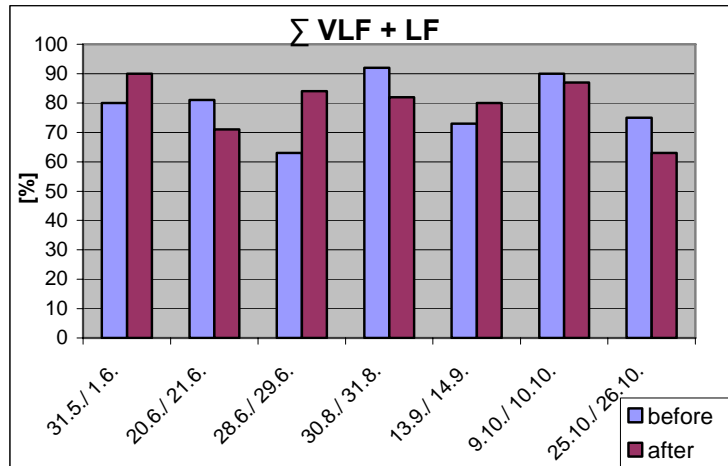


Fig. 23 Measures of Σ VLF + LF [%] before and after treatment

Fig. 24 Measures of Σ VLF + LF [%] before and after treatment

In frequency domain the decrease after the operation is also visible in HF on 30.8.06, reflecting the parasympathetic part and an increase of Σ VLF + LF, representing the sympathetic part of HRV measures.

4.2 Results Central study

An overview is given for the four periods measured, lasting altogether from 7.2.07 to 18.4.07. (See Fig. 25)

- Period 1: 7.2. - 21.2.07
- Period 2: 21.2. - 7.3.07
- Period 3: 9.3. - 23.3.07
- Period 4: 4.4. - 18.4.07

SD1 and RMSSD show measures in time domain, HF and Σ VLF + LF represent frequency domain indices.

Results

Each period shows measures from 7 days before treatment to 7 days after treatment.

SD1	7 days before	6 days before	5 days before	4 days before	3 days before	2 days before	1 day before	day of treatment	1 day after	2 days after	3 days after	4 days after	5 days after	6 days after	7 days after
	Period 1	58,0	66,0	71,0	55,0	44,0	74,0	60,0	61,0	74,0	85,0	77,0	85,0	85,0	85,0
Period 2		85,0	77,0	59,0	40,0	66,0	110,0	78,0	81,0		47,0	67,0	72,0	73,0	65,0
Period 3	73,0	65,0	74,0	85,0	91,0	49,0	70,0	68,0	69,0	73,0	69,0	88,0	67,0		45,0
Period 4	64,0			90,0	27,0	53,0	75,0	78,0	54,0	36,0		80,0	44,0		85,0

RMSSD	7 days before	6 days before	5 days before	4 days before	3 days before	2 days before	1 day before	day of treatment	1 day after	2 days after	3 days after	4 days after	5 days after	6 days after	7 days after
	Period 1	81,0	100,0	99,0	78,0	61,4	105,0	85,0	86,0	105,0	120,0	108,0	74,0	76,0	162,0
Period 2		162,0	108,0	83,6	55,4	94,0	155,0	110,0	114,0		65,4	95,0	101,0	104,0	90,0
Period 3	104,0	90,0	105,0	120,0	127,0	68,6	99,0	95,0	98,0	103,0	96,0	124,0	95,0		64,0
Period 4	52,0			128,0	38,6	74,0	106,0	110,0	77,0	50,2		113,0	62,4		120,0

HF	7 days before	6 days before	5 days before	4 days before	3 days before	2 days before	1 day before	day of treatment	1 day after	2 days after	3 days after	4 days after	5 days after	6 days after	7 days after
	Period 1	21,0	12,0	32,0	14,0	21,0	31,0	34,0	17,0	23,0	7,0	18,0	7,0	7,0	7,0
Period 2		7,0	18,0	37,0	36,0	23,0	27,0	25,0	19,0		21,0	18,0	24,0	23,0	21,0
Period 3	23,0	21,0	21,0	25,0	28,0	24,0	33,0	14,0	36,0	12,0	23,0	25,0	32,0		34,0
Period 4	14,0			41,0	22,0	32,0	30,0	31,0	33,0	20,0		39,0	29,0		40,0

ΣVLF+LF	7 days before	6 days before	5 days before	4 days before	3 days before	2 days before	1 day before	day of treatment	1 day after	2 days after	3 days after	4 days after	5 days after	6 days after	7 days after
	Period 1	79,0	88,0	68,0	86,0	79,0	69,0	66,0	83,0	77,0	93,0	82,0	93,0	93,0	93,0
Period 2		93,0	82,0	63,0	64,0	77,0	73,0	75,0	81,0		79,0	82,0	76,0	77,0	79,0
Period 3	77,0	79,0	79,0	75,0	72,0	76,0	67,0	86,0	64,0	88,0	77,0	75,0	68,0		66,0
Period 4	86,0			59,0	78,0	68,0	70,0	69,0	67,0	80,0		61,0	71,0		60,0

Fig. 25 HRV measures in time domain and frequency domain in four periods from 7.2.07 to 18.4.07

In the following, all parameters are described separately.

For every parameter a bar diagram shows changes between the morning measure before the treatment and the morning after the treatment as in the pilot study.

For every parameter the subjacent diagrams show the behaviour over the same 14 day period.

Results in time domain analysis

SD1 and RMSSD increased the day after the first three treatments (period 1 to 3) but decreased the day after the fourth treatment.

The distribution of SD1 and RMSSD over all four time periods shows an irregular behaviour.

Results

Measures in the 7 day periods after the treatment vary in a considerable extent, just the way they did before the treatment. (See Fig. 29 and Fig. 33)

SD1	7 days before	6 days before	5 days before	4 days before	3 days before	2 days before	1 day before	day of treatment	1 day after	2 days after	3 days after	4 days after	5 days after	6 days after	7 days after
period 1 (7.2. - 21.2.07)	58,0	66,0	71,0	55,0	44,0	74,0	60,0	61,0	74,0	85,0	77,0	85,0	85,0	85,0	
period 2 (21.2. - 7.3.07)		85,0	77,0	59,0	40,0	66,0	110,0	78,0	81,0		47,0	67,0	72,0	73,0	65,0
period 3 (9.3. - 23.3.07)	73,0	65,0	74,0	85,0	91,0	49,0	70,0	68,0	69,0	73,0	69,0	88,0	67,0		45,0
period 4 (4.4. - 18.4.07)	64,0			90,0	27,0	53,0	75,0	78,0	54,0	36,0		80,0	44,0		85,0
mean	65,00	72,00	74,00	72,25	50,50	60,50	78,75	71,25	69,50	64,67	64,33	80,00	67,00	79,00	65,00

Fig. 26 Distribution of SD1 [ms] in four periods from 7.2.07 to 18.4.07

SD1

Separately considering the SD1 only the day after the treatment, we notice that it increased after the first three treatments but decreased after the fourth treatment (-33%) after a period of a 10 day influenza. (Fig. 27 and Fig. 28)

Date	before	after	Diff. [%]
14.2.- 15.2.07	61,00	74,00	21,31%
28.2.- 1.3.07	78,00	81,00	3,85%
16.3.- 17.3.07	68,00	69,00	1,47%
11.4.- 12.4.07	78,00	54,00	-33,77%

Fig. 27 Measures in SD1 [ms] before and after treatment

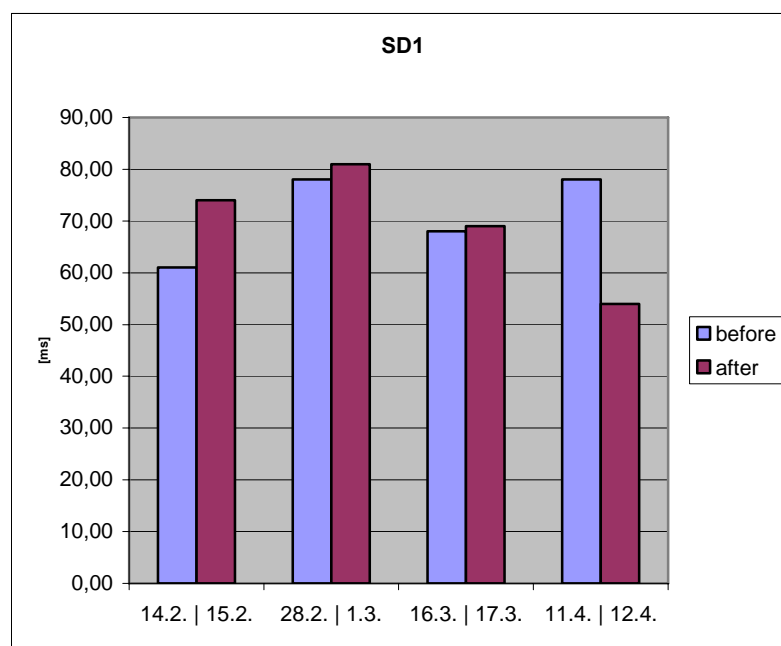


Fig. 28 Measures in SD1 [ms] before and after treatment

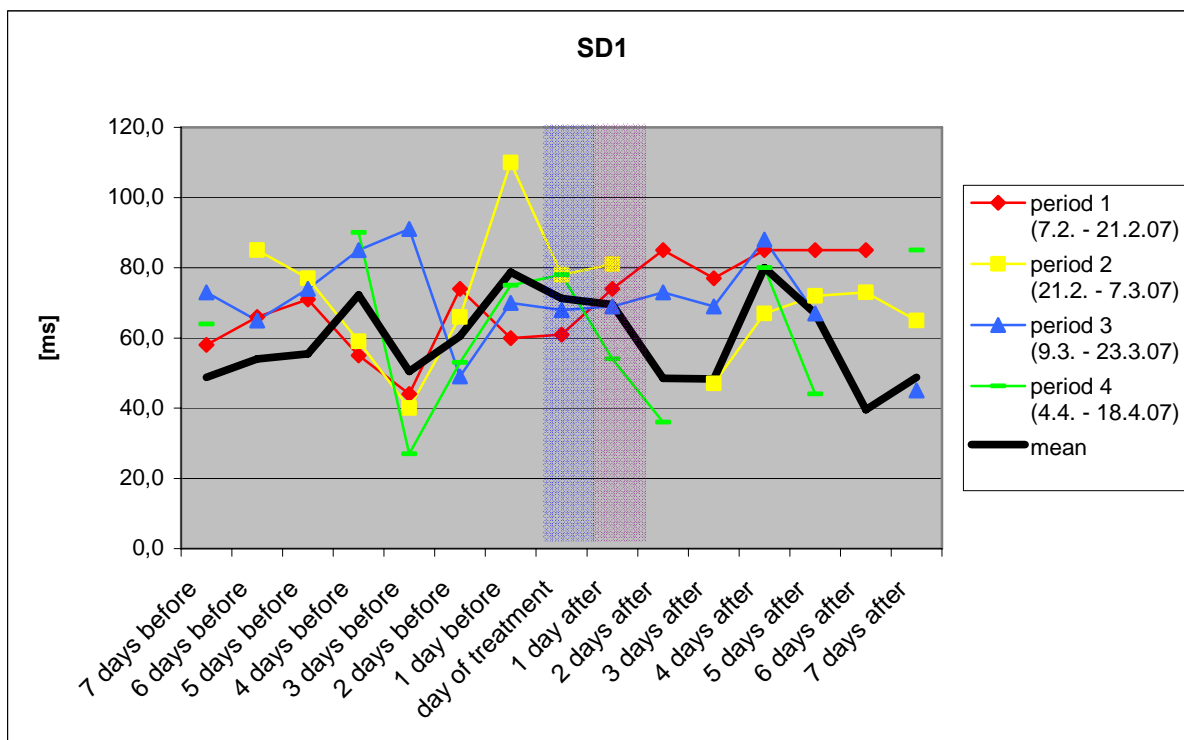


Fig. 29 Distribution of SD1 in four periods from 7.2.07 to 18.4.07

In the long run, only the first period of the 14 day measure shows an increase in SD1 after the treatment. (See red line in Fig. 29)

Same as for the measures of all four single periods the mean value of SD1 also shows an irregular behaviour before and after the treatment.

RMSSD

As with SD1, RMSSD increased the day after the first three treatments but decreased after the fourth treatment (-30%) after a period of a 10 day influenza.(Fig. 31 and Fig. 32)

RMSSD	7 days before	6 days before	5 days before	4 days before	3 days before	2 days before	1 day before	day of treatment	1 day after	2 days after	3 days after	4 days after	5 days after	6 days after	7 days after
period 1 (7.2. - 21.2.07)	81,0	100,0	99,0	78,0	61,4	105,0	85,0	86,0	105,0	120,0	108,0	74,0	76,0	162,0	
period 2 (21.2. - 7.3.07)		162,0	108,0	83,6	55,4	94,0	155,0	110,0	114,0		65,4	95,0	101,0	104,0	90,0
period 3 (9.3. - 23.3.07)	104,0	90,0	105,0	120,0	127,0	68,6	99,0	95,0	98,0	103,0	96,0	124,0	95,0		64,0
period 4 (4.4. - 18.4.07)	52,0			128,0	38,6	74,0	106,0	110,0	77,0	50,2		113,0	62,4		120,0
mean	79,0	117,3	104,0	102,4	70,6	85,4	111,3	100,3	98,5	91,1	89,8	101,5	83,6	133,0	91,3

Fig. 30 Distribution of RMSSD in four periods from 7.2.07 to 18.4.07

date	before	after	Diff. [%]
14.2.- 15.2.07	86,00	105,00	22,09%
28.2.- 1.3.07	110,00	114,00	3,64%
16.3.- 17.3.07	95,00	98,00	3,14%
11.4.- 12.4.07	110,00	77,00	-30,00%

Fig. 31 Measures in RMSSD [ms] before and after treatment

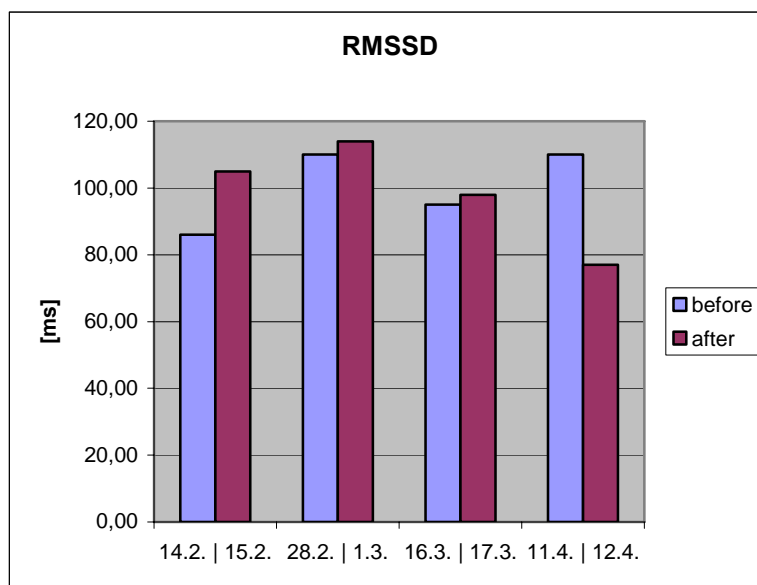


Fig. 32 Measures in RMSSD [ms] before and after treatment

The long run measures of all four periods show an irregular behaviour before and after the treatment. (See Fig. 33)

Same as for the measures of all four single periods the mean value of RMSSD also shows an irregular behaviour before and after the treatment.

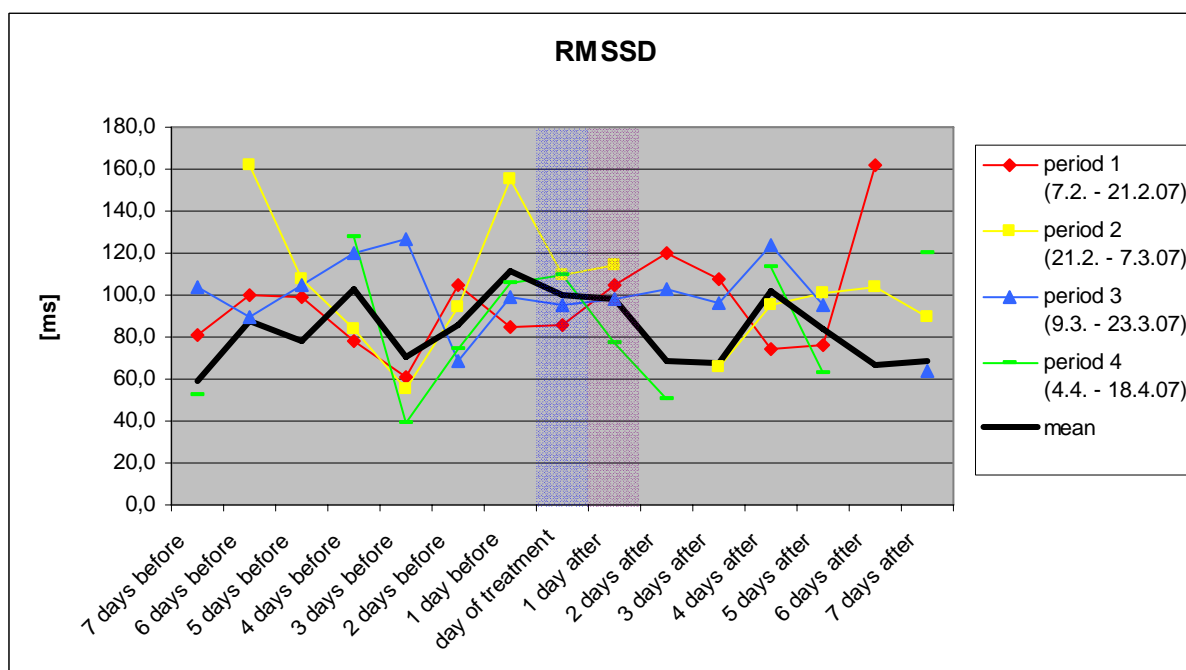


Fig. 33 Distribution of RMSSD in four periods from 7.2.07 to 18.4.

Pearson’s correlation for time domain parameters (SD1 and RMSSD) comparing measures before and after the treatment was very weak with ($r = 0,996$) which indicates hardly any difference between measures before and after the treatment.

Results in frequency domain analysis

In the 4 periods of 14 days each which were measured, a great variety on day to day changes could be observed.

The distribution of HF and \sum VLF+LF can be tracked in Fig. 34 and

Fig. 38)

HF	7 days before	6 days before	5 days before	4 days before	3 days before	2 days before	1 day before	day of treatment	1 day after	2 days after	3 days after	4 days after	5 days after	6 days after	7 days after
period 1 (7.2. - 21.2.07)	21,0	12,0	32,0	14,0	21,0	31,0	34,0	17,0	23,0	7,0	18,0	7,0	7,0	7,0	
period 2 (21.2. - 7.3.07)		7,0	18,0	37,0	36,0	23,0	27,0	25,0	19,0		21,0	18,0	24,0	23,0	21,0
period 3 (9.3. - 23.3.07)	23,0	21,0	21,0	25,0	28,0	24,0	33,0	14,0	36,0	12,0	23,0	25,0	32,0		34,0
period 4 (4.4. - 18.4.07)	14,0			41,0	22,0	32,0	30,0	31,0	33,0	20,0		39,0	29,0		40,0
mean	19,33	13,33	23,67	29,25	26,75	27,50	31,00	21,75	27,75	13,00	20,67	22,25	23,00	15,00	31,67

Fig. 34 Distribution of HF in four periods from 7.2.07 to 18.4.07

As in the pilot study, measures of frequency domain (both HF and \sum VLF+LF) did not show coherent increases on the following morning after the treatment. (See Fig. 36 and Fig. 41) After the first, third and fourth treatment the measures of HF increased, but decreased after the second treatment. (Fig 35 and Fig.36).

For \sum VLF + LF, the opposite applies. (See Fig. 39 and Fig. 40)

Results

date	Before	after	Diff. [%]
14.2.- 15.2.07	17,00	23,00	35,29%
28.2.- 1.3.07	25,00	19,00	-24,00%
16.3.- 17.3.07	14,00	36,00	157,14%
11.4.- 12.4.07	31,00	33,00	6,45%

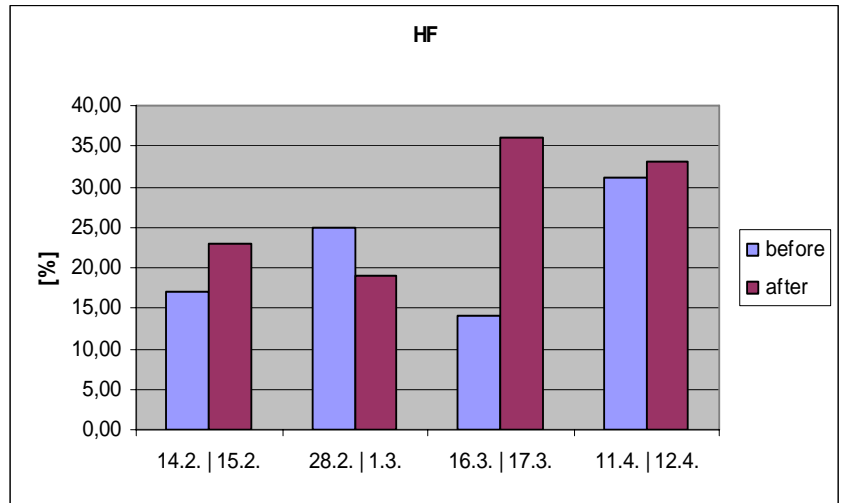


Fig. 35 Measures in HF [%] before and after treatment

Fig. 36 Measures in HF [%] before and after treatment

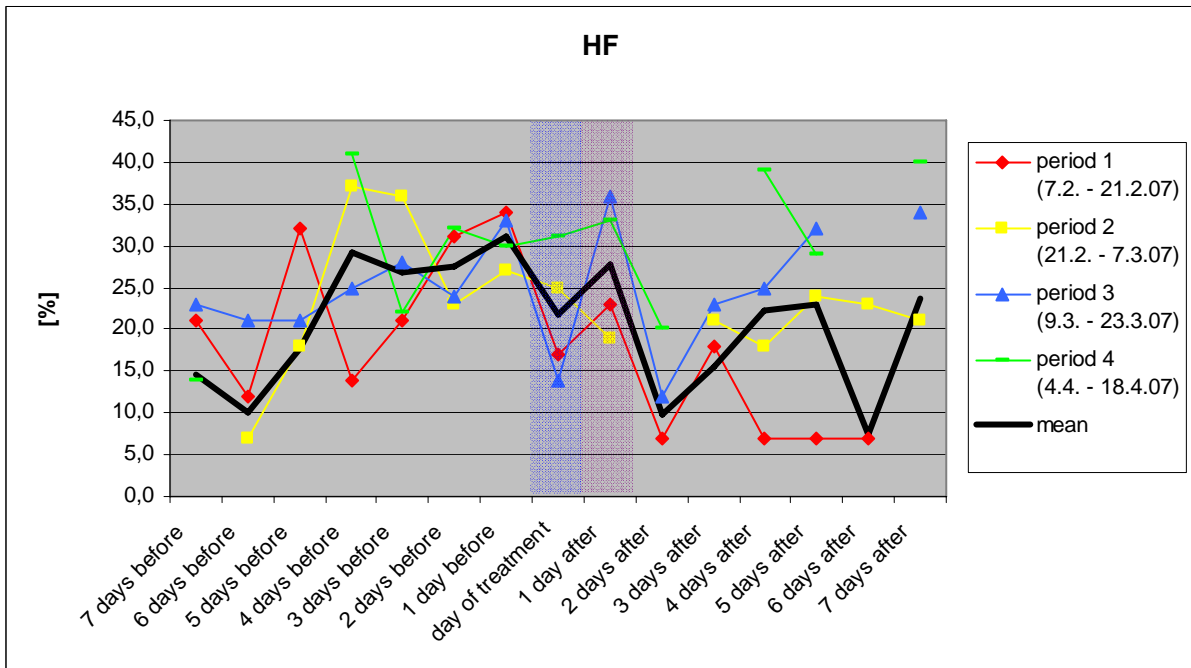


Fig. 37 Distribution of HF in four periods from 7.2.07 to 18.4.

The behaviour of frequency domain parameters the day after the treatment as well as the measures of all four measured single periods was irregular, as also was the mean value. (See Fig. 37)

RESULTS

Σ VLF+LF	7 days before	6 days before	5 days before	4 days before	3 days before	2 days before	1 day before	day of treatment	1 day after	2 days after	3 days after	4 days after	5 days after	6 days after	7 days after
period 1															
(7.2. - 21.2.07)	79,0	88,0	68,0	86,0	79,0	69,0	66,0	83,0	77,0	93,0	82,0	93,0	93,0	93,0	
period 2															
(21.2. - 7.3.07)		93,0	82,0	63,0	64,0	77,0	73,0	75,0	81,0		79,0	82,0	76,0	77,0	79,0
period 3															
(9.3. - 23.3.07)	77,0	79,0	79,0	75,0	72,0	76,0	67,0	86,0	64,0	88,0	77,0	75,0	68,0		66,0
period 4															
(4.4. - 18.4.07)	86,0			59,0	78,0	68,0	70,0	69,0	67,0	80,0		61,0	71,0		60,0
mean	82,5	86,7	76,3	70,8	73,3	72,5	69,0	78,3	72,3	87,0	79,3	72,7	77,0	85,0	68,3

Fig. 38 Distribution of Σ VLF+LF in four periods from 7.2.07 to 18.4.07

date	before	after	Diff. [%]
14.2.- 15.2.07	83	77	-7,22 %
28.2.- 1.3.07	75	81	8 %
16.3.- 17.3.07	86	64	-25,58%
11.4.- 12.4.07	69	67	-2,9 %

Fig. 39 Measures Σ VLF+LF [%] before and after treatment

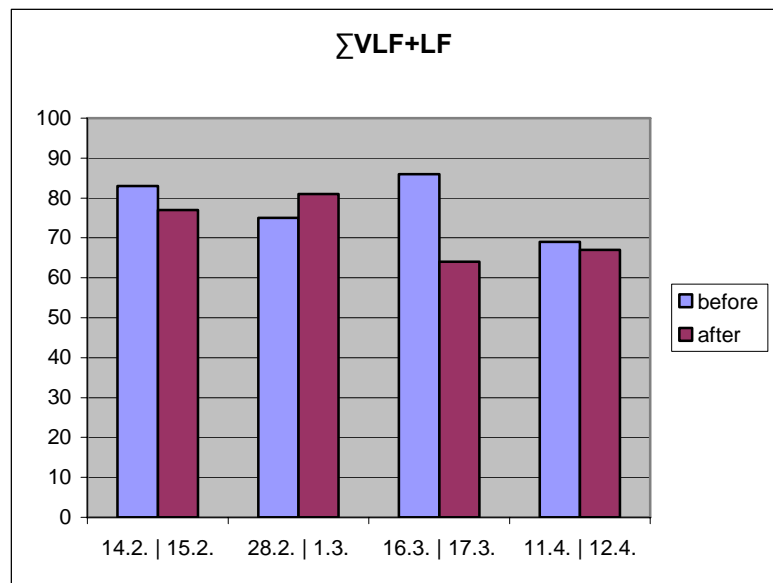


Fig. 40 Measures in Σ VLF+LF [%] before and after treatment

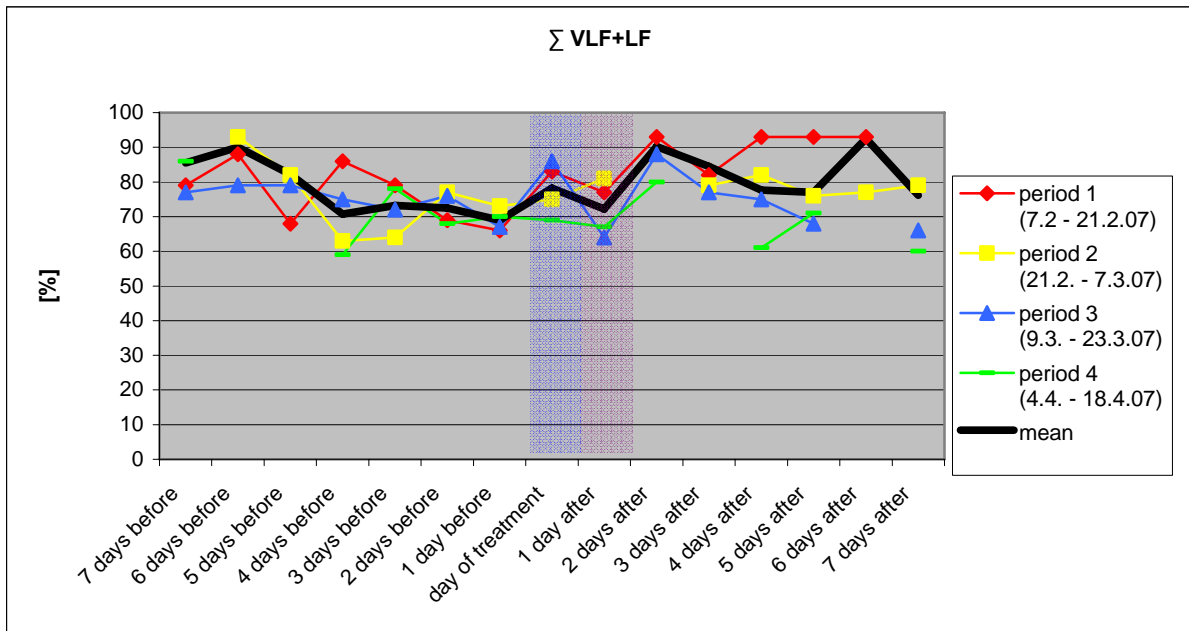


Fig. 41 Distribution of Σ VLF+LF in four periods from 7.2.07 to 18.4.07

Pearson’s correlation for frequency domain analysis (HF and Σ VLF+LF) comparing measures before and after the treatment over the fourteen day periods with $r = 1$ was not significant.

Training parameters

The results of the personal feelings during daily training on a scale from 1 (very good feeling) to 5 (very bad feeling) indicated an improvement after the Osteopathic treatments.

The mean of the personal feeling the seven days before the treatment was 3,13 whereas it decreased to 2,62 after the treatment. This corresponds to an improvement of 16,29% of the personal feeling. (See Fig. 42)

Personal feeling during training	7 days before	6 days before	5 days before	4 days before	3 days before	2 days before	1 day before	day of treatment	1 day after	2 days after	3 days after	4 days after	5 days after	6 days after	7 days after
period 1 (7.2. - 21.2.07)	5	3	2	3	3	3	4	3	2	3	1	3		3	
period 2 (21.2. - 7.3.07)		3	3	2	3	3	3	3	2		3	3	4	2	3
period 3 (9.3. - 23.3.07)	2	3		4	3	4	4	3	2	3	3	3	2		3
period 4 (4.4. - 18.4.07)	4			4	3	2	3	2	3	2	2	3			2
mean	3,67	3,00	2,50	3,25	3,00	3,00	3,50	2,75	2,25	2,67	2,25	3,00	3,00	2,50	2,67

Fig. 42 personal feeling during training

RESULTS

Intensity of training	7 days before	6 days before	5 days before	4 days before	3 days before	2 days before	1 day before	day of treatment	1 day after	2 days after	3 days after	4 days after	5 days after	6 days after	7 days after
period 1 (7.2. - 21.2.07)	2	2	3	3	2	3	2	2	2	3	3	2	1	1	
period 2 (21.2. - 7.3.07)		1	2	3	2	2	2	3	4	3	3	4	1	2	4
period 3 (9.3. - 23.3.07)	2	4		3	2	1	3	2	3	3	3	1	3	2	3
period 4 (4.4. - 18.4.07)	1		2	1	2	3	2	3	3	3	3	3		2	3
mean	1,67	2,33	2,33	2,50	2,00	2,25	2,25	2,50	3,00	3,00	3,00	2,50	1,67	1,75	3,33

Fig. 43 Intensity of training

The intensity of training as well as the period of training also increased after the treatments.

The mean of the intensity of training the seven days before the treatment was 2,19 whereas it increased to 2,61 after the treatment. This corresponds to an improvement of 19,18% of the intensity of training. (See Fig. 43)

duration of training [min]	7 days before	6 days before	5 days before	4 days before	3 days before	2 days before	1 day before	day of treatment	1 day after	2 days after	3 days after	4 days after	5 days after	6 days after	7 days after
period 1 (7.2. - 21.2.07)	25	182	193	158	65	203	120	135	200	225	185	195	145	110	
period 2 (21.2. - 7.3.07)		110	185	130	133	215	207	275	193	225	163	204	40	148	205
period 3 (9.3. - 23.3.07)	148	205	145	165	82	105	120	100	232	159	115	30	145	190	250
period 4 (4.4. - 18.4.07)	25	20	45	90	75	160	55	240	250	210	197	208	215	170	185
mean	66	129,3	142	135,8	88,75	170,8	125,5	187,5	218,8	204,8	165	159,3	136,3	154,5	213,3

Fig. 44 Duration of training

The mean of the duration of training the seven days before the treatment was 122 minutes whereas it increased to 178 minutes after the treatment. This corresponds to an improvement of 22% of the duration of training in th seven days after the treatment. (See Fig. 44)

5 Discussion

In the following chapters, I will explain my choice for a single subject design and present the results.

5.1 Single Subject Design

As individual influences have a very high impact on HRV measures (Betz and Brand, 2001) and pooling the results of different individuals might obscure significant findings, a single subject design was chosen for this investigation.

Kinugasa et al. (2004) also sustains that case by case analysis would appear to be a more appropriate method of investigating the impact of an intervention (e.g. an Osteopathic treatment) upon HRV.

“Despite the problems associated with data analyses in single-subject research (e.g. serial dependency and generality of findings), it is recommended that sports scientists use single-subject research designs in applied conditioning research to assess the effect of an intervention (e.g. a specific training method) and to predict performance for a particular athlete. The single subject approach is applicable to specific categories of subjects such as elite or overtrained athletes.” [...] “Further, we believe the application of single-subject-experimental designs and data analyses for assessment of athlete conditioning has an important place in effectively and efficiently monitoring elite athletes.”

(Kinugasa et al. 2004, p.1047-1048)

Experience has shown that it is not appropriate to pool HRV measures of different athletes, training more than 10 hours per week as HRV data would be too individual (personal statement of Kesselbacher A., personal trainer of the athlete measured in this study. 23.11.06).

5.2 Discussion of Results

5.2.1 Discussion of Pilot study

Circumstances concerning training intensity and duration of training as well as physical fitness were principally different during the pilot study and during the central study.

They were similar up to the end of June 06 (valid for first three measures). The athlete was healthy and trained regularly. Training intensity was between two and three on his recordings scale.

Values of time domain parameters (SD1 and RMSSD) of the first three treatments are comparable to the values in the central study. Both parameters showed an increase after the treatment. SD1 increased from 4,29% to 62,67% after the first three treatments. RMSSD moved from 3,94% to 56,06%.

Frequency domain measures moved after the treatment in HF from -56,8 to 52,6% and in Σ VLF + LF from -12,3 to 12,5 %.

From early July up to early August the athlete suffered from a number of infections and hardly trained during this period of time.

An ENT operation on 6.August reduced training to a minimum, at times even made it impossible (valid for fourth and fifth measurements). The decrease in SD1 and RMSSD can clearly be seen in the low figures before the treatment, due to the athlete's weak condition. In comparison the increase of both parameters after the therapy was remarkably high. SD1 increased 379% resp. 127% and RMSSD 380% resp. 127% after fourth and fifth treatment.

End of August the athlete resumed with moderate training (valid for sixth and seventh measurements). SD1 and RMSSD values before the treatment had already improved but did not reach anymore the value they had reached by the end of June. During this period the athlete still felt somewhat weak. SD1 increased 75,95% resp. 79,42%, and RMSSD 75,59 resp. 79,24% after the sixth and seventh treatments.

The pilot study showed a correlation between HRV changes and health condition. Furthermore, the reaction to the treatment also varied before, during and after the period of illness. The reason could have been the altered health conditions itself or the difference of duration and intensity of training.

Results were at the highest in the time with no or hardly any training and at the lowest under normal training conditions. One can also conclude that results were at the highest during illness and at the lowest while physically healthy.

To sum up, the increase of SD1 (representing the regulatory capacity of the athlete) and RMSSD (reflecting the body's ability to recover) the day after all seven Osteopathic

treatments can be interpreted with an enhanced capacity for physical performance. (See 2.5.5 Significance of HRV Measures and 2.5.6 HRV and Performance)

In frequency domain, both HF and \sum VLF + LF before and after the treatment did not show homogenous changes. Absolute values which might have been more expressive could not be calculated because of the different length of recordings. (See 2.5.4 Frequency Domain Measures)

5.2.2 Discussion of Central study

Training intensity and duration were significantly higher during the central study compared to the pilot study, with moderate training until June and new start from September on.

Training parameters remained about the same during the first three periods of the central study. Before the treatment of the fourth period the athlete had influenza for 10 days and started with training the day before treatment.

Results one day after the Osteopathic treatment:

In the central study, results of SD1 and RMSSD before and after the treatment are comparable to measures in the pilot study.

In the first three treatments SD 1 increased from 1,47% to 21,31% the day after the treatments compared to the day before the treatments and RMSSD from 3,14% to 22%.

Like in the pilot study, time domain measures increased the day after the Osteopathic treatment, except for the last measure after 10 days influenza. SD1 decreased to -33,77% and RMSSD -30%.

However, the increase rate after the first three treatments was smaller than in the pilot study. One possible explanation for the less significant result could be the increase of training load in the central study.

In frequency domain, HF power increased in three out of four treatments from 6,45% to 157,14% the day after the treatment, reflecting an increased parasympathetic activity after the treatment, a sign for enhanced ability to recover. No explanation was found for the decrease of HF power after the second treatment. The reason could have been psychological disturbances, but this was not confirmed by the athlete.

For \sum VLF + LF the opposite applies. Three out of four measures indicated a decrease of sympathetic activity reflecting an decreased sympathetic activity after the treatment.

Results in the longer 14 day periods:

The behaviour of time domain and frequency domain parameters was irregular. Only very weak correlation could be found in time domain measures ($r=0,996$) and no correlation could be found in Pearson's correlation for frequency domain measures ($r=1$). Mean values of all time and frequency parameters also were inconsistent.

The irregularity of all measures in the central study might be the result of the different training intensity and duration, changing every day because of personal feelings.

The feeling after the treatments increased positively, so did the duration and intensity of the trainings. Both were increased by the athlete himself because of the good feeling during training. He had the possibility to adapt duration and intensity of the training up to a certain extent within the training schedule of his trainer.

Training load has a major impact on HRV. This could be a reason, why HRV decreased again after the first incline the day after the first three treatments.

The decrease of heart rate variability after the fourth treatment could be interpreted as an "overload" of the body's internal regulatory systems and a decompensation of the body. Ten days influenza, new start with training sequences plus inputs of the treatment could have been too much for the regulatory systems. The personal feeling also decreased from 2 to 3 the day after the treatment.

5.3 Conclusion

The **pilot study** showed increased HRV in time domain parameters between measures the day before and the day after all seven Osteopathic treatments.

As a matter of fact, a parallel could be drawn between the increased HRV and positive conditions for increased sport performance, at least in the case of a short time treatment.

Same as in the pilot study, similar results were observed in the **central study** in time domain measures the day after treatment. A sudden illness totally changed the athlete's reaction to the Osteopathic treatment. The measures decreased drastically.

Contrary to the short period study, in all four 14 day time measures (7 days before and 7 days after every treatment) no correlation could be found in time or frequency domain analyses before and after the treatment.

Even though the measures did not reflect it, an improvement could be noticed in the personal feeling during training the 7 days after the treatment. Nevertheless, the subjective impression of the athlete during training was obviously better and he could thus train for longer periods.

To summarise, the hypothesis that Osteopathic treatments influence the heart rate variability of high-performance athletes proved to be true in short term. The measures taken during the long term treatment did not allow to draw such a conclusion even though the athlete felt better. The choice of the measuring system might have been a reason for the impossibility to demonstrate this “better feeling” through figures.

5.4 Clinical Relevance

Athletes always aim at enhancing their performance. Optimizing their training parameters might come to its limits in foreseeable future. Osteopathic treatments might offer an opportunity to optimise physical performance in sense of enhancing homodynamic balance.

5.5 Recommendations for further research

Further research is needed to test the premise that an Osteopathic treatment can increase the ability of the body to react in a more efficient way to changing influences, to better adapt and therefore can increase physical performance.

HRV is an expression of very complex interactions within different body systems and the VNS (see 2.5. Heart Rate Variability). External and internal influences (see 2.5.12. Limitations) should therefore be taken into consideration in any case.

Considering the fact that respiration influences HRV, standardised breathing rate (e.g. by synchronizing their breathing pattern with an electronic metronome rhythm) should be taken into account for the comparison of measures of different subjects.

If athletes are investigated, training load before and after the treatment should be standardised.

6 Summary

Athletes always aim to enhance their performance. The optimisation of their training parameters might come to its limits in foreseeable future. Osteopathic treatments might offer an opportunity to optimise physical performance in sense of enhancing homodynamic balance.

Subject of this research was to demonstrate the effects of an Osteopathic treatment on the body's possibility to achieve peak performance.

This research was conducted in two stages. The objective of the **pilot study** was to find out if there are any change seen in heart rate variability (HRV) before and the day after the treatment.

As results were promising, the **central study** was planned.

HRV seemed to be an ideal tool for this investigation as described in the introduction.

This measuring method was chosen to find out whether an osteopathic treatment can enhance the performance of an athlete.

HRV measures appear to provide a quantitative evaluation of the sympathovagal interaction modulating cardiovascular function (Malliani 1991). They have considerable potential to assess the vegetative nervous system (VNS), the body's most comprehensive regulatory system (Mück-Weymann 2005). HRV measures also reflect the flexibility and regulatory capacity of the organism to adapt to internal and external changes (Cerruti et al. 1995) and can be a determinant of physical fitness (Goldsmith et al. 1997).

Within the concept of homodynamic which has been discussed in the introduction, a balanced VNS provides an essential precondition for a better communication between the different regulatory systems of the body. This represents an important precondition for enhanced performance.

*“Systems-oriented models propose that HRV is an important indicator of both physiological resiliency and behavioural flexibility, **reflecting the individual's capacity to adapt effectively to stress and environmental demands.**”* (McCraty et al. 2001 p.1)

This statement underlines the regulatory capacity that may be deduced from HRV measurements.

Interactions between VNS, heart-rate and respiration are to be found in chapter 2.3 and 2.4.

In this study, time domain and frequency domain analysis of HRV were used to compare sympathetic and parasympathetic power before and after an Osteopathic treatment to evaluate the impact of the treatment on physical performance. The basics of HRV, its significance and how it is influenced is to be found in chapter 2.5, Heart Rate Variability.

The state of cardiac autonomic control was inferred from heart rate variability (HRV) and analysed over a period of 10 weeks. Measures were performed 7 days before and 7 days after the treatment in the morning.

A significant relationship was not to be found between an Osteopathic treatment and a change in HRV before and after an Osteopathic treatment. Therefore, it could not be proved that an Osteopathic treatment has a significant impact on the performance of a high-performance athlete but for the fact that the **personal feeling improved** during training periods the days after the treatments.

The expected increase of parasympathetic activity in HRV after the treatment as an indication of homodynamic properties of the body could not be confirmed. Unexpectedly for the researcher and thanks to the increase of the positive feeling during training, the periods of training were extended and the intensity increased by the athlete.

Both **pilot and central studies** showed an **increase** in the investigated time domain parameters the day after the treatment except the last treatment after a 10 day influenza. (For the pilot study HRV measures were only performed in the morning on the day of therapy and the following morning). The long term measurements (4 x 14 days) did not indicate such increase.

7 Acknowledgements

I would like to give very special thanks to Mag. Anton Kesselbacher, sports scientist and trainer of Andreas Giglmayr, the athlete of this study, for his patience to answer all my questions concerning HRV and statistics.

Andreas Giglmeier for participating in this study and to take on a one hour drive by car for each therapy.

Mag. Katharina Musil to constructively support and supervise my work.

I also would like to thank Mag. Jackline Priour to competently translate my thesis.

DI (FH) Günther Böhmüller, my brother, to have always time answering my questions concerning layout and graphic designs.

My parents and my friends who patiently supported me during this demanding phase, also deserve very special thanks.

8 Abstract

Study Design: Single subject design

Problem definition: Athletes always aim at enhancing their performance. Optimizing their training parameters might come to its limits in foreseeable future. Osteopathic treatments might offer an opportunity to optimise physical performance.

Research question: Is it possible to enhance the performance of high-performance athletes with Osteopathic treatments?

Hypothesis: Heart Rate Variability (HRV) can be increased in high-performance athletes by Osteopathic treatments.

Relevance for patients: Optimising performance is always the aim of high-performance athletes. Osteopathic treatments could be a valuable tool in optimising different body systems and therefore enhance physical performance.

Relevance for Osteopathie: Evidence of a positive influence of Osteopathic treatments on the performance of high-performance athletes can help to establish Osteopathy in the field of top-class sport.

Methodology: For this study a young (23 years of age) male, physically and mentally healthy high-performance tri-athlete was chosen. A pilot study was done over a period of about five months (31.5.06 – 26.10.06) to investigate changes in HRV in the morning before and the day after 7 Osteopathic treatments.

In February 2007 a single subject design study over a period of 10 weeks was started. Four Osteopathic treatments with intervals of at least 2 weeks in between were carried out. HRV short-term recordings, were made by the athlete every morning immediately after waking up. HRV recordings in time and frequency domain were analysed 7 days before and 7 days after

the treatment to show the behaviour of measures. Besides HRV measures, the athlete recorded his personal feelings during training on a scale from 1 (very good feeling) to 5 (very bad feeling) every day. Training intensity was recorded on a scale from 1 (regenerative training) to 5 (competition). Duration of daily training was recorded in minutes every day.

To ensure a physiologically comparable period of recording Osteopathic treatment were timely spread in the middle of moderate training sequences.

Results: Pilot study: In time domain SD1 and RMSSD all measures increased the day after all seven treatments. In frequency domain, measures before and after the treatment showed an irregular behaviour.

Central study: Time domain parameters of HRV increased the day after the first three treatments but decreased the day after the fourth treatment after a 10 day influenza. The distribution of time domain parameters over all four time periods (4x 14 days) showed an irregular behaviour before and after the treatment. Pearson's correlation for time domain parameters comparing measures before and after the treatment was very weak with ($r = 0,996$).

Measures in frequency domain analysis did not show coherent increases on the following morning after the treatment. Long term measurements (4 x 14 days) showed irregular behaviour before and after the treatment. Pearson's correlation for frequency domain analysis comparing measures before and after the treatment over the fourteen day periods with $r = 1$ was not significant.

The mean of personal feeling the seven days after the treatment increased 16,29% compared to the 7 days before the treatments.

Conclusion: The hypothesis that Osteopathic treatments can positively influence the heart rate variability and therefore physical performance of high-performance athletes proved to be true in short term measures.

Contrary to the short period study, in all four 14 day time measures (7 days before and 7 days after every treatment) no correlation could be found in time or frequency domain analyses before and after the treatment.

Even though the measures did not reflect it, an improvement could be noticed in the personal feeling during training the 7 days after the treatment.

Perspectives: To test the premise that an Osteopathic treatment can increase the ability of the body for physical performance on a longer term, other measuring devices might be thought about to be applied.

9 References

Anrep G., Pascual W., Rossler R.: Respiratory Variations of the Heart Rate - The Reflex Mechanism of the Respiratory Arrhythmia. *Proceedings of the Royal Society of London. Series B, Biological Sciences*, Vol. 119, No. 813 (Jan. 1, 1936), pp. 191-217

Arbeitsgemeinschaft der Wissenschaftlichen Medizinischen Fachgesellschaften: Herzrhythmusanalyse in der Arbeitsmedizin. Leitlinien der Deutschen Gesellschaft für Arbeitsmedizin und Umweltmedizin e.V. (DGAUM) AWMF-Leitlinien-RegisterNr. 002/021, 2006 <http://www.uni-duesseldorf.de/AWMF/II/002-021.htm>

Bakewell S., The Autonomic Nervous System, Update in Anaesthesia, Addenbrooke's Hospital, Issue 5 Article 6, Cambridge (1995).
http://www.nda.ox.ac.uk/wfsa/html/u05/u05_010.htm

Beal M.: Viscerosomatic Reflexes: A Review. AAO Millennium Yearbook, reprinted from the Journal of the American Osteopathic Association, Indianapolis, edited by Beal M. 2001, reprinted from Journal of the American Osteopathic Association. December 1985. 85:12:53-6, pp 123-138

Betz M., Brand S.: Objektivieren von Entspannungstechniken mit Hilfe der Herzfrequenzvariabilität. In:Hottenrott K.: Herzfrequenzvariabilität im Sport. *Schriften der Deutschen Vereinigung für Sportwissenschaft*. Band 129. Verlag: Cwalina, Hamburg, 2001

Bischof M.: Biophotonen Das Licht in unseren Zellen, *Zweitausendeins* 1995, 13. Auflage 2005

Blasi A., Jo J., Valladares E., Morgan B., Skatrud J., and Khoo M.: Cardiovascular variability after arousal from sleep: time-varying spectral analysis. *J Appl Physiol* 95: 1394-1404, 2003
<http://jap.physiology.org/cgi/content/full/95/4/1394>

Buchheit M., Gindre C.: Cardiac parasympathetic regulation: respective associations

with cardiorespiratory fitness and training load. *Am J Physiol Heart Circ Physiol* 291: H451-H458, 2006. <http://ajpheart.physiology.org/cgi/reprint/00008.2006v2?ck=nck>

Buchheit M., Simon C., Piquard F., Ehrhart J., and Brandenberger G.: Effects of increased training load on vagal-related indexes of heart rate variability. *Am J Physiol Heart Circ Physiol* 287: H2813-H2818, 2004

<http://ajpheart.physiology.org/cgi/content/full/287/6/H2813>

Camm A., Malik M., Bigger J., Breithardt G, Cerutti S., Cohen R., Coumel P., et al. 1996. Heart Rate Variability. Standards of Measurement, Physiological Interpretation, and Clinical Use. *Circulation*. 93(5): S.1043-1065.

Cerutti S., Bianchi A.M., Mainardi L.T.: Spectral analysis of the heart rate variability signal. In: Malik, M. und Camm, A.J. (Hrsg.): Heart rate variability. Armonk, NY: Futura Publishing Company, Inc. 1995 p.63-74

Cooke W., Hoag J., Crossman A., Kuusela T., Tahvanainen K., Eckberg, D.: *The Journal of Physiology* 517, 617–628. 1999

Deboer R., Karemaker J., Strackee J.: *American Journal of Physiology* 253, H680–689. 1987

De Haas G.: Fahrprüfung ohne Streß, Ein Steßbewältigungs- und Untersuchungskonzept mit besonderer Berücksichtigung chronobiologischer Prozesse. Hochschule für angewandte Psychologie, Zürich 2005

Du N., Bai S., Oguri K., Kato Y., Matsumoto I., Kawase H., Toshio Matsuoka T.: Heart Rate Recovery after exercise and neural regulation of heart rate variability in 30-40 year old female marathon runners. *Journal of Sports Science and Medicine* (2005) 4, 9-17

<http://www.jssm.org/vol4/n1/2/v4n1-2pdf.pdf>

Duus P.: Neurologisch-topische Diagnostik: Anatomie, Physiologie, Klinik. 4. überarb. Auflage, Georg Thieme Verlag, Stuttgart, 1987

Dobson P., O’Keeffe E.: Research into the efficacy of the Gas Discharge Visualisation Technique as a measure of physical and mental health. Erstveröffentlichung 2001, downloaded 5.1.2006 www.gdvtechnique.com

Engel R.: 'Cranial' State of Mind - Does cranial osteopathy influence the patient's state of consciousness? Master Thesis for the degree „Master of Science (Osteopathie)“ at the Donau-Universität Krems submitted to the Wiener Schule für Osteopathie 2006 http://www.osteopathicresearch.com/paper_pdf/Engel.pdf

Eckberg Dwain L., MD: Sympathovagal Balance A Critical Appraisal American Heart Association Circulation. 1997;96:3224-3232.) http://circ.ahajournals.org/cgi/content/full/96/9/3224?ijkey=d6238b90fa19c8762535927a4100f15cc3b21dd9&keytype=tf_ipsecsha

Esperer H.D.: Physiologische Grundlagen der Herzfrequenzvariabilität. In: Hottenrott K., (Hrsg.), Herzfrequenzvariabilität im Fitness- und Gesundheitssport. Schriften der Deutschen Vereinigung für Sportwissenschaft. Band 142, S.11-40, Verlag: Cwalina, Hamburg 2003

Ferscha A., Pokan R., Bachl N., Smekal G.: Herzfrequenzvariabilität in Ruhe und unter Belastung. Methodische Aspekte und deren Aussagekraft. In: *Österreichisches Institut für Sportmedizin (Hrsg.): Handbuch für Sportmedizin*. 28. Jahrgang. Wien 1998

Frymann V., Carney R., Springall P.: Effect of Osteopathic Medical Management on Neurologic Development in Children. . The AAO Millennium Yearbook, American Academy of Osteopathy, Indianapolis, edited by Beal M. 2001, reprinted from the Journal of the American Osteopathic Association. June 1992. 92:6:729-744

Furlan R., Piazza S., Dell’Orto S., Genitle E., Cerutti S., Pagani M., Malliani A.: Early and late effects of exercise and athletic training on neural mechanisms controlling heart rate Cardiovascular research 1993: 27: 482-488

Gamelin F.X., Berthoin S., Bosquet L.: Validity of the Polar S810 Heart Rate Monitor to Measure R-R Intervals at Rest. *Medicine & Science in Sports & Exercise*. 38(5):887-893, May 2006.

Garet M, Tournaire N, Roche F, Laurent R, Lacour JR, Barthelemy JC, Pichot V.:Laboratory of Clinical and Exercise Physiology, PPEH Group, Synapse Research Group, University Jean Monnet, France. Individual Interdependence between nocturnal ANS activity and performance in swimmers. *Md Sci Sports Exerc.* 2004 Dec;36(12):2112-8.

GDV-Tecology: BEO GDV Technique, based on Kirlian Effect, advanced scientific tool to study mind-body functions , www.gdvtec.com, down loaded 12.4.06

Goldberger Jeffrey J.: Sympathovagal balance: how should we measure it? *Am J Physiol Heart Circ Physiol*, April 1, 1999; 276(4): H1273 - H1280.

<http://ajpheart.physiology.org/cgi/reprint/276/4/H1273>

Goldsmith R., Rochelle L., Bigger J. Jr., Bloomfield D., Steinman R.: Physical fitness as a determinant of vagal modulation. *Applied Sciences Medicine & Science in Sports & Exercise.* 29(6):812-817, June 1997

Gutenbrunner Ch.: Die Bedeutung der Chronobiologie bei der vegetativen Steuerung der Organfunktion. Wiener internationale Akademie für Ganzheitsmedizin, 2005

Haberl F.: The influence of Osteopathic treatment on the performance of hobby runners. A Comparative, Clinical Study Using Osteopathic Treatment to Enhance the Performance of Hobby Runners. Masterthesis – Osteopathy at the Donau Universität Krems 2006. http://www.osteopathicresearch.com/paper_pdf/Haberl.pdf

Hirsch J. A., Bishop B.: Respiratory sinus arrhythmia in humans: how breathing pattern modulates heart rate. *Am J Physiol Heart Circ Physiol* 241: H620-H629, 1981;

Hon E., Lee S.: Electronic evaluations of the fetal heart rate patterns preceding fetal death: further observations. *Am J Obstet Gynecol.* 1965;87:814-826

Hoos O.: Spektalanalyse der Herzfrequenzvariabilität im Sport- Methoden und Anwendungen, Möglichkeiten und Grenzen. In: Hottenrott K.: Herzfrequenzvariabilität: Methoden und

Anwendungen in Sport und Medizin. *Schriften der Deutschen Vereinigung für Sportwissenschaft*. Band 162. Verlag: Cwalina, Hamburg 2006

Hoover H.: Functional Technic in Osteopathic Manipulative Treatment. The AAO Millennium Yearbook, American Academy of Osteopathy, Indianapolis, edited by Beal M. 2001, reprinted from Journal of the American Osteopathic Association. December 1956. pp 233-237.

Horn A.: Diagnostik der Herzfrequenzvariabilität in der Sportmedizin –Rahmenbedingungen und diagnostische Möglichkeiten. *Dissertation*: Ruhr-Universität Bochum, Universitätsbibliothek, Fakultät für Sportwissenschaft, 2004

Hottenrott K.: Grundlagen der Herzfrequenzvariabilität und Anwendungsmöglichkeiten im Sport. In: Hottenrott K.: Herzfrequenzvariabilität im Sport. *Schriften der Deutschen Vereinigung für Sportwissenschaft*. Band 129. Verlag: Cwalina, Hamburg, 2001

Houle M. and Billman G.: Low-frequency component of the heart rate variability spectrum: a poor marker of sympathetic activity *Am J Physiol Heart Circ Physiol* Vol. 276, Issue 1, H215-H223, January 1999

<http://ajpheart.physiology.org/cgi/content/full/276/1/H215>

Hugdahl K: Cognitive influences on human autonomic nervous system function. *Curr.Opin.Neurobiol.* 1996, 6:252-258.

Iellamo F., Legramante J., Pigozzi F., Spataro A., Norbiato G., Lucini D., Pagani M.: Conversion From Vagal to Sympathetic Predominance With Strenuous Training in High-Performance World Class Athletes. American Heart Association, Inc., *Circulation*. 2002;105:2719.) <http://circ.ahajournals.org/cgi/content/full/105/23/2719>

Karemaker M.: Autonomic integration: the physiological basis of cardiovascular variability. *The Journal of Physiology* 517 (2), 316–316. doi:10.1111/j.1469-7793.1999.0316t.x (1999)

Kesselbacher A.: Frequenzanalyse der HRV und Zeitreihenanalyse der Trainingsdaten von Triathleten. Diplomarbeit, Sportuniversität Salzburg 2003

Kestin I.: Control of Heart Rate. Update in Anaesthesia, Issue 3, Article 3, 1993

Kinugasa T., Cerin E. and Hooper S.: Single-Subject Research Designs and Data Analyses for Assessing Elite Athletes' Conditioning. Sports Med 2004; 34 (15)

Kleiger R., Bigger J., Bosner M., Chung M., Cook J., Rolnitzky L., Steinman R, Fleiss J. Department of Medicine, Washington University School of Medicine, St. Louis, Missouri. Stability over time of variables measuring heart rate variability in normal subjects. *Am J Cardiol.* 1991 Sep 1;68(6):626-30.

Korr I. M.: The Biological Basis for the Osteopathic Concept, The AAO Millennium Yearbook, Indianapolis, edited by Beal M. 2001, reprinted from the American Academy of Osteopathy's 1963 Yearbook, pp. 114-120

König B.O., Schumacher O.Y., Schmidt-Trucksäss A., Berg A.:Herzfrequenzvariabilität- Schon reif für die Praxis? Leistungssport, Zeitschrift für die Fortbildung von Trainern, Übungsleiter und Sportlehrer, Deutscher Sportbund, 33.Jahrgang, Mai 2003

Lau S., Haueisen J., Schukat-Talamazzini E., Voss A., Goering M., Leder U., Figulla H.: Entropy Estimation Methods in HRV Analysis of Patientswith Myocardial Infarction, Friedrich –Schilleruniversität Jena, Jenaer Schriften zur Mathematik und Informatik 2005
<http://www.minet.uni-jena.de/Math-Net/reports/sources/2006/06-02report.pdf>

Liem T., Craniosacrale Osteopathie, Hippokrates Verlag, Stuttgart 2001

Liem T., Dobler T.K.: Leitfaden Osteopathie, Parietale Techniken, Urban und Fischer, München, 2002

Löllgen D., Jung K., Mück-Weymann M.: Herzratenvariabilität im Sport- Methodische Überlegungen zur vergleichenden Messung mittels Polar S810 und Standardmethoden der Medizin. In: Hottenrott K.: Herzfrequenzvariabilität im Fitness- und Gesundheitssport. Schriften der Deutschen Vereinigung für Sportwissenschaft. Band 142. Verlag: Cwalina, Hamburg 2003

Mahlke Ch., Rauh R., Bauer R., Mück-Weymann M.: Validisierung des Polar Advantage für standardisierte Bestimmung der Herzratenvariabilität. In:Hottenrott K.: Herzfrequenzvariabilität im Sport. Schriften der Deutschen Vereinigung für Sportwissenschaft. Band 129. Verlag: Cwalina, Hamburg, 2001

Malliani A., Pagani M., Lombardi F. and Cerutti S.: Cardiovascular neural regulation explored in the frequency domain. 1991;84;482-492 *Circulation*
<http://circ.ahajournals.org/cgi/reprint/84/2/482.pdf>

Malliani A., Pagani M., Furlan R., Guzzetti S., Lucini D., Montano N., Cerutti S., Mela G.: Individual Recognition by Heart Rate Variability of Two Different Autonomic Profiles Related to Posture, American Heart Association Inc., *Circulation*. 1997; 96:4143-4145.
<http://circ.ahajournals.org/cgi/content/full/96/12/4143?ijkey=186de65cb1f149e380c01560337969ea88f2e1be>

Malliani Alberto: The Pattern of Sympathovagal Balance Explored in the Frequency Domain
News Physiol Sci 14: 111-117, 1999
<http://physiologyonline.physiology.org/cgi/content/full/14/3/111>

Malik M., Bigger J. T., Camm A.J., Kleiger R.E., Malliani A., Moss A.J., Schwartz P.J.: Heart rate variability: Standards of measurement, physiological interpretation, and clinical use. *Eur. Heart J.*, March 1996; 17: 354 – 381
<http://www.circ.ahajournals.org/cgi/content/full/93/5/1043>

Martinmaki K, Rusko H, Kooistra L, Kettunen J, Saalasti S.: Intraindividual Validation of Heart Rate Variability Indices to Measure Vagal Effects on the Heart KIHU - Research Institute for Olympic Sports, Jyväskylä, Finland; University of Jyväskylä, Department of Biology of Physical Activity, Jyväskylä, Finland.:. *Am J Physiol Heart Circ Physiol*. 2005 Sep 19;

McCraty R., Tiller W., Atkinson M.; In: Proceedings of the Brain-Mind. Applied Neurophysiology EEG Neurofeedback Meeting. Key West, Florida, 1996.

McCraty R., Atkinson M., Tomasino D.: Science of the Heart. HeartMath Research Center, Institute of HeartMath, Publication No. 01-001. Boulder Creek, CA, 2001.

<http://www.thenaturalsolution.biz/sitebuildercontent/sitebuilderfiles/scienceoftheheart.pdf>

<http://www.heartmath.org/research/research-publications.html>

Moser M., Bonin v. D., Frühwirth M., Lackner H.: Jede Krankheit ein musikalisches Problem. Rhythmus und Hygiogenese, *dieDrei* 8-9, 2004

http://www.joanneum.at/uploads/tx_publicationlibrary/img2881.pdf

Mück-Weymann M., Beise R.: Herzkohärenztraining – eine moderne Form der Stressbewältigung. Forum Stressmedizin 2005 – I: 1-5.

Oschman J. L.: Energy Medicine in Therapeutics and Human Performance, Butterworth-Heinemann, an imprint of Elsevier Limited, Philadelphia 2003, reprinted 2006

Pischinger A.: Das System der Grundregulation Grundlagen einer ganzheitsbiologischen Medizin. Karl F. Haug Verlag, Heidelberg 1975, 8. Auflage 1990

Pluim B.M., Swenne C. A., Zwinderman A.H., Maan A.C., van der Laarse A, Doornbos J., Van der Wall E.E.: Correlation of heart rate variability with cardiac functional and metabolic variables in cyclists with training induced left ventricular hypertrophy *Heart* 1999, 81:612-617

Rogers J., and Rogers J.: The Role of Osteopathic Manipulative Therapy in the Treatment of Coronary Heart Disease The AAO Millennium Yearbook, Indianapolis, edited by Beal M. 2001, reprinted from the Journal of the American Osteopathic Association. September 1976. 76:1:71-81 pgs. 343-351

Ruddy T.: Osteopathic Rhythmic Resistive Duction Therapy, The AAO Millennium Yearbook, Indianapolis, edited by Beal M. 2001, reprinted from the Academy of Applied Osteopathy's 1961 Yearbook, pgs. 169-179

Schaefer S.: Arrhythmien und eingeschränkte Herzfrequenzvariabilität bei Patienten mit Score-quantifizierter Sepsis und Score-quantifiziertem MODS – eine prospektive Studie auf einer internistischen Intensivstation. Dissertation zur Erlangung des akademischen Grades Doktor der Medizin an der Medizinischen Fakultät der Martin-Luther-Universität Halle-Wittenberg, 2006

<http://sundoc.bibliothek.uni-halle.de/diss-online/06/07H005/>

Stein P., Kleiger R., Rottman J.: Differing effects of age on heart rate variability in men and women. Division of Cardiology, Barnes-Jewish Hospital, Washington University Medical Center, St. Louis, Missouri 63110.: *Am J Cardiol.* 1997 Aug 1;80(3):302-5

Stiles Edward G.: Osteopathic Manipulation in a Hospital Environment, *The AAO Millennium Yearbook*, Indianapolis, edited by Beal M. 2001, *reprinted from the Journal of the American Osteopathic Association. December 1976. 76:12:67-82, p.p 328-342*

Task Force of the European Society of Cardiology the North American Society of Pacing Electrophysiology: Heart Rate Variability Standards of Measurement, Physiological Interpretation, and Clinical Use. *Circulation.* 1996;93:1043-1065, American Heart Association Inc., 1996 <http://www.circ.ahajournals.org/cgi/content/full/93/5/1043>

Taylor J.; Eckberg D.: Fundamental Relations Between Short-term RR Interval and Arterial Pressure Oscillations in Humans. *Circulation.* 1996;93:1527-1532. <http://circ.ahajournals.org/cgi/content/full/93/8/1527>

Tucek, G. Traditional oriental music therapy – a regulatory and relational approach. *Music Therapy Today*, Vol.VII (3) 623-647, 2006 http://www.musictherapyworld.de/modules/mmmagazine/index_dynamisch.php?issue=46&article=179

Udgaonkar J.: Entropy in Biology. *Resonance Journal*, USA, Sept 2001 <http://www.ias.ac.in/resonance/Sept2001/pdf/Sept2001p61-66.pdf>

Vestweber K., Hottenrott K.: Einfluß einer speziellen Entspannungs- und Konzentrationstechnik (Freeze-Frame) auf Parameter der Herzfrequenzvariabilität. In:

Hottenrott K.: Herzfrequenzvariabilität im Sport. Schriften der Deutschen Vereinigung für Sportwissenschaft. Band 129. Verlag: Cwalina, Hamburg 2001

Wang X, Thayer J., Treiber F, Snieder H.: Ethnic differences and heritability of heart rate variability in african- and European american youth. Georgia Prevention Institute, Department of Pediatrics, Medical College of Georgia, Augusta, Georgia.: *Am J Cardiol.* 2005 Oct 15;96(8):1166-72. Epub 2005 Aug 31.

Weeber E.: Ausdauersport ja, aber bitte richtig, Sportmedizinische Erkenntnisse und Fakten. www.tsv-steingaden.de/ausdauersport/WirkungenAusdauertrainings.pdf down-loaded 14.4.07

Weibel R.: Stress-Management, Strategien zur erfolgreichen Stressbewältigung, HRM-Dossier Nr. 25 (Schriftenreihe für Human Resource Management-Fachleute) SPEKTRAMedia, Zürich, 2004

Werdan K., Schmidt H., Hennen R., Müller-Werdan U.: Herzfrequenzvariabilität- Etablierte Indikationen und neue Ansätze in der Medizin. In: Hottenrott K.: Herzfrequenzvariabilität: Methoden und Anwendungen in Sport und Medizin. *Schriften der Deutschen Vereinigung für Sportwissenschaft.* Band 162. Verlag: Cwalina, Hamburg 2006

Wolf M., Varigos G., Hunt D., and Sloman J.: Sinus arrhythmia in acute myocardial infarction. *Medical Journal of Australia*, 2:52–53, 1978.

Wühr E.: Form und Funktion des Kraniomandibulären Systems für Osteopathen, Osteopathie als Systemische Medizin, Kooperation zwischen Osteopath und Zahnarzt, unveröffentl. Skript WSO 2007

Yamamoto Y., Hughson R. L. and Peterson J. C.: Autonomic control of heart rate during exercise studied by heart rate variability spectral analysis, *J Appl Physiol* 71: 1136-1142, 1991;

List of Figures

Fig. 1 (Bakewell 1996, modified after Böhmüller 2007).....	8
Fig. 2 Origin and service areas of the VNS (Slater 2000 after Hamill 1997, modified after Böhmüller.....	10
Fig. 3 (Campbell et al. 2006, Results).....	14
Fig. 4 (Moser 2004, p.30).....	16
Fig. 5 The Heart's Electromagnetic Field (McCrathy 2001, p.20.....	17
Fig. 6 (Mc Craty et al. 2001, p.14).....	19
Fig. 7 (McCraty 2001, p.13, modified after Böhmüller 2007).....	21
Fig. 8 Malliani (1991) p.483, modified after Böhmüller 2007	21
Fig. 9 (Task Force 1996, Standard measurement of HRV.).....	24
Fig. 10 Selected time domain heart rate variability indices (Malik et al. 1996 p.6, modified after Böhmüller 2007)	25
Fig. 11 Selected frequency domain heart rate variability indices (Mallik et al 1996, modified after Böhmüller 2007)	27
Fig. 12 (McCraty et al 2001, p.14).....	27
Fig. 13 (Malliani 1997 et al, Results).....	30
Fig. 14 (McCraty et al. 2001, p.18).....	34
Fig. 15 (Malliani et a. 1991, p.487).....	35
Fig. 16 Measures of time domain and frequency domain indices before and after 7 Osteopathic treatments	44
Fig. 17 Measures in SD1 [ms] before and after treatment	45
Fig. 18 Measures in SD1 [ms] before and after treatment	45
Fig. 19 Measures in RMSSD [ms] before and after treatment.....	45
Fig. 20 Measures in RMSSD [ms] before and after treatment.....	45
Fig. 21 Measures in HF [%] before and after treatment.....	46
Fig. 22 Measures in HF [%] before and after treatment.....	46
Fig. 23 Measures of \sum VLF + LF [%} before and after treatment.....	47
Fig. 24 Measures of \sum VLF + LF [%} before and after treatment.....	47
Fig. 25 HRV measures in time domain and frequency domain in four periods from 7.2.07 to 18.4.07.....	48
Fig. 26 Distribution of SD1 [ms] in four periods from 7.2.07 to 18.4.07	49

Fig. 27 Measures in SD1 [ms] before and after treatment 49

Fig. 28 Measures in SD1 [ms] before and after treatment 49

Fig. 29 Distribution of SD1 in four periods from 7.2.07 to 18.4.07 50

Fig. 30 Distribution of RMSSD in four periods from 7.2.07 to 18.4.07 50

Fig. 31 Measures in RMSSD [ms] before and after treatment..... 51

Fig. 32 Measures in RMSSD [ms] before and after treatment..... 51

Fig. 33 Distribution of RMSSD in four periods from 7.2.07 to 18.4. 51

Fig. 34 Distribution of HF in four periods from 7.2.07 to 18.4.07 52

Fig. 35 Measures in HF [%] before and after treatment..... 53

Fig. 36 Measures in HF [%] before and after treatment..... 53

Fig. 37 Distribution of HF in four periods from 7.2.07 to 18.4. 53

Fig. 38 Distribution of \sum VLF+LF in four periods from 7.2.07 to 18.4.07 54

Fig. 39 Measures \sum VLF+LF [%] before and after treatment 54

Fig. 40 Measures in \sum VLF+LF [%] before and after treatment 54

Fig. 41 Distribution of \sum VLF+LF in four periods from 7.2.07 to 18.4.07 55

Fig. 42 personal feeling during training 55

Fig. 43 Intensity of training..... 56

Fig. 44 Duration of training..... 56