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Heart Rate Variability as a Measure of Exercise- and Training Response

A parallel eight weeks experimental training study comparing effects of sprint interval training and continuously moderate intensity training on short- and long-term heart rate variability in moderately fit young women and men

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Abstract

Aims: Heart rate variability (HRV) is proposed to be a measure of cardiac autonomic activity. This study was designed to investigate the short- and long-term HRV before, during and after eight weeks (3x/week) of sprint interval training (SIT), compared to eight weeks (3x/week) of continuously moderate intensity training (CT) in young and healthy women and men, and to examine associations between HRV and response in maximal oxygen consumption (VO_{2max}).

Methods: 22 moderately fit young subjects were divided into the two groups, matched on VO_{2max}, sex and weight. Both groups did three sessions per week, and training were carried out outside. SIT (four males, eight females) did 5-10, 30-seconds near maximal intensity runs in a graded track, with three minutes of rest (passive walking) between bouts. CT (thee males, seven females) ran at 70-80% of their HR_{peak} for 30-60 minutes on each session.

During the training period, subjects did two short-term recordings of HRV, measured at rest in the morning (five minutes of sitting, followed by five minutes of standing). Recordings were done after training days (AT) and after resting days (AR). Pre-, mid- and post training subjects performed a test of VO_{2max} and a 24-hour recording of HRV (long-term). R-R intervals were recorded using a hear rate monitor Polar RS800CX, Polar Electro Oy, Kempele, Finland) and R-R data were analyzed in Hearts software. HRV indices included are time-domain (mean HR, SDNN), frequency-domain (LF, HF and LF/HF ratio) and non linear analysis (Poincaré Plot; SD1 and SD2, α1, APEN). Data are presented as means ± SEM. Short-term data were analyzed for AR and AT in three sub periods (period 1: week 0-2, period 2: week 3-5, period 3: week 6-9). Long-term data were analyzed for the whole 24-hour period and separate for night hours (1-5 a.m.).

Results: In SIT, HRV was significantly lower on AT days compared to AR days both in sitting and standing posture during the first training weeks. These changes were not observed in period 2 and 3. From period 1 to period 3, SIT showed a significant decrease in HRV on AR days in standing posture ($p < 0.05$), and tended to decrease also in sitting posture. In CT, no changes were observed between AT and AR or between periods. We found no increase in 24-hours HRV post training. Both groups increased VO_{2max}; 2.4 ± 0.7 and 2.0 ± 0.9 ml kg^{-1} min^{-1} for SIT and CT respectively ($p <0.05$). We found a significant correlation between changes in short-term HF power, (ln, ms^2) on AR days in sitting posture and training response (ΔVO_{2max}) for both groups together ($r=0.531$, $p=0.011$) and for SIT group alone ($r=0.615$, $p=0.033$).

Conclusion: It can be concluded that during the first weeks of SIT, HRV is lower after training days compared to after resting days. Furthermore, a lower HRV after resting days appears during the last weeks of SIT, indicating a withdrawal in cardiac vagal activity and a training overload. On the contrary, during an eight weeks period of CT, HRV do not change after training days compared to after resting days. Although SIT and CT are efficient to increase VO_{2max}, our data do not support benefits on HRV in moderately fit young women and men. However, HRV can be a valuable tool to evaluate the long-term recovery of and for planning a proper training load in moderately fit subjects.

Keywords: sprint interval training, heart rate variability, HRV recovery, continuously moderate intensity training

Preface

This year has been the hardest, but also most exciting year in my school carrier, and definitely the year I have learned the most in Sports Sciences.

I am so grateful to the subjects who participated in our training study. It was quite time consuming and sometimes physically challenging. You met up for your appointments, and showed an astonishing enthusiasm during test and training sessions. Without you this project and this thesis would have been nothing. Furthermore a great thanks to Sigbjørn Litleskare and Marit Sandvei, my fellow students. We were a hardworking and devoted team planning and accomplishing an experimental intervention study. Thanks also to Line Hårklau, Kristoffer Jensen, Hans Kristian Stadheim and Per Inge Rustad for help and support when needed.

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A special thanks to Mikko Tulppo and Antti Kiviniemi for your hospitality in Oulu and for helping us analyzing the heart rate variability data, and teaching us to understand the different values. I am looking forward to continue our collaboration. Thanks also to Trine Stensrud, Eystein Enoksen and Jan Cabri for making this training study come true.

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Abbreviations

Term	Explanation	Unit
VO_{2max}	Maximal oxygen consumption	$ml \cdot kg^{-1} \cdot min^{-1}$
HR_{peak}	Peak heart rate from VO_{2max} test	$beats \cdot min^{-1}$
R-R interval	Time between R-peaks from ECG	ms
SD	Standard deviation	-
SEM	Standard error of the mean	-
HRV	Heart rate variability	-
SDNN	Standard deviation of all R-R intervals	ms
SD1	Horizontal SD in a HRV scattergram e.g. from Poincaré Plot	ms
SD2	Vertical SD in a HRV scattergram e.g. from Poincaré Plot	ms
HF power	High frequency power	ms^2
LF power	Low frequency power	ms^2
VLF power	Very low frequency	ms^2
ULF power	Ultra low frequency	ms^2
α_1	Fractal scaling exponent	
ApEn	Approximate entropy	
ln	Natural logarithm of the value (0-10)	
ms	Milliseconds	
AT	Morning after training day	
AR	Morning after resting day	
SIT	Sprint interval training	
CT	Continuously moderate intensity training	

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1.0 Introduction

The cardiovascular system is controlled by the two divisions in the autonomic nervous system; the parasympathetic and sympathetic system (Hall & Guyton, 2011). These two divisions influence the cardiovascular system through a quite complex interplay, in which is not fully understood. Changes in heart rate (HR), blood pressure and peripheral vascular tone in response to internal and external demands are all regulated by the two divisions (Hall & Guyton, 2011).

The measurement of the oscillations in HR, termed heart rate variability (HRV) provides a non-invasive tool for assessing cardiac autonomic control (Aubert, Seps, & Beckers, 2003; Berntson et al., 1997). HRV describes the variations in time-intervals between beats and are calculated from the R-wave to R-wave from an electrocardiogram (ECG). Studies on HR and HRV have attracted an increasing interest because high parasympathetic HRV indices have been related to low cardiovascular risks (Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology (1996)). Furthermore, from a clinical point of view HRV has received a lot of attention because abnormalities in HRV after myocardial infarction are associated with increased risk for death (Huikuri et al., 2000; Kleiger, Miller, Bigger, Jr., & Moss, 1987)

During recent decades many studies have reported changes in HRV in response to endurance exercise and endurance training. Technological development has made it possible to make reliable R-R interval recordings using commercial HR monitors, which can expand the research field of autonomic regulation of HR dynamics (Achten & Jeukendrup, 2003)

It is assumed that endurance training may cause a shift towards increased parasympathetic activity (e.g. vagal). Most studies have reported that endurance training increases cardiac vagal control at rest, which also can be a part of the explanation of a lower resting HR in trained subjects (Tulppo et al., 2003; al-Ani, Munir, White, Townend, & Coote, 1996). However, there are contradictory findings and the training induced increase in HRV has not been observed systematically (Verheyden, Eijnde, Beckers, Vanhees, & Aubert, 2006; Aubert et al., 2003; Catai et al., 2002; Perini, Fisher, Veicsteinas, &

Pendergast, 2002). This can be due to different HRV methods, different training protocols or small populations (Borresen & Lambert, 2008). HRV can be measured in several ways, including short-term resting measurements in different body postures, during exercise, during exercise recovery, and during daily living (recordings for 24 hours or more).

HRV recovery after single bouts of endurance exercise has been investigated by several researches (Kaikkonen, Rusko, & Martinmaki, 2008; Martinmaki & Rusko, 2008; Kaikkonen, Nummela, & Rusko, 2007). During the recovery phase, HR and HRV returns to baseline values, that shows the time-dependency of the intensity and dur ation of the exercise bout (Borresen & Lambert, 2008).

Few studies have investigated the effects of one ex ercise bout on H RV recovery the following night or day. Recently, Hynynen, Vesterinen, Rusko & Nummela (2010) found that HRV was lower the night after short-duration moderate intensity exercise and the night after marathon compared to a night after rest.

In order to optimize good training programs and to evaluate the short- and long-term effects in health and per formance, it is suggested that HRV can be a practical tool for both athletes and s edentary people (Kiviniemi et al., 2010; Kiviniemi, Hautala, Kinnunen, & Tulppo, 2007; Mourot et al., 2004). It has also been suggested that HRV measured before training shows positive correlation to training response in cardiorespiratory fitness in sedentary subjects (Hautala et al., 2003).

Today, sprint interval training has become a popular research theme, and it has been proved that this type of short duration (~30 seconds) supra high intensity (all-out) bouts can induce the same effects as continuously moderate intensity training on performance parameters (Burgomaster et al., 2008; Burgomaster, Heigenhauser, & Gibala, 2006; Gibala et al., 2006).

In relation to HRV, Millar et al. (2009) studied the HRV recovery after one and four Wingate tests (30 seconds all-out on a c ycle ergometer), showing that multiple Wingate tests resulted in prolonged alterations in HRV.

To date, no study has identified the effects of sprint interval training on HRV during an experimental training intervention.

1.1 Aims of the study

The aim of the present study was to compare the effects of sprint interval training (SIT) and continuously moderate intensity training (CT) on short- and long-term (24-hours) HRV before, during and after an eight weeks training period with three sessions per week in moderately fit subjects.

This proposes three main purposes:

1. To compare and follow short-term HRV at rest (five minutes of sitting, five minutes of standing) in mornings after training (AT) and mornings after resting days (AR) during the eight weeks training period for SIT and CT.
2. To examine the effects of SIT and CT on 24-hours HRV
3. To assess the association between response in maximal oxygen consumption (VO_{2max}) and
   a) baseline HRV
   b) HRV response

We hypothesized that SIT would show a lower HRV on AT days compared to AR days, and that both groups would increase short- and long-term HRV after eight weeks of training with three sessions per week.

2.0 Theory

2.1 History of heart rate variability (HRV)

Over the last 30 years there has been an increasing interest in studying the oscillitations of HR calculated from the ECG. (R-R interval) These oscilliations in R-R interval are termed heart rate variability (HRV).

HR has a periodic cardiovascular signal in the same way as blood pressure. The fluctuations in these signals have interested physicians for generations. In 1778, Albrecht von Haller investigated the beat of a healthy heart. He found that these beats were not absolutely regular, but they were in synchrony with respiration (found in - Peltola, 2010). The variation of HR followed by inspiration and respiration were observed in a dog by Carl Ludwig in 1847. This alteration of the HR during the respiratory cycle is today known as respiratory sinus arrhythmia (Task Force of the ESC and NASP, 1996).

Since then, simple HR measurements have been investigated. It was not until the latter century that research on HRV expanded. The development in technology has invigorated the research field of cardiovascular signals. In the 1970s, Ewing and colleagues reported R-R interval changes on various bedside tests in diabetic patients. This was in order to detect autonomic neuropathy (Ewing, Borsey, Bellavere, & Clarke, 1981; Ewing, Campbell, Murray, Neilson, & Clarke, 1978).

During the 1980s, a power spectral analysis of the HRV was introduced (Akselrod et al., 1981). In the same period, the clinical relevance of HRV became clear. It was confirmed that a decline in HRV increased the risk for arrhythmic events and mortality in patients with myocardial infarction (Task Force of the ESC and NASP, 1996).

HRV analysis has been used to assess autonomic function in various types of cardiac diseases, including myocardial infarction, congestive heart failure, coronary heart disease, and essential hypertension. In addition diabetes mellitus are associated with a reduced vagal tone and an elevated sympathetic activity (Task Force of the ESC and NASP, 1996).

2.2 Physiological background

The heart and the cardiovascular system is mainly controlled by the autonomic nervous system that aims to regulate blood pressure via cardiac output and peripheral vascular tone in response to internal and external demands. Change of body posture and physical activity can be such external demands. This section will describe the autonomic regulation of HR and HRV.

The probably most striking characteristics of the autonomic nervous system are the rapidity and intensity in which it can change visceral functions. For instance, within 3 -5 seconds it can increase the HR to twice normal (Hall & Guyton, 2011). Other functions, like sweating, can also begin within seconds and the same applies for involuntary emptying of the urinary bladder. The two main components of the autonomic nervous system are the parasympathetic and sympathetic system. Figure 2-1 depicts the general organization of the peripheral innervations of the two divisions, adapted from Hall and Guyton (2011).

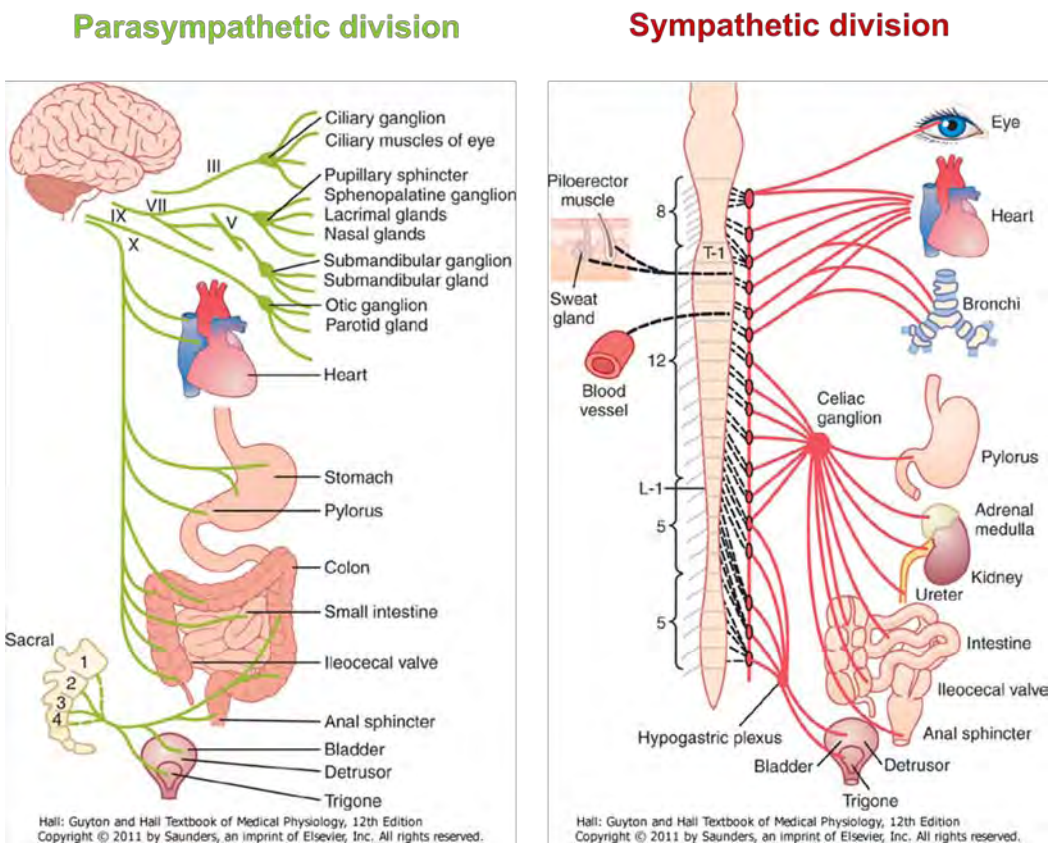


Figure 2-1: An overview of bodily functions controlled by the parasympathetic (left) and sympathetic nervous system (right). Adapted from Hall & Guyton (2011)

2.2.1 Autonomic regulation of HR and HRV

Normal heart rhythm is defined by the rate of sinus node depolarization. This rate is regulated by the parasympathetic and sympathetic divisions that may operate in parallel, but act by different structural pathways and transmitter systems (Hall & Guyton, 2011). The autonomic nervous system is mainly activated by centers located in the spinal cord, brain stem, and hypothalamus. Briefly, the sympathetic nervous system increases HR and contractility of the heart muscle, whereas parasympathetic activity induces the opposite effects.

2.2.1.1 Parasympathetic influence

The vagus nerves are responsible for the parasympathetic control of the heart. About 75% of all parasympathetic nerve fibers are in the vagus nerves; the tenth cranial nerve (see figure 2-1). The vagus nerves are the only cranial nerves that exit the head and neck region and reach their destination in thorax and abdominal regions. That is why the term “vagal” often is used instead of parasympathetic in relation to HRV (also in the present thesis). Parasympathetic fibers from the vagus nerves innervates the sinoartrial (SA) node, the atrioventricular (AV) pathways and the atrial muscle (McArdle, Katch, & Katch, 2007). The parasympathetic system has both preganglionic and postganglionic neurons. The preganglionic neurons pass mostly interrupted all the way to the organ that is to be controlled and synapses with the postganglionic fibers, which are located in the wall of the organ (Hall & Guyton, 2011). The vagal preganglionic neurons originate from the dorsal motor nucleus and nucleus ambiguous in the medulla oblongata.

The parasympathetic nerve fibers use acetylcholine as a neurotransmitter. Acetylcholine stimulates the postganglionic neurons which slows the rate of the SA node depolarization, which results in reduced HR. Those fibers that secrete acetylcholine are said to be cholinergic, having choline as primary component in acethylcoline. In addition to the parasympathetic stimulated decrease in HR, vagal stimulation can decrease the strength of heart contraction by 20-30%. This is slight decrease, and it is due to the vagal fibers distribution to the atria, rather than the ventricles. Vagal stimulation allows the heart to “rest”, for instance between bouts of strenuous activity, or during sleep.

2.2.1.2 Sympathetic influence

Sympathetic nerve fibers innervate the whole heart, including the SA node, AV pathways and the arterial and v entricular myocardium. The sympathetic stimulation causes opposite effects compared to the parasympathetic stimulation, and i ncreases the overall activity of the heart. The sympathetic preganglionic neurons originate from the intermediolateral cells of the spinal cord (Hall & Guyton, 2011). The neurons differ from the parasympathetic neurons by having much shorter myelinated preganglionic neurons, and much longer unmyelinated postganglionic neurons. This is one reason explaining the slower effect of the sympathetic activity to the heart compared to vagal activity.

Sympathetic nerve fibers secrete mainly noradrenalin. Those fibers that secrete noradrenalin are said to be *adrenergic*. This term is derived from adrenalin (Hall & Guyton, 2011). The adrenergic receptors are situated in the target cells, ready to mediate effects. These receptors are known as α- and β-receptors. In heart cells there are mainly β-receptors (β_1). D uring sympathetic stimulation of the heart, the effects are primarily due to noradrenalin. Noradrenalin speeds up the rhythm of the SA node, leading to an al teration in the activity of the hearts` electrical impulse system, showing increased speed of the impulse. Strong sympathetic stimulation can increase can increase HR up to 200 beats/min, and rarely, even 250 beat s/min in young humans (Hall & Guyton, 2011). Furthermore, sympathetic excitation on the heart can increase the force of heart contraction up to as much as double of the normal value. As a result of this, an increase in cardiac output is observed, and dur ing maximal sympathetic stimulation cardiac output can increase up to a threefold (Hall & Guyton, 2011).

The sympathetic system is strongly activated during many emotional states. The sympathetic stress response refers to the activation of the sympathetic system due to mental or physical stress. Increased HR and blood pressure are indicators of dominance in sympathetic activity. When the sympathetic nervous system is activated, noradrenalin is released from the effector cells, and at the same time adrenalin is released to the blood as a r esult of the noradrenergic stimulation of the adrenal cortex. This typically happens in situations having huge mental or physical stress, such as in front of a school presentation or during a fast run for the bus.

2.2.2 Reflexes and factors affecting autonomic control of HR and HRV

2.2.2.1 Respiration

During inspiration, HR increases and during expiration it decreases. This is mediated by reflexive changes in the vagal activity to the SA node. Cyclical changes in HR appear in association with respiration in normal subjects, introduced previously as respiratory sinus arrhythmia (RSA). The influence of respiration on HR is complex and involves central and reflex interactions (Hall & Guyton, 2011). It has been suggested that intrathoracic pressure changes during respiratory cycle influence the left ventricular stroke volume by affecting the cardiac venous return. An alternate explanation proposes that the baroreflex-mediated firing of the muscle-sympathetic nerve is phased by respiration. Another theory proposes the meaning of the central oscillitations in autonomic activity, associated to respiratory motorneuron activity (Eckberg, 2003)

The function of the respiratory-related cardiovascular fluctuation is to increase pulmonary gas exchange activity, by increasing pulmonary circulation during inspiration (Hayano, Yasuma, Okada, Mukai, & Fujinami, 1996). RSA can be abolished by atropine or vagotomy and in that manner one can exclude the influence of the parasympathetic regulation (Hayano et al., 1996; Akselrod et al., 1985).

2.2.2.2 Blood pressure fluctuation

The baroreflex is the mechanism involved in the maintenance of the blood pressure. This reflex involves blood pressure monitoring with stretch sensitive mechanoreceptors. Baroreceptors are situated in the walls of some arteries (including carotid sinus, aortic arch and coronary arteries) and react to the stretch caused by an increase in blood pressure in order to maintain homeostasis (Hall & Guyton, 2011). Increased baroreceptor firing causes an increased cardiac vagal outflow, which decelerates HR and the force of heart contraction. The opposite happens when blood pressure decreases, showing a decrease in vagal activity, and thereby an increase in HR and cardiac contractility. To explain this, it is easy to compare different body postures and observe the changes. When rising up from supine to standing the arterial blood

pressure falls and fewer impulses are sent to the vasomotor center. As a consequence vagal activity decreases and sympathetic activity increases, resulting in an increase in acceleration in both HR and arterial blood pressure. The baroreflex has been proposed as being a vagally mediated control system between the HR and blood pressure. There is claimed a sympathetic baroreflex as well, seen as an increase in vascular tone via increased sympathetic activity to arteries when blood pressure decreases (O'Leary, Kimmerly, Cechetto, & Shoemaker, 2003).

2.2.2.3 Other factors

HRV has been reported being dependent on age and gender . Aging itself reduces HRV, documented by several studies showing a decline in HRV with increasing age (Achten & Jeukendrup, 2003) This age-related changes seems to be modified by gender (Pikkujamsa, Makikallio, Airaksinen, & Huikuri, 2001; Jensen-Urstad et al., 1997). Women tend to have higher levels of HRV compared to men, although these differences seem to disappear during and after the fifth decade of life (Ramaekers, Ector, Aubert, Rubens, & Van de Werf, 1998; Umetani, Singer, McCraty, & Atkinson, 1998; Jensen-Urstad et al., 1997). However, conclusions must be interpreted carefully, due to different age range being studies as well as the analysis methods and conditions used in the quantification of HRV (Aubert et al., 2003).

In addition, chemoreceptors, the renin-angiotensin system, the control of blood volume, and the thermoregulation system may affect the cardiovascular system and autonomic control (Achten & Jeukendrup, 2003).

2.2.3 Measuring autonomic activity

HR is regulated by a complex set of interactions between parasympathetic and sympathetic control. The effects of these two divisions can vary independently or coactively (Berntson, Cacioppo, & Quigley, 1993). Because the two divisions have opposite effects on the control of the heart, HR may increase due to a vagal withdrawal, sympathetic activation or both. To investigate this relation the use of vagal blockade has been introduced. The influence of cardiac vagal effects can be seen immediately, within milliseconds, whereas the sympathetic influence can be seen in a few seconds.

In the research field of HRV, specific drugs have been used to identify parasympathetic (and sympathetic) activity. One of the first drugs being examined in relation to HRV was atropine (Akselrod et al., 1985). Atropine is a competitive antagonist for the muscarinic acetylcholine receptor. It is classified as an anticholinergic drug. The effects of beta-blockers and calcium channel blockers have been examined in both heart patients (postinfarction and hypertensive patients) and healthy subjects (Taylor, Myers, Halliwill, Seidel, & Eckberg, 2001). By analyzing HRV in a frequency domain it is possible to observe the autonomic activities of these drugs (parasympathetic and sympathetic) and their effects on cardiovascular diseases. Studies have also been performed with antiarrhytmic agents, anesthetics, narcotics, sedatives and chemotherapeutic agents (Task Force of the ESC and NASP, 1996)

The use of pharmacological blockade to assess the parasympathetic and sympathetic contributions at rest, have also been used during exercise at different intensities. During onset of exercise it has been suggested that the increase in heart rate is due to parasympathetic withdrawal (Tulppo, Makikallio, Takala, Seppanen, & Huikuri, 1996). From submaximal to more vigorous levels and during maximal intensity, it is proposed that the sympathetic system is activated and dominating (Borresen & Lambert, 2008).

2.3 The methodological basis of HRV

2.3.1 Assessing the HRV signal

To obtain a HRV signal, high quality ECG data are needed. Traditionally ECG recordings are done with a Holter monitor. Nowadays, these are portable and recordings can be done out side the hospital. A portable Holter monitor is an ambulatory device which can monitor the electrical activity of the heart and record the heart rhythm continuously over periods of 24 hours or more (McArdle et al., 2007). The Holter monitor is connected to the subject via a series of wired non-invasive electrodes on the chest. The number of electrodes usually varies from three to eight, and depends on the model of the Holter monitor. These electrodes detect the heart rhythm. QRS complexes are identified from the ECG recording (figure 2-3). The peak of the R-wave is used as a marker, because it normally has distinguishable amplitude. R-peak detection requires a robust algorithm. The more accurate the R-peak detection is, the less error there will be in HRV signal an analysis. Measurement of HRV usually requires high-quality ECG with a sampling rate at least in the range of 250-500Hz (Task Force of the ESC and NASP, 1996). This corresponds to a resolution of ≤ 4 ms. Nowadays, modern Holter monitors are usually using 1000Hz (Aubert et al., 2003). Low sampling frequency may cause dispersion in the R-peak estimation.

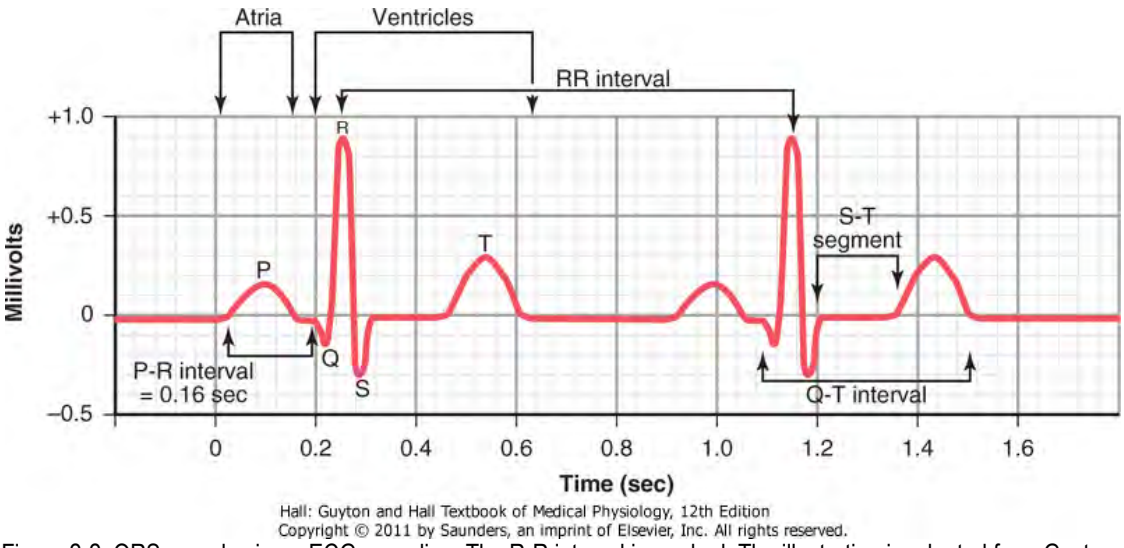


Figure 2-2: QRS-complex in an ECG recording. The R-R interval is marked. The illustration is adapted from Guyton and Hall (2011).

The development of wireless HR monitoring with elastic electrode belts represents an upcoming alternative to classic fixed or ambulatory ECG

recordings for use in medicine- and sports sciences (Achten & Jeukendrup, 2003). HR monitors normally consist of an electrode belt, a transmitter and a receiver (usually shaped as an armband). The electrode belt allows a detection of R-R intervals with a resolution of 1 ms and a sampling frequency of 1000Hz (Ruha, Sallinen, & Nissila, 1997). Gamelin, Berthoin and Bosquet (2006) did a validation study on the Polar s810 and showed a similar HRV from ECG and electrode belt in supine position. In standing position they found a lower HRV from electrode belt compared to ECG. The use of the same device during HRV studies may help to avoid prospective differences (Gamelin, Berthoin, & Bosquet, 2006).

2.3.2 Editing the HRV signal

The R-R interval from the ECG recording is generated in times series, and this is also termed HRV signal or R-R interval tachogram that is discrete series of events due to the differences between the duration of two following heart beats. This time between beats is expressed in milliseconds (ms).

R-R interval time series are in most cases not perfect. They contain a different number of abnormal R-R intervals and are often termed artifacts. These intervals differ from the sinus rhythm in length and represent both technical and physiological disturbance. Artifacts can be avoided by having steady state conditions in a laboratory for a short duration, at least in healthy subjects. However, HRV are not only investigated in these conditions, and during ECG recordings lasting for several hours, it is virtually impossible to obtain a clean recording.

There are different technical artifacts affecting the HRV signal. Problems may appear when electrodes are poorly fastened, from motion or from sweat. It can also appear noise in the software/monitor used to detect the R-R interval algorithm, resulting in a threshold failure for R-R interval identification. When looking at tachograms for long-term recordings, technical artifacts usually occur in larger epochs containing several consecutive abnormal R-R intervals.

The Task Force of ESC and NASP (1996) has recommended that one should use HRV without ectopic beats and other noise. However, due to the growing

interest of HRV and the possibility of doing field measurements, efficient and reliable editing methods are needed. Several commercial, automatic artifact correction programs have been introduced. These have different levels of accuracy, leading to differences in HRV analysis. Hence, Task Force recommends manual editing with visual inspection by a specialist. There are two main different approaches to this problem. The first one is fast and simple, and the main idea is to delete artifacts. Deletion method decreases the length of the HRV signal. The other editing method is replacement (e.g. interpolation), where the abnormal R-R intervals are replaced with new interpolated R-R intervals. The effects of editing on HRV can be remarkable in manner of different HRV indices, and should be taken into account when interpreting results (Salo, Huikuri, & Seppanen, 2001).

2.3.3 Analysis methods of HRV

HRV analysis methods can be categorized as time-domain methods, frequency-domain methods, geometric methods and non-linear methods. In addition, HR-turbulence and baroreflex sensitivity can be used in HRV analysis (Loimaala, Huikuri, Oja, Pasanen, & Vuori, 2000; Schmidt et al., 1999). In this thesis the principal categories are described shortly, and the mathematical processing of the HRV signal is out of the scope for this thesis. For detailed information, please see Task Force of ESC and NASP (1996) or Kubios HRV user guide (Niskanen & Tarvainen, 2008, downloaded from http://kubios.uku.fi).

2.3.3.1 Time-domain analysis

The parameters in the time-domain analysis are easily computed using simple statistical methods. The time-domain expresses HRV in time units (ms). The simplest index to compute is the standard deviation of the R-R intervals (SDNN) over the time interval selected from a recording (standard deviation of all normal to normal R-R intervals). The index represents overall HRV and describes fluctuations at all frequencies during the period of recording. The magnitude of the SDNN depends on the duration of the recording, but it can normally be used in short recordings. However, it is recommended that comparisons should be made between segments of similar length. A normal value of SDNN in healthy subjects identified from 24-hours recordings is suggested to be ~141 ± 39 (Task

Force of the ESC and NASP, 1996), whereas values below 80 are found in patients with acute myocardial infarction (Peltola, 2010). Other time-domain methods are described in Task Force of ECS and NASP (1996).

2.3.3.2 Frequency-domain analysis

The classical time-domain indices cannot describe the autonomic balance, which can be assessed by the spectral analysis of HRV. As the power of high frequency (HF, 0.15-0.4 Hz) HR osciliations mainly reflects the parasympathetic nerve activity, the power of low frequency (LF, 0.04-0.15 Hz) HR osciliations represents both cardiac sympathetic and parasympathetic nerve activity.

The power spectral methods for HRV analysis have been applied to get information on the distribution of the power as a function of frequency. In order to provide this information, R-R interval time series have to be decomposed into quantified frequency components or integrated over a defined frequency band. The power spectrum estimations are provided by using a transformation method. The most commonly used are Fast Fourier Transformation (FFT) and parametric power spectrum estimation based on autoregressive modeling (AR). For details about these transformation methods, please see Task Force (1996) or Kubios userguide (Niskanen & Tarvainen, 2008).

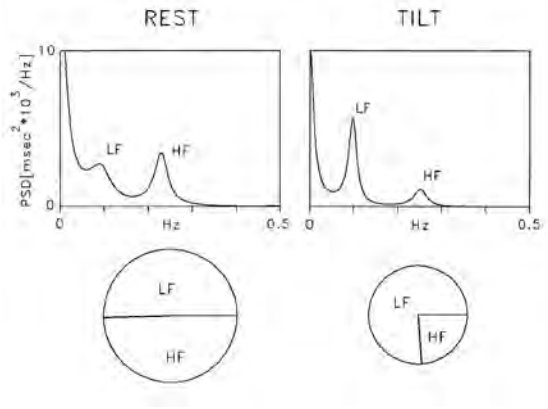
The power spectrum contains three main frequency bands; very low frequency (VLF), low frequency (LF) and high frequency (HF). In figure 2-4, you can see a power spectrum for healthy subject during rest and tilt.

The LF component is created in heart rhythm that is normally observed around 0.1 Hz. This slow oscilliation has been suggested as being related to mechanisms regulating blood pressure. However, the physiological understanding of the LF component is still controversial. Both parasympathetic and sympathetic divisions are involved in the LF rhythms. An increase in LF power has been proposed as being a marker for sympathetic activation (especially when expressed normalized to HF), but it is not fully accepted (Eckberg, 1997).

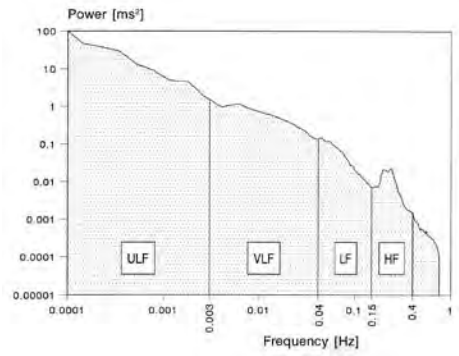
The HF component is usually identified in the frequency range between 0.15Hz and 0.4Hz. This has been seen in relation to respiration. Respiration related HF

power is affected by the intrathoracic pressure and mechanical changes caused by breathing activity. This is mediated by the vagus nerve, and is thought to be a marker of parasympathetic activation (Eckberg, 2003).

The LF/HF is suggested to be a marker of sympathovagal balance (Malliani, Pagani, Lombardi, & Cerutti, 1991). In figure 2-4 you can see the distribution of the two components during two different body postures, suggesting a higher sympathetic activity during tilt and thereby a higher ratio. A low LF/HF ratio measured at rest is considered healthy, and can be seen in relation to a balanced homeostasis and non-stressed condition.



2-4



2-5

Figure 2-4: HRV power spectrum from a healthy subject during rest (supine?) and during tilt (degrees?). The sector diagram shows the relative distribution of LF and HF component during the two positions. Adapted from Task Force (Task Force of the ESC and NASP, 1996).

Figure 2.5: HRV power spectrum showing the different frequency bands of the spectral components. Adatpted from Task Force (Task Force of the ESC and NASP, 1996)

The VLF component includes long period HR rhythms. The origin of these fluctuations are suggested to be affected by thermoregulation systems, the renin-angiotensin system and perhaps humoral factors (Peltola, 2010). However, this is far not understood in detail, and not essential for this thesis. Furthermore the ultra low frequency (ULF) component can be determined from the power spectrum, but it is still only speculations regarding the physiological mechanisms (Peltola, 2010)

2.3.2.3 Non-linear analysis

During the last couple of decades it has been an increasing interest in the HRV analysis based on the chaos theory and non-linear dynamics. The basic concept of the non-linear HRV methods is to try to provide the non-periodic

behavior of the HRV signal, and to capture the complexity in the R-R interval dynamics. There are several non-linear methods, including return maps, fractal scaling analysis and entropy measures. These have been tested in various sets of R-R interval data (Peltola, 2010).

The most common non-linear method is the return-map method named Poincaré Plot. It can be discussed why this is categorized as a qualitative non-linear method and not time-domain. A Poincaré Plot is a gr aphical representation of the correlation between successive R-R intervals. It is generated by plotting each R-R interval as a function of its previous R-R interval (Niskanen & Tarvainen, 2008). The standard descriptors of the Poincaré Plot are SD1 and SD2. Figure 2-6 exemplifies these plots. The line of identity is the 45 degree diagonal line on the plot. SD1 measures the dispersion of the data points perpendicular to the line of identity, whereas SD2 measures the dispersion of the data points *along* the line of identity. SD1 and S D2 are basically standard deviation measures (Tulppo et al., 1996). SD1 represents the instantaneous beat-to-beat variability, while SD2 represents the continuous long-term beat-to-beat variability (Niskanen & Tarvainen, 2008; Tulppo et al., 1996; Huikuri et al., 1996)

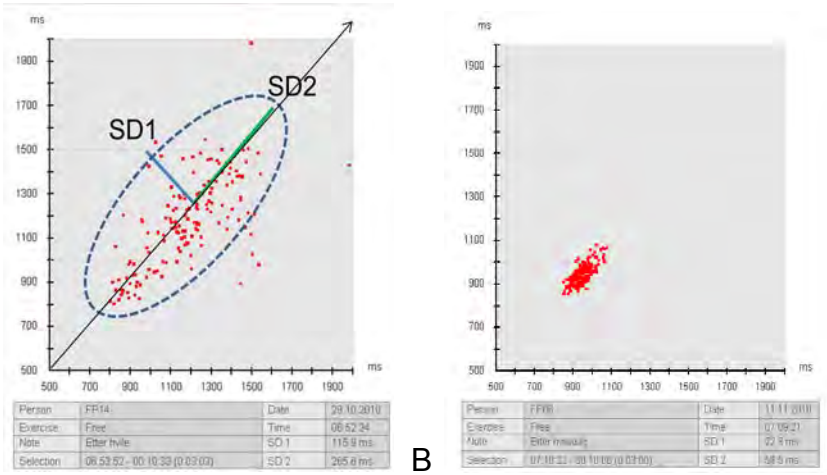


Figure 2-6: Two Poincare plots from Polar Pro Trainer software from short-term recordings in sitting posture. SD1 and SD2 are marked on the left. The two plots also illustrate huge differences between subjects.

In 1983, Mandelbrot introduced the term “fractal” (found in -Peltola, 2010) A fractal describes a set of points that resembles the whole set even when examined at very small scales. The fractal properties quantification assesses the self-similarity of the HR oscilliation over multiple time scales. This can be

examined by detrended fluctuation analysis (DFA) technique (Peng, Havlin, Stanley, & Goldberger, 1995). DFA quantifies the fractal correlation properties of the R-R intervals. The root-mean-square fluctuations of the integrated and detrended data are measured in observation windows of different sizes. Furthermore the fluctuations are plotted against the size of the window on a log-log scale. The scaling exponent (α_1) represents the slope of this line. The scaling exponent differs between uncorrelated and correlated data. α1 represents the "roughness" or randomness of the time series, and large values indicate a smooth time series. Values of the scaling exponent for a normal healty subject are typically near 1. A more detailed description of DFA computation can be found in Goldberger (1996; 1992) or Peng et al. (1995).

Approximate entropy (ApEn) measures the complexity or irregularity of the signal. ApEn quantifies the likelihood than runs in patterns that are close, will remain close in the next incremental comparison (Peltola, 2010; Tulppo et al., 1996). Large values of ApEn indicate large irregularity whereas smaller values indicate a more regular signal.

2.4 HRV response to endurance exercise and endurance training

It is well known that endurance training may decrease HR at rest and during submaximal exercise (Borresen & Lambert, 2008). Maximal HR may decrease slightly after training or remain unchanged. The effect of endurance training on HRV remains partly inconclusive. This it probably due to different designs and methodological approaches (Borresen & Lambert, 2008; Aubert et al., 2003), and it`s recommended that training schedule (including intensity and duration) and population are properly and fully described. Furthermore it is strongly suggested than, when presenting HRV data related to exercise physiology, a detailed description on analysis methods should be presented. Most studies have a small number of participants, diminishing the power of the statistics (Aubert et al., 2003). In this section selected studies are presented, delimited to the support the aims of the present study.

2.4.1 HRV response to one bout of endurance exercise

The cardiovascular system responds rapidly to endurance exercise. The acute changes during a bout of exercise represent an integration of neural, hormonal and mechanical mechanisms (McArdle et al., 2007). As mentioned earlier the autonomic nervous system plays an important role in making fast adjustments in the cardiovascular system. Information from baroreceptors and receptors originating in the skeletal contracting muscles is integrated in the central nervous system. Furthermore an appropriate output to the peripheral organs is provided. Studies using pharmacological blockade has managed to identify the influence from the two different divisions. At the onset of endurance exercise, a rapid increase in HR is observed. This increase is mostly due to a reduction in vagal activity (Tulppo, Makikallio, Seppanen, Laukkanen, & Huikuri, 1998; Tulppo et al., 1996). Further increase in intensity provides further decrease in vagal activity, but also a concomitant increase in sympathetic activity. So, the relative role of the sympathetic activation increases with exercise intensity. During maximal exercise intensity, little or no vagal activity can be detected (Tulppo et al., 1996; Robinson, Epstein, Beiser, & Braunwald, 1966) Studies on HRV during exercise include both constant load exercise sessions and incremental exercise (Tulppo et al., 1998; Tulppo et al., 1996; Nakamura, Yamamoto, & Muraoka, 1993; Yamamoto, Hughson, & Peterson, 1991).

Immediate responses in HR and HRV after exercise have also been investigated, in order to evaluate the recovery of the body (Niemela et al., 2008; Borresen & Lambert, 2008). In 1982, Savin et al. suggested that the decline of HR after maximal exercise is an intrinsic property of the intact circulation. Furthermore they claimed that the time-course of the decline is dependent on autonomic activity (Savin, Davidson, & Haskell, 1982).

The HRV recovery has been of great interest during the last years. The recovery phase is often split in two phases, concerning the time points after the exercise bout, having an immediate- and a prolonged phase. The main focus of measuring HRV after exercise has been to evaluate whether any HRV parameter measured at one or more time points differs from the pre-exercise baseline value. In general, HRV recovery is affected by exercise intensity and

duration. Dependent on these two factors, exercise-reduced HRV has been reported to gradually return to baseline level within short time (~1 hour) (Borresen & Lambert, 2008). However, a short delay in HRV recovery has been observed following moderate- to high intensity exercise (Kaikkonen, Hynynen, Mann, Rusko, & Nummela, 2010; Kaikkonen et al., 2008; Martinmaki & Rusko, 2008; Seiler, Haugen, & Kuffel, 2007). Yet, few studies have investigated the more long-term effect of prolonged and intensive exercise bouts.

In 2001, Hautala and collegaues studied the HRV recovery the night after a 75km ski-race. They observed a decrease in HRV (HF power) compared to the night before the ski-race, showing an alteration of the vagal activity to the heart (Hautala et al., 2001). Recently, Hynynen et al. (2010) compared nocturnal HRV after both moderate intensity exercise (52 ± 26 min, 72% of HR_{max}) and marathon, compared to night after resting day in physically active men (VO_{2max} 52.5). They found an increased HR and a decreased HRV after both the marathon and moderate endurance exercise compared to control night (Hynynen, Vesterinen, Rusko, & Nummela, 2010). After marathon the HRV indices were significantly lower compared to the night after moderate exercise ($p < 0.01$). The most marked decrease in HRV was the absolute HF power, showing these values: 1214 ± 920, 952 ± 754 and 4 45 ± 612 (ms^2) for rest, moderate exercise, and marathon respectively. By showing a reduction in HRV of ~66%, authors emphasizes that the use of HRV seems to detect exercise-induced changes in autonomic modulation better than the sensitivity of HR (Hynynen et al., 2010)

2.4.2 HRV response to endurance training

General cardiovascular adaptations to endurance training are well-documented. It`s suggested that about a half of the increase in VO_{2max} caused by endurance training is due to increased cardiac output. The other half is due to the increase in arteriovenous oxygen difference (Levine, 2008). The increase in cardiac output is mostly attributable to an increase in stroke volume , which gives a lower HR at rest and during submaximal exercise intensity (McArdle et al., 2007) . Furthermore endurance training induces a small decrease or no change in HR_{max} (Borresen & Lambert, 2008). All these training induced changes in the

cardiovascular- and cardiorespiratory system are in relation to powerful regulation of the autonomic nervous system (Borresen & Lambert, 2008; Aubert et al., 2003). Training induced changes on autonomic cardiac function depends on many factors, such as duration, intensity and frequency of training (Aubert et al., 2003). Findings from cross-sectional studies indicate a higher HRV in trained subjects compared to untrained subjects (Borresen & Lambert, 2008; Aubert et al., 2003), most marked in endurance trained subjects (Aubert, Beckers, & Ramaekers, 2001).

In longitudinal studies, it has generally been hypothesized that a training-induced decrease in HR is accompanied by an increase in HRV (Carter, Banister, & Blaber, 2003; Tulppo et al., 2003) In table 2-1, intervention studies (from year 2000) are listed; inspired and modified from the reviews of Aubert et al., (2003) and Borresen & Lambert (2008).

In 1989, one of the first training studies was published, written by Seals and Chase. They recruited sedentary middle-aged and older men and divided them in a training group and a control group. The training group exercised three-four times per week, doing walking or jogging for thirty minutes on each session. The intervention period was 30-38 weeks (Seals & Chase, 1989). They found an increase in VO_{2max} and a small, but significant decrease in resting HR for the training group. Furthermore they found an increase in HRV and concluded that regular physical exercise may increase cardiac vagal tone at rest.

This finding has also been confirmed in later studies, adding measures of HRV. Melanson and Freedson (2001) showed that 12 weeks of moderate- to vigorous intensity endurance training (30 minutes, three days a week) increased markers of parasympathetic activity. This was proven by a significant increase in HF power (Melanson & Freedson, 2001). An increase in HF power have been confirmed in more studies (Pichot et al., 2005; Carter et al., 2003; Leicht, Allen, & Hoey, 2003; Tulppo et al., 2003; Hautala et al., 2003), but have reported no alteration in HF power (Verheyden et al., 2006; Carter et al., 2003; Perini et al., 2002; Loimaala et al., 2000; Stein, Ehsani, Domitrovich, Kleiger, & Rottman, 1999) As mentioned earlier, different age groups are used, and also different training protocols.

2.4.2.1 Effects of gender and age

Most early studies on the relationship between HRV and endurance training are done on men. I 2003, Carter and colleagues studied the effect of age and gender on HRV after 12 weeks of endurance training. 24 subjects were divided into four age and gender groups (20F, 20M, 40F, 40M). HRV was collected in supine position and during submaximal cycling before and after the training period. The training program was briefly planned, and it consisted of two blocks, in which the last four weeks consisted of a 20% increase in the duration of the training sessions (Carter et al., 2003). All groups improved a 2-mile criterion performance, but the young group had a larger increase compared to the old group. Training, age and gender interactions indicated that there was decrease in HR for the young groups (20F, 20M) and the old male group. HR was unaltered for the old female group. For the HRV indices a total group mean increase was observed in SDNN, total power and HF power. The young group had a larger increase in total- and HF power. The results of the study indicate that younger subjects had a larger autonomic adjustment to training compared to older subjects. In addition, their data also showed that 40 year old women received small autonomic adaptation. The authors pointed out the small subgroup sample size as a limitation regarding quantification of age and gender effects (Carter et al., 2003).

2.4.2.2 Long-lasting intervention studies

The majority of training studies lasts for 8-12 weeks, without follow ups. In 2000, Loimala et al. published the results from their five months controlled aerobic training study in middle-aged sedentary/moderately fit men (35-55 years). They compared the effects of low- and high intensity training on HR, HRV and baroreflex sensitivity (24-hours measurements). The low intensity group was instructed to do jogging or walking 4-6 times per week at a HR corresponding to 55% of the VO_{2max} measured at baseline. The high intensity group performed jogging 4-6 times per week at a HR level corresponding to 75% of their baseline VO_{2max}. One session each week were supervised. A decrease in resting HR (24-hours period) for the high intensity training group was proved (- 4 beats). Despite this finding there were no changes in HRV or baroreflex sensitivity for the 24-hours measurement or separate for sleeping hours (Loimaala et al.,

2000). In this study all groups improved VO_{2max}, but the high intensity group improved more than the control group. The same for endurance time, in which also was significantly higher in the low intensity training group compared to controls.

In 2004, Hautala et al. published a follow up study of a controlled training program. After eight weeks of training (Hautala et al., 2003), the participants underwent a 10-months home-based training program in accordance to the recommendations of the American College of Sports Medicine. HRV was assessed from 24-hours recordings, and analysis were done for the whole period and separate for night hours. HF power at night remained higher than baseline after the home-based training program (6.7 ± 1.3, 7.3 ± 1.1 and 7.0 ln ms^2 for pre, post controlled training and post home-based training respectively). As expected, physical performance (VO_{2peak}, time to exhaustion, pace max) tended to decrease during the home-based training program (Hautala et al., 2004) However, some new findings were pointed out. They found a correlation between maximal running performance (time to exhaustion) and changes in HF power ($r=0.44$ $p<0.05$). A similar association was found using the changes in LF/HF ratio. On the contrary, the changes in VO_{2peak} did not correlate with changes in any HRV parameter.

2.4.2.3 HRV and exercise prescription

Kiviniemi, Hautala, Kinnunen and Tulppo (2007) studied the use of prospective short-term (five minutes of sitting, followed by five minutes of standing) morning HRV measurements as a guide to a four weeks endurance training program (6x a week). The basic idea was to decrease the training stimulus when HRV decreased (Kiviniemi et al., 2007). Increase or no change in HRV resulted in high-intensity training that day (80 ± 1% of HR_{max}), whereas a decrease resulted in low-intensity exercise (66 ± 1 % of HR_{max}). They compared the HRV-guided training to a predefined training group, and found that the changes in maximal performance (maximal workload) were significantly greater in the HRV-guided group. There was no difference between groups in the changes of $VO2_{peak.}$ The predefined training group had more high intensity sessions than the HRV-guided group (4 vs. 3), and the weekly training impulse tended to be higher among the predefined training group ($p = 0.063$). Based on these findings the

authors concluded that cardiorespiratory fitness can be improved effectively by using HRV as a guide for daily exercise prescription (Kiviniemi et al., 2007). As this study was done using moderately fit males, the authors did a similar study including women. Furthermore they extended the training period to eight weeks (Kiviniemi et al., 2010). Based on HRV measurements every morning (two minutes of sitting followed by three minutes of standing), moderate intensity training (70% of HR_{max}) or high intensity training (85% of HR_{max}) was prescribed. Each subject had a tailored monitor who did real time analysis of the SD1 value from standing posture. Standing posture was chosen to avoid possible saturation, expressed as unchanged or decreased HRV despite increased cardiac vagal outflow (Kiviniemi et al., 2004). This is suspectible at low HR levels. In that study, they had two HRV-guided training groups, where the second one (HRV-II) only performed high intensity exercises if HRV had increased. This group was tailored only women (n=12). They found no significant changes in mean HR or mean SD1 for men after a single or two successive vigorous-intensity exercises. In women, SD1 was significantly decreased on both the day after a single vigorous-intensity exercise and after two consecutive days. Compared to predefined training and HRV-I, HRV-II performed fewer high intensity exercise session. Despite this, the response in VO_{2peak} didn't differ between the groups. These findings support their previous conclusion, adding that women may benefit in fitness with a lower training load (Kiviniemi et al., 2010).

Table 2-1: Effects of endurance training on short- and long-term heart rate variability and the relation to performance.

Study	Subjects	Training program	HRV methods	Main findings
Kiviniemi et al. 2010	53 moderately fit subjects (21 males, 32 females) 4 groups: ST, HRVI, HRVII (only women) and control.	8 weeks (4-6x per week) 40 min sessions, moderate (70% of HR_{max}) or vigorous intensity (85% of HR_{max}) Self chosen activity (running, cyling, other, combination)	Daily short-term recordings in the morning (sitting and standing). Baseline values from 7-days mean.	↑ HF stand and VO_{2peak} =* changes in $VO_{2peak.}$ ↑* changes in loadmax for HRVI group compared to ST. HRVII performed less high intensity sessions.
Nummela et al. 2009	24 sedentary male subjects. One training group, but in results they are divided in responders and non-responders.	4 weeks 2 hours/week at 76 ± 4% of HRreserve.	Night recordings weekly after training days.	↑ HF in week 4 compared to week 1 for responders. Significant correlation between change in Vmax and change in nocturnal HF power.
Kiviniemi et al. 2007	26 moderately fit men. 2 groups: Predefined training group and HRV-guided training group.	4 weeks 4-6x/week, 40 min low/high intensity; 1 group guided by HRV, 1 group predefined (2 low intensity, 4 high intensity)	Morning recordings; sitting and standing (total 6 minutes). Daily.	= SD1 during standing ↑* changes in both $load_{max}$ and VO_{peak} in HRV group. Increase in loadmax only for tra group?
Pichot et al. 2005	11 elderly men 73.5 ± 4.2 years	14 weeks Interval training on a cycle ergometer 4x/week, 9 intervals of 1 min 85% of $HR_{max.}$ Incl. recovery 45min	Pre-post Holter recordings; 24 hours, analysed for daytime and night hours.	↑ VO_{2max} 18.6% ↑ nocturnal HRV parameters: PNN50, RMSSD and HF power.
Hautala et al. 2003	39 sedentary men 35 ± 9 years	8 weeks 6x a week at moderate intensity (70-80% of HR_{max}) 30-60 minutes	24 hours recording pre.	↑ VO_{2max} 11% ± 5.5% HF power night assoiated with training response
Tulppo et al. 2003	2 groups + control. Low volume High volume	8 weeks 6x per week at 70-80% of HR_{max}; group 1, 30 minutes per session, group 1, 60 minutes per session.	24 hours pre and post.	↑ VO_{2max}, ↑ HF, ↓ LF and LF/HF ratio. ↓ short-term scaling exponent. No difference between groups.
Leicht et al. 2003	13 untrained men 18-27 years	8 weeks Cycling 5x a week, 25-60 min/day at > 80% HR_{max}	Supine rest and during submaximal exercise	↑ VO_{2max}, ↓resting HR, ↑ HF and total power at rest
Carter et al. 2003	Young (19-21 years) vs. middle aged group (40-45 years)	12 weeks Running 4x a week, 45-60 min at 70-90% of HRmax. 2 taper periods (mid) and post.	Supine rest and during submaximal cycling.	↑ performance (2-mile criterion) ↓resting and submaximal HR. ↑HF and total power at rest for both groups.
Catai et al. 2002	10 sedentary young (21 years), 7 sedentary middle-aged (53 years)	12 weeks Walking/running 3x/week, 70-85% of HR_{peak}	Supine and seated rest. 24 hours ECG pre and post.	↑VO_2 at anaerobic threshold ↓Resting HR ↔ HRV, but higher in young subjects.
Perini et al. 2002	15 (7 men, 8 women) old subjects. 73.9±3.5 years.	8 weeks Cycle ergometer, 60 minutes 3x/week. Interval.	Supine, sitting, and submaximal exercise.	↑aerobic power. ↔ HRV
Loimaala et al. 2000	Sedentary, middle-aged subjects.	5 months. Low intensity group, high intensity group.	24-hours ECG	↑VO_{2max} and endurance time. ↔HRV. ↓resting HR in high-intensity group.

See abbreviations or methods for explanation of HRV terms. ↑ increase, ↓ decrease, ↔ no change

2.5 HRV response to sprint interval exercise and training

Sprint interval training involves short bouts (6-30 seconds) of exercise at near maximal or maximal intensities (all-out) with long recovery periods (1.5 min – 4 minutes). It has been proved similar responses in performance as those of aerobic "traditional" endurance training (Burgomaster et al., 2008; Gibala et al., 2006; Burgomaster, Hughes, Heigenhauser, Bradwell, & Gibala, 2005; MacDougall et al., 1998). Sprint interval training and sprint interval exercise influence the physiology of the body differently from moderate or low intensity training and exercise. During low intensity exercise, the main energy comes from aerobic metabolism, whereas SIT requires a mix of both aerobic and anaerobic energy supply, and as the number of intervals increase the rate of glycolisis decreases whereas the aerobic energy production increases (Laursen, 2010) In addition the two exercise protocols differs in use of muscle fibers and neural recruitment. As sprint interval exercise is more explosive, the recruitment of fast twitch muscle fibers are greater (Iaia & Bangsbo, 2010). The studies on sprint interval training, both response to one bout, and long-term adaptation has mostly been done using cycling protocols (MacDougall et al., 1998).

However, the effects of this type of exercise (multiple bouts) have not been examined in relation to autonomic activity. Recently, Millar et al (2009) published an article on HRV recovery following a single and multiple Wingate tests (30s all-out on cycling) in ten, young and healthy men. ECG data were collected in supine position before and after completion of the Wingate tests, and analysis included were time-domain, fractal scaling, spectral analysis and HR complexity. Following a single Wingate test, all HRV measures had returned to baseline within one hour recovery. This was not the case when doing multiple tests showing significant increase in HR and LF/HF ratio for all recovery time points compared to baseline (Millar, Rakobowchuk, McCartney, & MacDonald, 2009).

To date, no study has compared the effects of sprint interval training and continuously moderate intensity training on autonomic function, measured as HRV.

3.0 Methods

This present master thesis was part of a larger training study. There were three main outcomes; performance, insulin sensitivity and heart rate variability. In addition physical activity was measured daily throughout the intervention period. Data collection started in the middle of August 2010 and ended in late December 2010. For this thesis the main outcome is heart rate variability, and the methods related to this are described in detail. Other tests and measurements are described shortly. The study was approved by the Regional Medical Ethics Committee (Appendix I) and performed according to the principles of the declaration of Helsinki. All data were unidentified and stored according to the Norwegian Data Supervision (e.g. Datatilsynet).

3.1 Recruitment and inclusion

We recruited participants by advertising with flyers in the neighborhood of the Norwegian School of Sports Sciences (NSSS), at the University of Oslo and at Oslo University College. We also made a Facebook page and a site on the school`s web page. Invitations for the Facebook page were sent to all of our friends, and they were asked to pass it on. The page was directly linked to the site at the NSSS web. Potential subjects were asked to contact us by telephone or e-mail. 48 potential subjects, both men and women, were asked to come to the laboratory for a standard health questionnaire (Appendix IV), a training questionnaire (Appendix V) and a VO_{2max} test. The schemes were used to identify any illness, or injuries, and sports- and exercise background. Inclusion and exclusion criteria for the study are listed underneath.

Inclusion criteria:

- Age 18- 35 years
- BMI < 30 kg/m^2
- No systematic endurance training the last two years

Exclusion criteria:

- Smokers
- Serious illness (Appendix nr: health declaration for study subjects)
- under 85 % adherence (19 / 24 sessions)

3.2 Subjects

29 subjects were included in the training study (11 male, 18 females). Four subjects dropped out of the intervention. One of the subjects ended her participation because of problems with an old injury. Number two dropped out because he was not comfortable with the training protocol. Number three ended after four weeks due to a fulltime job offer, and he felt the participation took too much time. One more subject dropped out right before the post-training test of VO_{2max} due to an exam period at school.

Finally, 22 subjects completed both training intervention and had a satisfactory number of short-term HRV recordings. Subject characteristics are described based on these 22 subjects (table 3.1). For details about the inclusion of HRV recordings, see testing procedures.

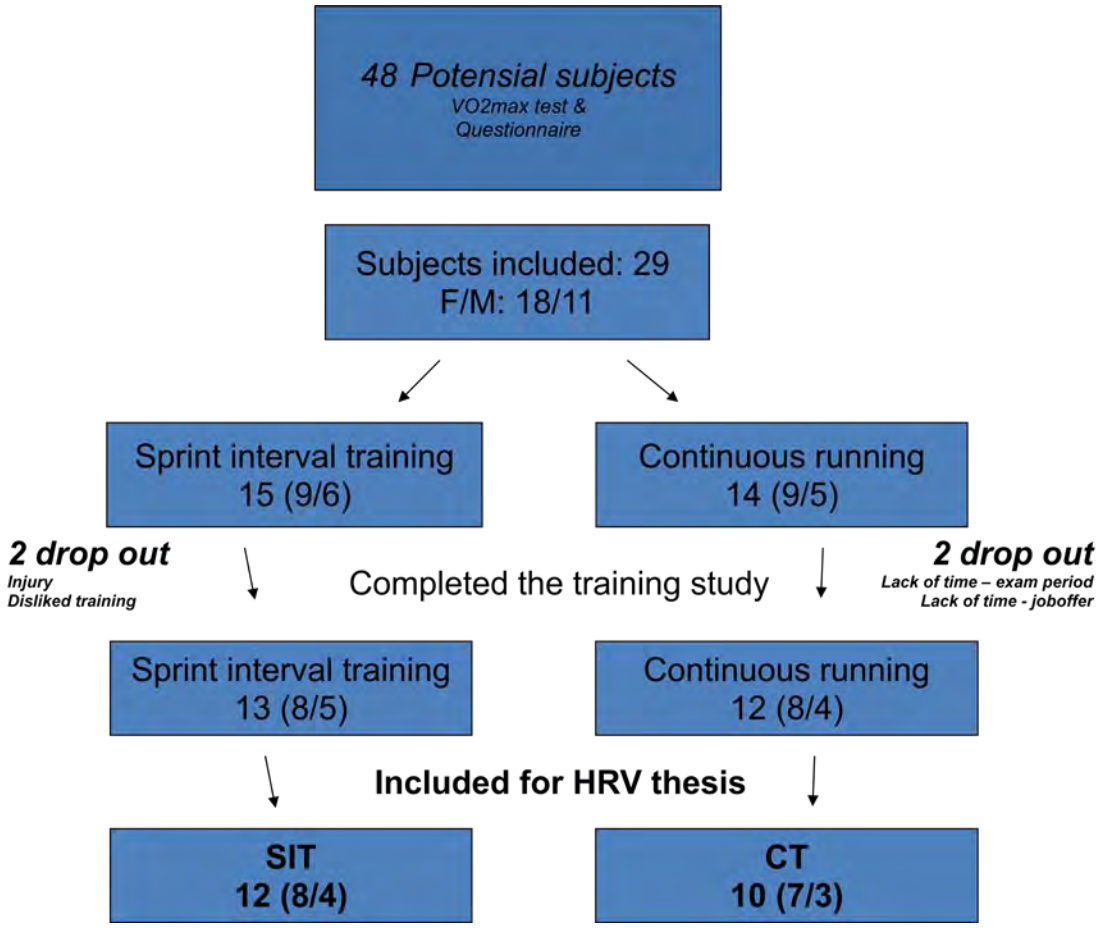


Figure 3-1: The inclusion process and drop-outs for the HRV training study.

There were no significant differences between any baseline characteristics between SIT and CT (table 3-2) regarding sex, height, weight and VO_{2max}. Subjects reported no regular endurance training (e.g. < 2 sessions per week) during the last two years, and their fitness level varied from untrained/sedentary to moderately fit. The subjects were asked to proceed their normal activity level and diet during the intervention period. Subjects with sickness or short injury periods performed an extra training week to receive a satisfactory number of sessions.

All subjects got detailed information about the test- and training procedures, and signed an informed consent according to the declaration of Helsinki and the local ethical committee. They were informed about the right to end their participation in the study (Appendix III).

Table 3-1: Subject characteristics before training intervention.

	SIT (n = 12, ♂4 ♀8)	**CT** (n = 10, ♂3 ♀7)
Age (years)	25.5 ± 0.9	25.4 ± 1.4
Min-max age (years)	22-31	20-35
Bodymass(kg)	69.7 ± 4.2	70.0 ± 3.5
Height (cm)	171.3 ± 2.5	173.0 ± 2.2
$VO_{2\,max}$		
ml/min	3544 ± 235	3270 ± 154
ml kg^{-1} min^{-1}	51.0 ± 1.6	47.2 ± 1.8

Values are expressed as means ± SEM. N= 22.

3.2 Study design

The study was designed as a parallel longitudinal experimental intervention study. Twenty-two subjects completed eight weeks of training (three sessions per week) in either the SIT (n=12) or CT group (n=10). Based on sex, weight and VO_{2max}, all subjects were randomized (by coin) into one of the two groups. Before and after the training period, subjects performed measurements of VO_{2max}, body composition, oral glucose tolerance (OGTT), lung function, long-term 24-hours HRV, anaerobic repeated sprints test, submaximal (stepwise) running test and a shuttle run test. In addition subjects did two short-term measurements of HRV each training week. Testing days are summarized in table 3.2.

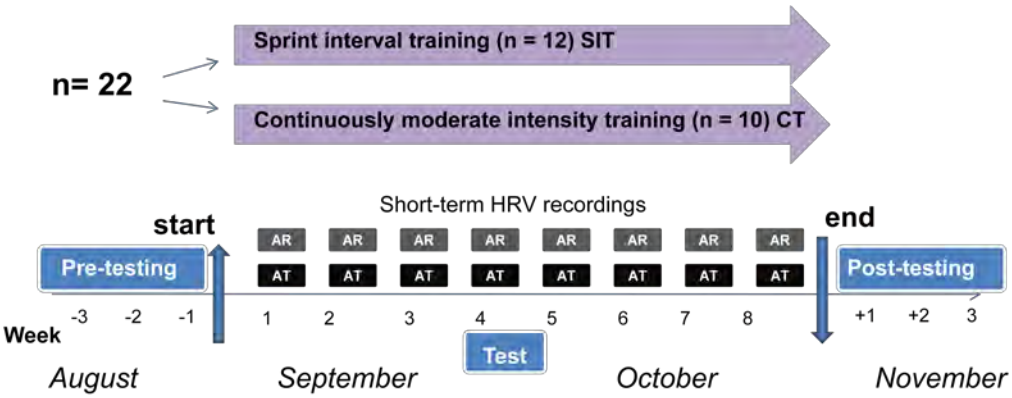


Figure 3-2: The overall design and process for the HRV training study. Figure shows included subjects. AR; after resting day, AT; after training day.

3.4 Testing procedures

The subjects had to meet up for six testing days pre training, one test day mid-training and five days post training. Baseline testing is described in table 3-2. In this master thesis only the VO_{2max} and the HRV measurements are described in detail. Prior to baseline testing, subjects were familiarized with all testing procedures, including two accustoming tests of VO_{2max}, to minimize the possible learning effect. All physical tests were separated by a r esting day. Subjects were not allowed to eat or drink two hours before the physical tests. Post-testing was completed in the same order as the pre-testing.

Table 3-2: Summary of the testing days.

Week	Days	Testing days	Tests and measures
1	Monday	Day 0	Familiarization; VO_{2max} test
	Tuesday		
	Wednesday	Day 1	Start 24-hours HRV-measurement
	Thursday	Day 2	Body composition, OGTT, lungfunction
	Friday	Day 3	$VO2_{max}$ and HR_{peak}*
	Saturday		
	Sunday		
2	Monday	Day 4	Submaximal running test and repeated sprints test
	Tuesday		
	Wednesday	Day 5	Shuttle run test
	Thursday		
	Friday		**Start training**

*The highest HR achieved during the pre-testing of VO_{2max} (familiarization tests included).

3.4.1 Measurement of VO_{2max} and HR_{peak}

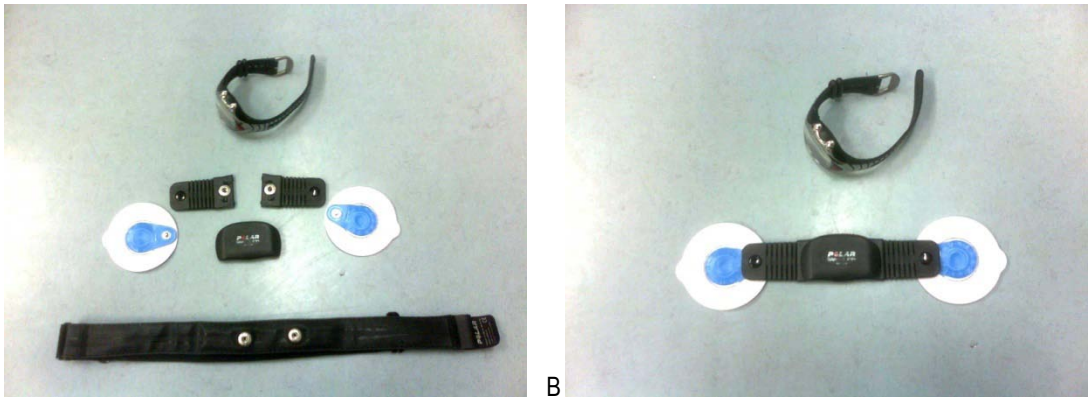
Subjects performed a graded test of maximal oxygen uptake on a treadmill (Woodway pps55 sport, Woodway GmbH, weil an Rhein, Germany). The test started with a 15 minutes warm up. The first ten minutes of warm up were done on a flat treadmill, while the last five minutes were at 5.3% incline (five minutes easy, five minutes light strenuous, five minutes strenuous). After warm up subjects rested for one to two minutes before the test began. The VO_{2max} test was done at 5.3% incline. The starting speed was calculated from the inclusion- and familiarization tests. Pulmonary oxygen uptake was measured over the lungs by breathing through a two-way low resistance mouthpiece (wearing a nose-clip) coupled to a valve (Hans Rudolph 2700 series large two-way non-rebreathing valve, Hans Rudolph, Inc., Kansas City USA). This valve was connected to an ergospirometry system with a mixing chamber (Oxycon Pro; Eric Jaeger, Hoechberg, Germany). Here the expiration air were mixed and then analyzed for contents of O_2 and CO_2. The volume of the expiration air was measured by a turbin (Triple V volume transducter) with an uncertainty of less than 2%. The system was calibrated regularly and manually using gases of known concentration and a three liters pump (model 5530, Hans Rudolph, Kansas City, USA).

During the test work rate (speed) was increased every minute by 1km/h the first minutes. When the subjects seemed exhausted they were asked if they wanted to increase by 1 km/h, 0.5 km/h or if they wanted to keep the current speed. Subjects were verbally motivated by the test leader to run to exhaustion. The oxygen uptake was measured every 30 seconds and the average of the two highest VO_2 measurements was defined as VO_{2max}. A flattening of the curve was used as the main criteria for achieving $VO_{2max.}$ In addition, RER (>1.10), lactate (> 5 mmol/l), the subjects' subjective feeling of exhaustion and the observation by the test leader were used as helping criteria. Lactate was measured two minutes after completing the test.

HR was measured continuously during the test using a Polar HR monitor (Polar RS800CX, Polar Electro Oy, Kempele, Finland). The highest observed HR during VO_{2max} testing (incl. familiarization tests) was defined as HR_{peak}.

3.4.2 Long-term 24-hours HRV measurements.

Subjects arrived at the laboratory between 7 and 8 a.m. in the morning. After removing body hair, the skin was cleaned with isopropanol and two ECG electrodes (Ambu VL-00-S blue sensor electrodes) were placed on the chest. The electrodes were coupled with the Polar receiver (Wearlink W.I.N.D) compatible with a Polar HR monitor (RS800CX; Polar Electro Oy, Kempele, Finland). The HR monitor were set up for recording R-R intervals with a timing resolution of 1 ms. The Polar monitor were placed on the non dominant arm. The subjects were asked to live as normal, without doing any strenuous physical activity or exercise. Since the 24-hours HRV recording was the day before the OGTT they were also asked not to eat or drink (except water) after 10 p.m. The subjects came back the morning after to remove the electrodes and to stop the recording. Data were transferred to a computer and stored for further analysis. Afterwards the subjects performed a body composition test (Inbody 720, Biospace Co, Ltd, Seuol, Korea) and a OGTT.



A B

Figure 3-3: The different parts of the HRV equipment used in the present study. The correct coupling are shown in B, whereas the loose parts are shown in A, and in addition the electrode belt (used for short-term recordings and mid-24 measurements) is shown in A.

Due to a strict time schedule and only six sets of coupling parts to the electrodes (figure 3-3) it was hard to redo any recordings pre and post training. Recordings lasting less than 18 hours were excluded for analysis, and recordings having less than four hours sleeping time were also excluded (by visual inspection, see figure 3-4).

For the mid testing of 24-hours HRV, electrode belts were used. Subjects started their recordings at home the morning after the VO_{2max} test. They were

instructed to clean the skin properly, and to use a conductive aqua gel (Aquasonic) to keep the contact stable throughout the day and night.

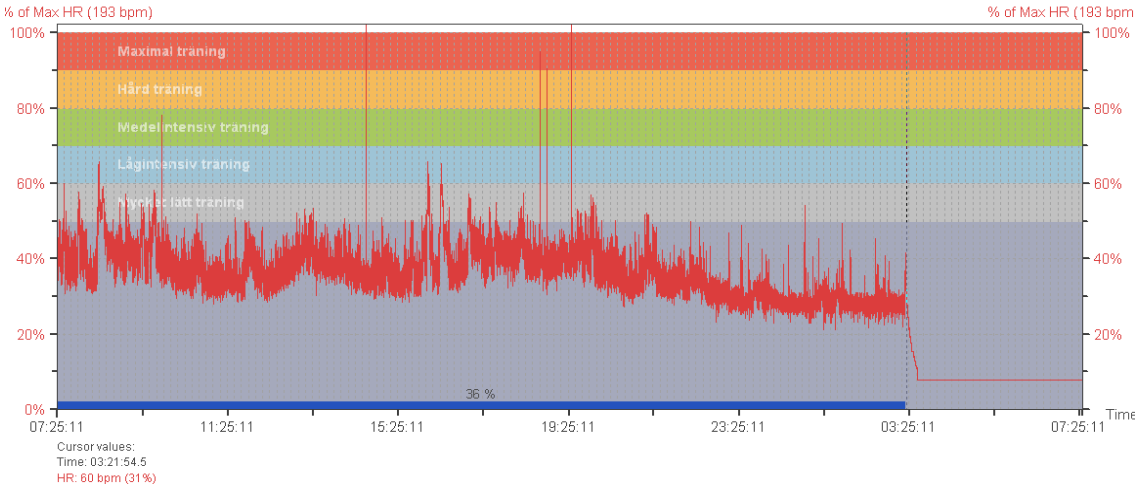


Figure 3-4: Heart rate data from one subject. Registration stopped at 03.18 due to a technical problem, and recording were excluded for analysis (night).

3.4.4 Short-term HRV measurements at home

The subjects were instructed to do two short-term HRV-measurements at home every week during the training period using the Polar electrode belt (Appendix VI). The recordings were done in the morning right after awakening and emptying their urinary bladder. They were allowed to breathe spontaneously. The subjects underwent five minutes of sitting followed by five minutes standing two times a week; once the morning after a resting day (AR) and once the morning after a training day (AT). Data were transferred every second week and stored in Polar Pro Trainer. Figure 3-5 shows the recordings and t raining sessions for one subject.

Unfortunately, some recordings had to be excluded for analysis due to time of measurements (< 12 p.m.), wrong day, or non R-R data.

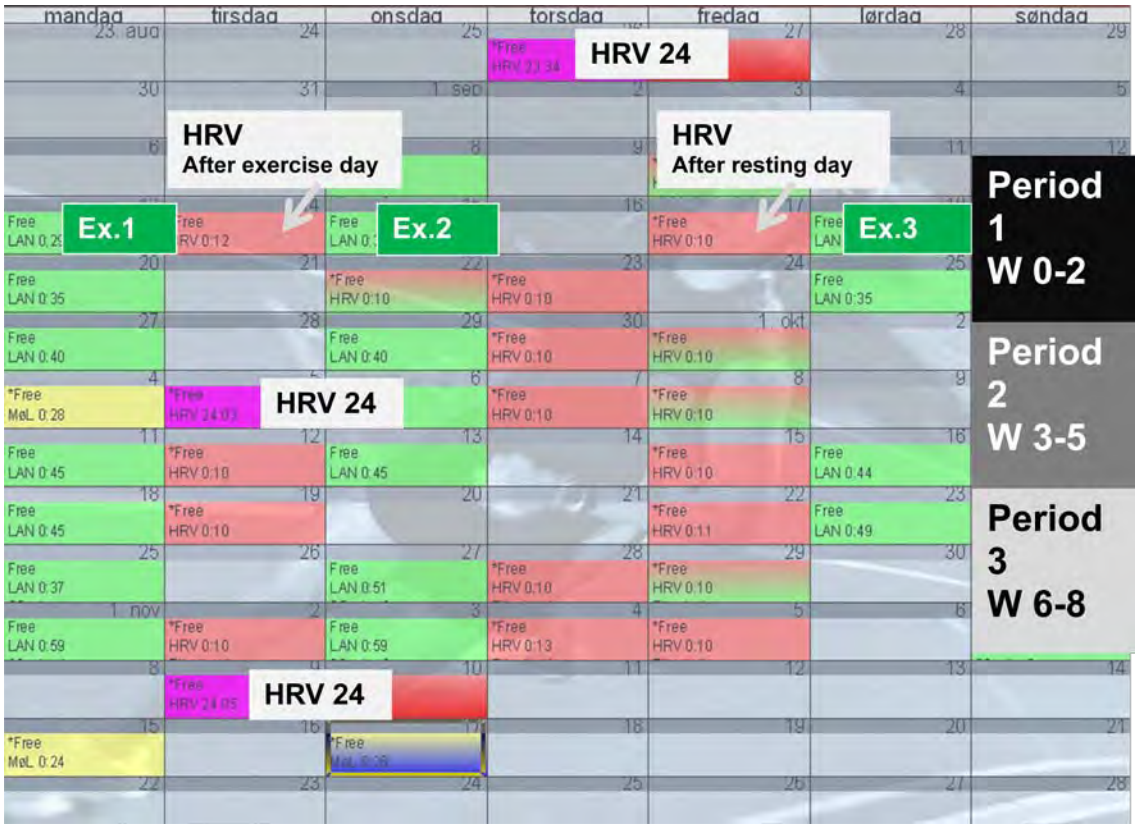


Figure 3-5: The calendar for one subject in Polar Pro Trainer. Light red fields representing short-term HRV recordings. Grey fields, or only red field represents resting days.

The training period was divided in three sub periods; week 0-2 in Period 1 (p1), week 3-5 in Period 2 (p2) and week 6-9 in Period 3 (p2). Some subjects started the training a half week earlier than others, and recordings during this week are set to be in period 1(week 0). Subjects who did not have at least one recording in each period of the intervention were excluded. SIT had a total of 173 (85/88 – AT/AR) qualified recordings whereas CT had a total of 150 (72/78 –AT/AR). The possible maximal number of recordings was 192 for SIT (96/96) and 160 for CT (80/80). In table 3-3, the number of recordings for each day in the three periods is shown.

Table 3-3: Number of short-term recordings include for analysis for SIT and CT, for AR and AT in P1, P2, P3.

	P1		P2		P3	
	AT	AR	AT	AR	AT	AR
CT	18	24	27	25	27	29
SIT	28	26	27	32	30	30

3.4.4.1 Analysis procedures for HRV data

All R-R data were edited and analyzed at the Department of Exercise and Medical Physiology, Verve Research, Oulu, Finland using HRV software (Hearts8, Heart Signal, Oulu, Finland). The R-R data files were exported in ASCII format (in Polar Pro Trainer) for HRV analysis.

In Hearts software the R-R interval time series were edited by visual inspection, and artifacts were either replaced (less than 10 beats) or deleted (more than 10 beats). The procedure is described in detail elsewhere (Salo et al., 2001; Tulppo et al., 1996; Huikuri et al., 1994; Huikuri et al., 1993). On the 24-hours recordings, R-R intervals were calculated from the entire 24-h recording and also separately for the hours representing night time (1 a.m. – 5 a.m.).

For the short-term measurements, data were separated and analyzed for sitting and standing periods of three minutes duration (the last three minutes of the five minutes periods for both sitting and standing period were used for HRV analysis). Figure 3-6 depicts an example short-term recording.

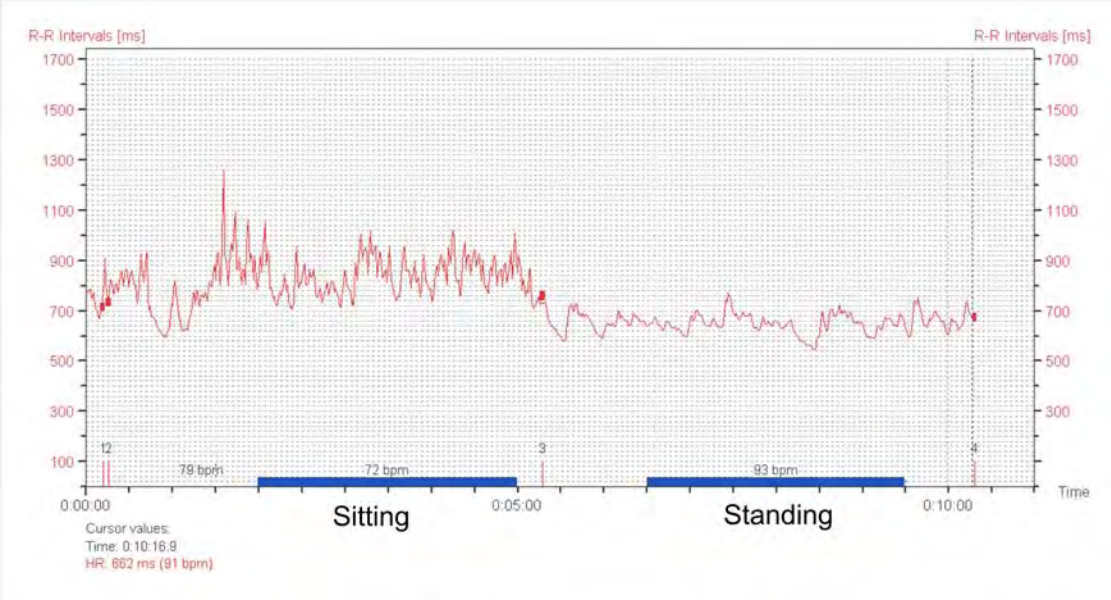


Figure 3-6: Example file of short-term recording in Polar Pro Trainer. Y-axis shows R-R intervals in milliseconds. X-axis shows real time (minutes). Blue lines represents period for sitting and standing posture respectively.

HRV analysis methods included in this thesis are time-domain, frequency-domain (spectral) and non-linear analysis.

In this study, the mean HR (corresponding to the mean value of R-R intervals) and the standard deviation of R-R intervals (SDNN) are used as time-domain methods.

For the spectral analysis, an autoregressive model (order 20) was used to estimate the power spectrum densities of HRV. Block level (block size: 500) detrend was applied to the data. The computer program automatically calculates the autoregressive coefficients to define the power spectrum density. Data were analyzed for 60 minutes segments during the long-term recording. Subjects had less than 2% unqualified beats, which corresponds to > 95000 beats for the 24-hours measurements. For the short-term data, subjects had less than 1% undesirable beats.

The power spectrum was quantified by measuring the area under the two main frequency bands: low frequency power (LF) at 0.04-0.15 Hz and high frequency power (HF) from 0.15-0.4 Hz. From the entire 24-hours measurements ultralow frequency (ULF) power (<0.0033Hz) and very low frequency (VLF) power (0.0033-0.04) were also used. Values are presented as ms^2. The details about the procedures are described elsewhere (Tulppo et al., 2003; Tulppo et al., 1996; Huikuri et al., 1994; Huikuri et al., 1993)

In both short- and long-term data, detrended fluctuation analysis (DFA) was used as a non-linear method. For the 24-hours measurements, the scaling exponent (α^1) was calculated from 1-hour segments, and the mean value of these segments was used.

Additionally, approximate entropy (ApEn), SD1 and SD2 were quantified from both short- and long-term recordings by previously described methods (see theory chapter)

3.5 Training program

The training period lasted for eight weeks. Both SIT and CT exercised three times per week, with at least one resting day between the sessions. Nearly all sessions were supervised and controlled by master students at NSSS. The training sessions were arranged outside the NSSS in either morning or evening sessions. The SIT group did their sprint intervals in a graded track, with an estimated gradient between 5-8%. The CT group was running on partly hilly tracks around the lake "Sognsvann" (191 meters above sea level). Subjects used a HR monitor on each session (Polar RS800CX, Polar Electro Oy, Kempele, Finland), to control and register the exercise intensity. The subjects used relative exercise intensity (%) calculated from their HR_{peak} during training sessions (Polar Electro, 2011). Once every second week the data from the HR monitors were transferred to a computer and stored for further analysis.

3.5.1 SIT

SIT performed repeated 30 seconds sprint intervals (5-10) at near maximal intensity. Subjects were verbally supported and motivated by the training instructor. During the two (~1-2) first intervals, subjects were guided to run at a lower speed, in order to keep the pace up during the remaining intervals. We did not measure the distance they covered, but each subject encouraged tried to reach and push their limit on each sprint. They were also encouraged to follow their HR, and push to reach 90% of HR_{peak}. However, since HR is not a good measure for intensity in anaerobic short duration exercise, the use of the monitor to regulate intensity were not as important as the subjective feeling (Seiler, 2010; Aasen et al., 2005). Each session began with ten minutes warm up at intensity below 85% of $HR_{peak,}$ followed by three strides of ~80 meters. Subjects were guided to do some stretching before they started their sprints. After finishing the training intervention the subjects were asked to rate their perceived exertion (RPE) during the sprint intervals (average feeling) by using the 6-20 Borg scale (Borg, 1982).

Table 3-4: The progression plan for the exercise sessions for SIT group

Week 1	2	3	4	5	6	7	8
5 X 30 s r = 3 min	6 X 30 s r = 3 min	7 X 30 s r = 3 min	8 X 30 s r = 3 min (6 min after the 4th)	8 X 30 s r = 3 min (6 min after the 4th)	9 X 30 s r = 3 min (6 min after the 5th)	10 X 30 s r = 3 min (6 min after the 5th)	10 X 30 s r = 3 min (6 min after the 5th)

r; recovery period, s; seconds, min; minutes

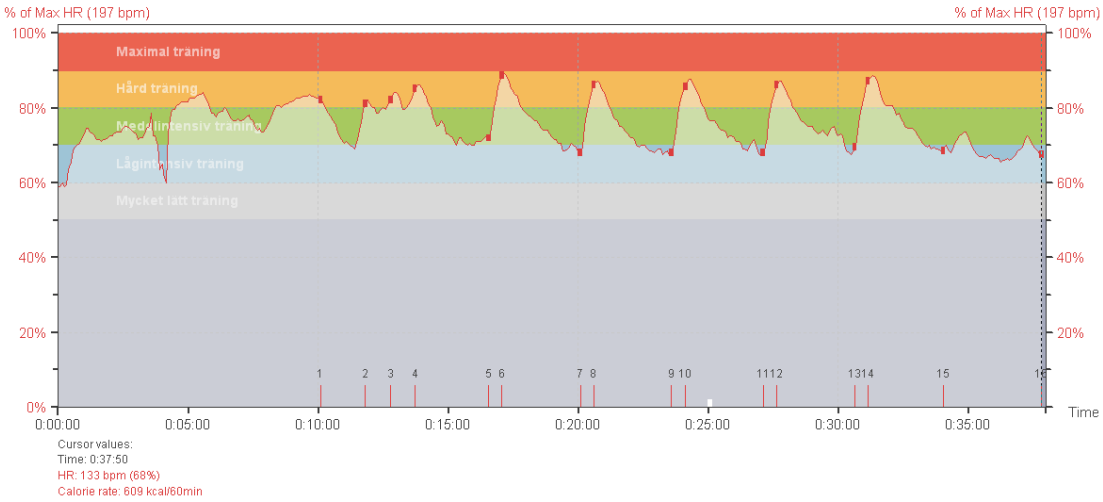


Figure 3-7: HR recording during a SIT session in week 1

3.5.2 CT

CT performed continuous running at moderate intensity, controlled by individual HR. They were instructed to run at the Polar intensity zone 3, between 70 and 80% of HR_{peak} (i.e. HR_{max} (Polar Electro, 2011)). This intensity zone is described as “moderate” by Polar (figure 3-9), but according to ACSM (ACSM, 1998) the intensity is described as “hard” . The Norwegian Olympic Federation (Olympiatoppen) states the present exercise intensity in zone 2. Training in this intensity zone aims to improve the aerobic capacity and exercise economy by improving fat metabolism and thereby the utilization of maximal aerobic capacity (Aasen et al., 2005). All sessions started with ten minutes warm up below 70% of HR_{peak} and ended with five minutes cool down at the same intensity level. After finishing the training intervention the subjects were asked to write their average RPE from the running sessions (excluded warm up and cool down) by using the 6-20 Borg scale (Borg, 1982). The progression plan for CT consisted of increasing the duration of each session (table 3-5).

Table 3-5: The progression plan for the exercise sessions for CT group

Week 1	2	3	4	5	6	7	8
30 min	35 min	40 min	45 min	50 min	55 min	60 min	60 min

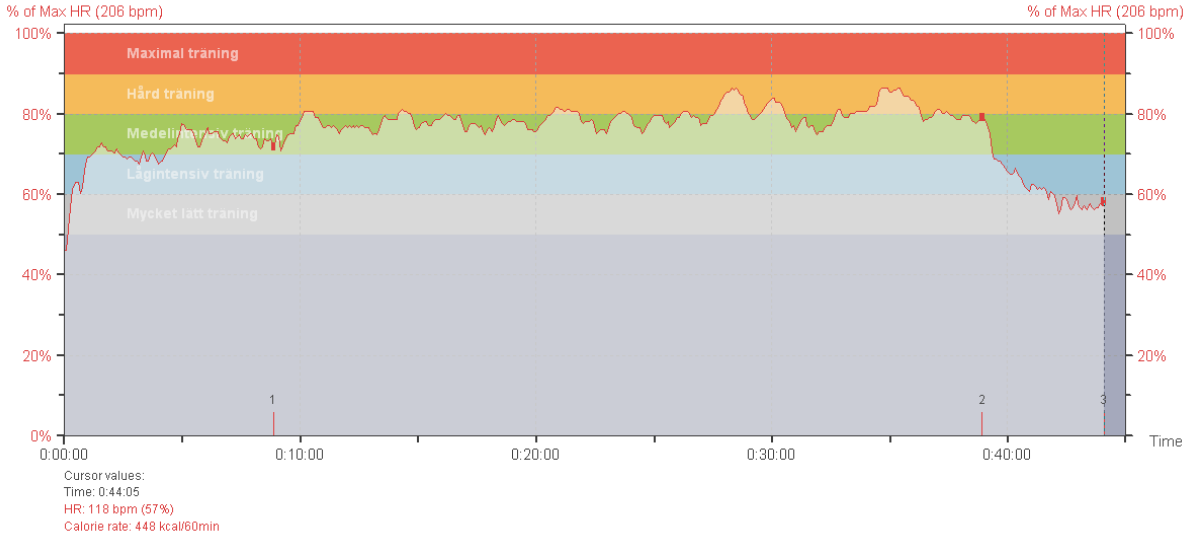


Figure 3-8: HR recording during a CT session in week 1.

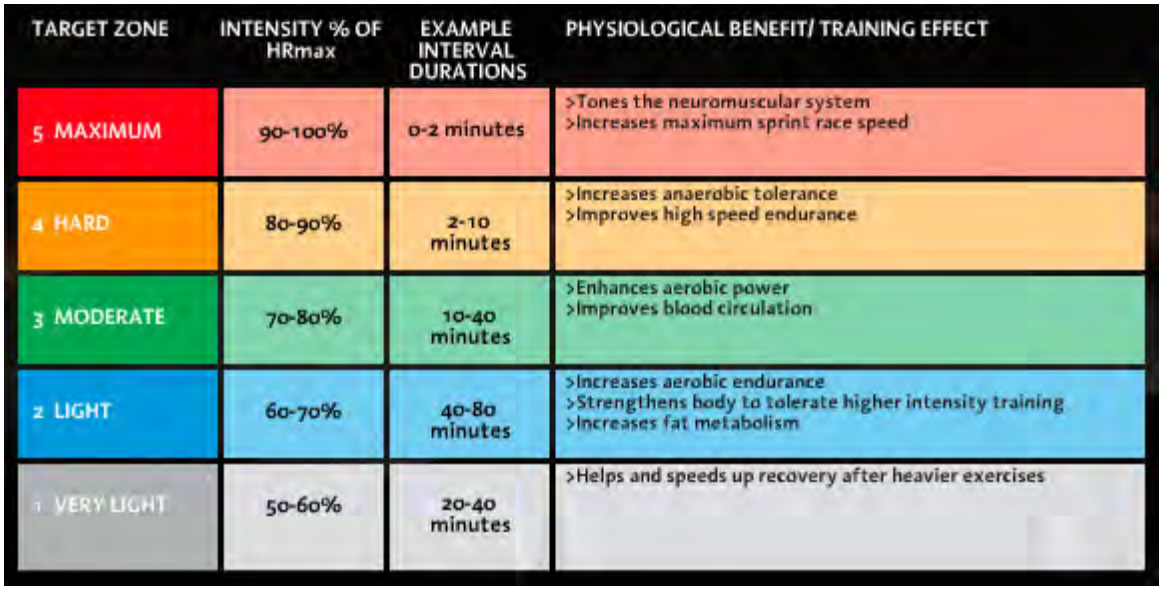


Figure 3-9: Intensity zones downloaded from the website of Polar. (Polar Electro, 2011)

3.6 Data analysis and statistics

All HR and R-R data were saved in a calendar (figure 3-5) for each subject in Polar Protrainer software (Polar Electro Oy, Kempele, Finland). HR files from the training sessions were analyzed in this software, and mean intensity were calculated for both groups. Furthermore peak HR during the sprint intervals and the minimum HR during the recovery period were analyzed. All HR values were plotted in Microsoft Excel (2007 version) for descriptive statistics.

The results are expressed as mean ± SEM (standard error of the mean) for the two groups respectively. Some values are also listed with range (max-min). A Gaussian distribution of the data was verified by the Shapiro-Wilk goodness-of-fit test. For normally distributed data, the differences within the groups were analyzed with a two-tailed Student`s paired t-test. The differences between the two groups were analyzed with an unpaired two-tailed t-test. Wilcoxon signed-rank test (related samples) and Mann Whitney U test (independent samples) were used in not normally distributed data material. Furthermore, analysis of variance (ANOVA) was used to compare the three periods in short-term recordings, and pre-, mid and post in long-term recordings. The post-hoc test used was Fisher´s least significant difference test (LSD). However, ANOVA and LSD were only performed when data indicated a gradual change from the beginning to the end of the training period. Statistical significance was accepted as $p < 0.05$.

Data from spectral analysis were skewed, and a logarithmic transformation (ln) was performed on all spectral components including HF, LF, VLF and ULF. This is quite common in spectral data, and either normalized or log-transformed values are presented (Tulppo et al., 2003).

Pearson product moment- (normal distributed data) or Spearman rho (≥ 1 variable not normal distributed data) correlation coefficient was used to determine the relationship between HRV and training response (VO_{2max}). Statistical analysis was performed in Statistical Package of Social Science (SPSS, version 18) and Microsoft Excel (2007 version). All figures are made in Microsoft Excel, and all tables are made in Microsoft Word (2007 version).

3.7 Economy

The study was financed by the Department of Sports Performance at NSSS. There were no conflict of interest, and neither the participants nor the master students received any payment. However, the participants received sponsored shoes, tights and t-shirt from Puma and some “YT” products from Tine after exercise sessions. The equipment, such as the HR monitor and activity monitor from Polar, was lent out to each participant during the training intervention.

Notifications

It can be noted that some tables includes a comprehensive table text, in order to present data to our collaborators in Finland. The final goal for this thesis is to make a publication. Hence, English writing is chosen.

4.0 Results

Twenty-two young subjects (25.5 ± 0.8 years) had a s atisfactory number of short-term HRV recordings, and finished the eight weeks training intervention with three running sessions per week.

4.1 Training program

The average number of sessions during the intervention period was 22 for SIT and 23 for CT. The total training time for SIT was 564 ± 12 minutes, and for CT 983 ± 18 minutes. SIT had significant lower training volume compared to CT ($p < 0.001$). This was also true when adding the specific warm up for SIT to the total training time (total ~70 minutes). The intensity for CT was 79.0 ± 0.6% of HR_{peak} whereas the intensity for SIT was 73.1 ± 0.9 % of HR_{peak}. The highest HR for SIT during the sprint interval bouts was 91.4 ± 0.4% of HR_{peak}. During recovery periods (3 minutes), HR decreased to 60.9 ± 1.5% of HR_{peak}. For absolute values and range, see table 4-1.

Table 4-1: Selected training characteristics for SIT and CT.

	SIT (n = 12, ♂4 ♀8)	**CT** (n = 10, ♂3 ♀7)
Number of sessions	22 ± 0.6	23 ± 0.4
min-max sessions	19-24	20-24
Total training time[1], min	564 ± 12	983 ± 19[#]
HR		
beats· min^{-1}	145.2 ± 3.4	156.6 ± 2.5
% of HR_{peak}	73.1 ± 0.9	79.0 ± 0.6
% of HR_{peak}	68.5-78.3	75.5-81.8
RPE[2]	18.4 ± 0.4	13.4 ± 0.5
min-max RPE	17-20	10-15
Peak HR during sprint intervals		
beats· min^{-1}	181.3 ± 3.0	-
% of HR_{peak}	91.4 ± 0.4	-
min-max, % of HR_{peak}	89.3-94.0	
Minimum HR during recovery		
beats· min^{-1}	120.9 ± 3.8	-
% of HR_{peak}	60.9 ± 1.5	-
% of HR_{peak}	51.1-70.7	

Values are expressed as means ± SEM. HR; heart rate, RPE; Ratings of perceived exertion, Borg 6-20 scale. [1]Exluding warm up and cool down. [2]Perceived exertion were rated retrospectively at the end of the training period; average feeling during continuously moderate intensity running for CT, and perceived exertion during sprint intervals for SIT Range; the difference between the highest and lowest observed value. # $P < 0.05$; difference between SIT and CT.

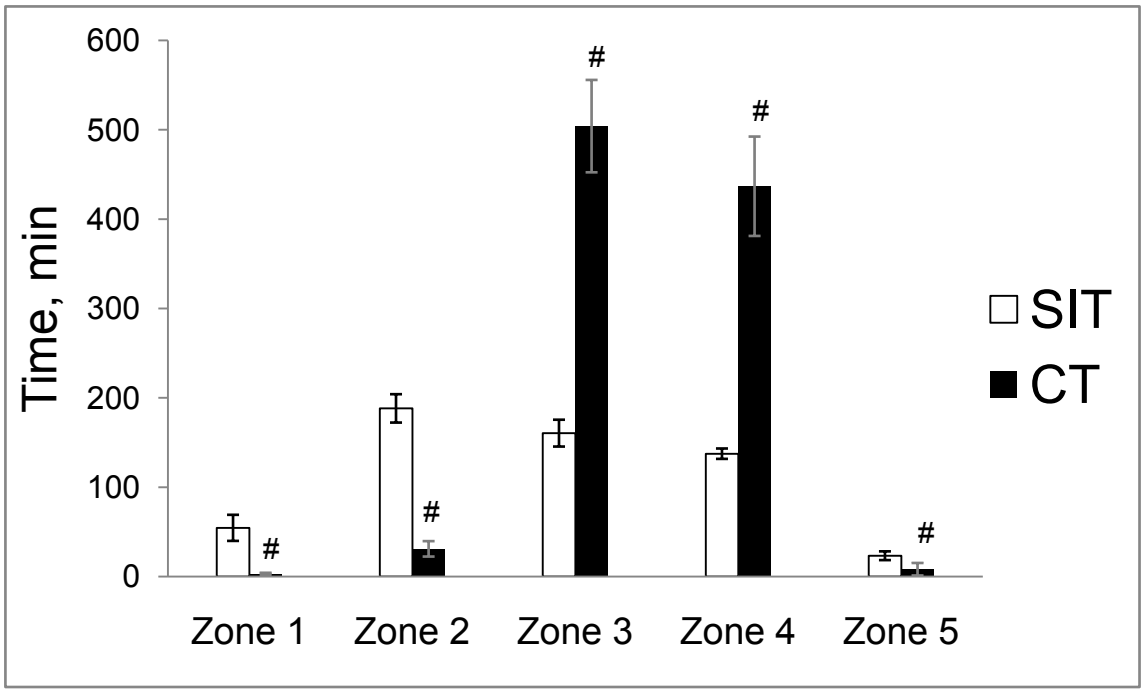


Figure 4-1: Time (min) in different intensity zones for SIT and CT calculated from eight weeks of training. Zone 1; below 60% of HR_{max}, Zone 2; between 60 and 70% of HR_{max}, Zone 3; between 70 and 80% of HR_{max}, Zone 4; between 80 and 90% of HR_{max}, Zone 5; between 90 and 100% of HR_{max}. Total minutes for SIT was 564 ± 12 minutes, and for CT 983 ± 18 minutes # $P < 0.05$ comparisons between groups.

HR was monitored at all training sessions and stored in Polar Pro Trainer for analysis. In figure 4-1 the time (min) in the different exercise intensity zones is shown. The intensity zones that were used are those that are programmed in the Polar monitors (Polar Electro, 2011). There were significant differences in all intensity zones between SIT and CT, both in absolute and relative values ($p < 0.05$). CT had more time in intensity zones 3 and 4, whereas in zone 1, 2 and 5, SIT had more time. In figure 4-1-2 and 4-1-3 you can see an example session showing the distribution in the different intensity zones for SIT and CT, respectively.

In addition to intensity measured by HR, all subjects rated their perceived exertion of the exercise sessions after the training period on the 6-20 Borg scale (Borg, 1982). SIT rated their sprint intervals as “very- to extremely hard” whereas CT rated their continuous running as “light to somewhat hard” (table 4-1).

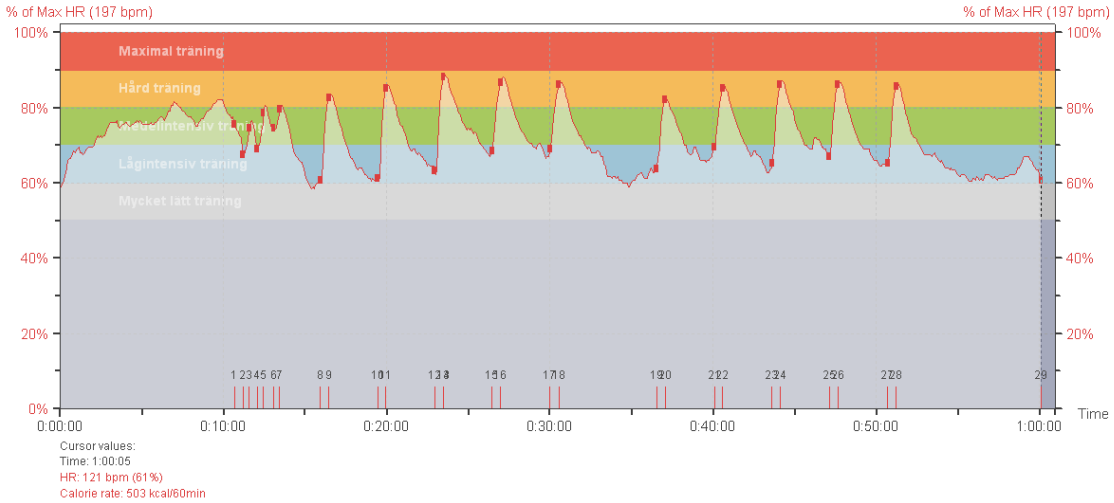


Figure 4-1-2: Example file in Polar Pro Trainer showing a sprint interval session in the last training week. Time (min) is shown on the x-axis whereas % of HR_{peak} is shown on the y-axis. The different colored areas show the different intensity zones. The figure shows the entire session including warm up and cool down.

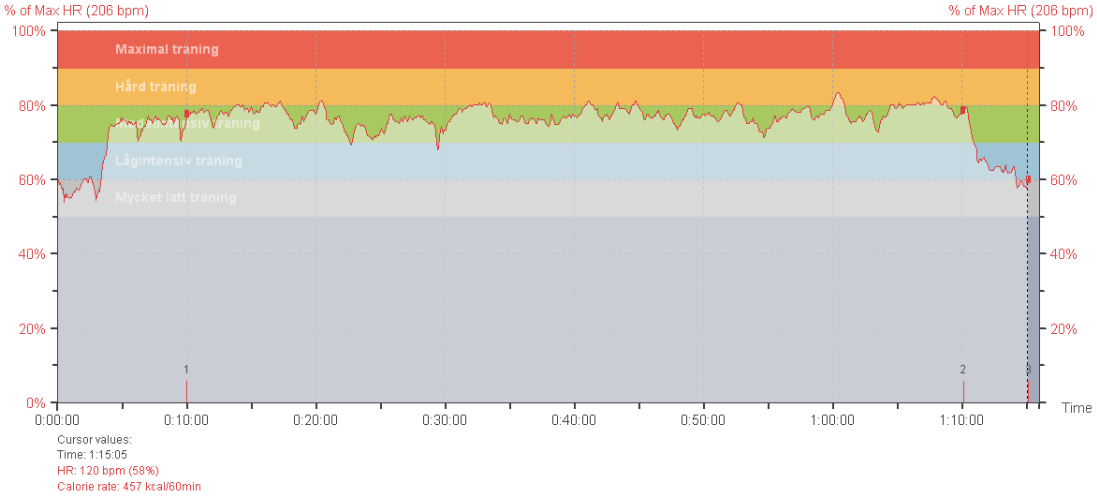


Figure 4-1-3: Example file in Polar Pro Trainer showing a continuously moderate intensity training session in the last training week. Time (min) is shown on the x-axis whereas % of HR_{peak} is shown on the y-axis. The different colored areas show the different intensity zones. The figure shows the entire session including warm up and cool down.

4.2 Maximal oxygen consumption and body composition

Table 4-2: Selected subject characteristics before and after eight weeks of training, in SIT and CT.

	SIT (n = 12, ♂4 ♀8)		**CT** (n = 10, ♂3 ♀7)	
	Pre	Post	Pre	Post
$VO_{2\ max}$				
ml/min	3544 ± 235	3697 ± 235*	3270 ± 154	3388 ± 172
ml· kg^{-1} ·min^{-1}	51.0 ± 1.6	53.5 ± 1.6*	47.2 ± 1.8	49.2 ± 2.0*
$HR_{peak,}$ beats min^{-1}	198.3 ± 3.8	195.2 ± 3.3*	198.4 ± 2.9	194.4 ± 3.1*
Body mass, kg	69.7 ± 4.2	69.8 ± 4.1	70.0 ± 3.5	69.3 ± 2.7
BMI, kg m^2	23.9 ± 0.9	24.0 ± 1.6	23.4 ± 1.1	23.2 ± 1.0
Body fat, %	24.0 ± 1.5	24.1 ± 1.6	24.6 ± 2.0	24.0 ± 2.0
Muscle mass, %	42.3 ± 0.9	42.3 ± 1.0	42.1 ± 1.2	42.3 ± 1.2

Values are expressed as means ± SEM. BMI; body mass index: (mass, kg /(height, m^2)) *P < 0.05, difference between pre- and post values.

Both groups increased VO_{2max} significantly ($p < 0.05$), but there were no difference between groups. SIT increased their $VO2_{max}$ by 2.4 ± 0.7 ml kg^{-1} min^{-1} ($p < 0.01$) and CT had an increase of 2.0 ± 0.9 ml kg^{-1} min^{-1} ($p < 0.05$) for the same parameter. For the absolute values of VO_{2max}, SIT increased significantly ($p < 0.05$), whereas CT tended to increase the absolute values ($p = 0.052$). HR_{peak} decreased in both groups; 3 ± 1 and 4 ± 1 beats·min^{-1} for SIT and CT respectively ($p < 0.05$). There were no changes in body composition before and after the training intervention; no change in body mass, body fat or muscle mass occurred.

4.3 Short-term measurements of HRV

Short-term HRV are presented separate for sitting posture (table 4-3-1) and for standing posture (table 4-3-2). SIT (n=12) had a total of 173 (72/78) qualified recordings (AR/AT), whereas CT (n=10) had a total of 150 (85/88).

4.3.1 Sitting posture HRV

For SIT there were significant changes between HRV on AR days compared to AT days in Period 1, in sitting posture (table 4-3-1). They had a significantly lower HR on AR days ($p < 0.01$), and SDNN, HF power (ln, ms^2), and SD1 were significantly higher ($p < 0.01$). The LF/HF ratio and the α1 were significantly lower on AR days compared to AT days in the same period ($p < 0.05$). These changes were not observed in period 2 and 3. For CT no changes were observed between AR and AT in any period.

During the training period, HR in sitting posture on AR days gradually increased for SIT; 63.4 ± 1.8, 64.8 ± 2.2, 67.8 ± 2.7 beats/min in period 1, 2 and 3 respectively ($p < 0.05$, p3-p1). SDNN tended to decrease showing these values; 109.4 ± 5.0, 100.5 ± 8.2, 91.2 ± 10.2 ms in period 1, 2 and 3 respectively ($p=0.097$,p3-p1). Despite no change in LF- and HF power between periods for AR and AT in SIT subjects, LF/HF ratio decreased on AT days from 5.3 ± 0.7 (period 1) to 3.4 ± 0.5 (period 3). There were no other changes observed on AT days between periods for SIT.

CT had similar overall HRV values in sitting posture on AR days compared to SIT ($p > 0.3$). On AT days in period 1 there was a significant difference in LF/HF ratio between groups. CT showed lower ratio compared to SIT (table 4-3-1). From the non-linear analysis, a significant difference between groups in α1 was found on AT days in period 1($p < 0.05$). These differences were not apparent in period 2 and 3.

4.3.2 Standing posture HRV

SIT showed a decrease in HRV on AT days compared to AR days in period 1, also for standing posture. An increased SDNN, HF power ($p <0.05$) and SD1 ($p=0.007$) were observed. Furthermore a lower HR was observed AR in the two

first training weeks, compared to AT (table 4-3-2). These changes were not observed during the last six weeks of the training intervention. However, SIT tended to have a lower APEN value on AR days compared to AT day in period 3 ($p = 0.055$). For CT, no changes were observed between AR and AT in any period.

During the training intervention HR increased by 5.4 ± 2.0 beats·min^{-1} for SIT from period 1 till period 3 ($p < 0.05$, table 4.3.3). SD1 and HF power decreased significantly from period 1 to period 3 in standing posture ($p < 0.01$). LF power also showed a significant decrease ($p = 0.026$), but no change in LF/HF ratio (table 4-3-2). The α1 increased from Period 1 to period 3 ($p < 0.05$) on AR days. There were no significant differences on AT days between periods for SIT in standing posture. For CT, no changes were observed between period 1 and period 3 in either AR or AT.

CT and SIT were similar in almost all HRV parameters in standing posture on AR and AT days ($p > 0.05$), but for HR in period 1 on AR days, there was a nearly significant higher HR in CT ($p= 0.056$).

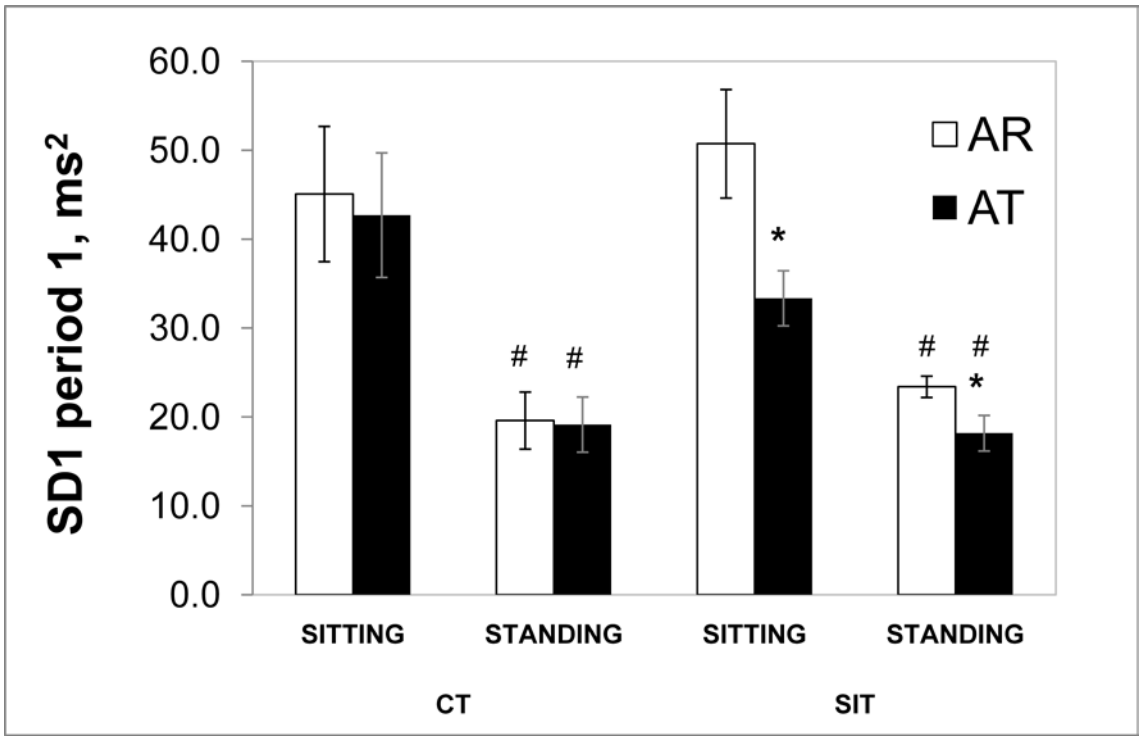


Figure 4-3: Sitting and standing values of SD1 for SIT (right) and CT (left); comparison between AR (white) and AT (black). * P < 0.05; difference between AR and AT. # P < 0.05; difference between sitting and standing posture.

Table 4-3-1: Short-term HRV in sitting posture

Table 4-3-1: Sitting short-term HRV during eight weeks of sprint interval (SIT) and continuously moderate intensity training (CT). R-R intervals were recorded for 10 minutes in the morning (5 min sitting followed by 5 min standing) and sitting HRV was calculated between 1.5-4.5 min of sitting. Subjects did two recordings per week; one after a training day (AT) and one after a resting day (AR) Data in Period 1 represents means of registrations during week 0,1 and 2; data in Period 2 represents means of registration in week 3-5; data in Period 3 represents means of registration during week 6-8. Number of days with HRV analysis for SIT were (AT/AR) 28/26,27/32,30/30 in Period 1, Period 2, and Period 3, respectively (total 173). Number of days with HRV analysis for CT were (AT/AR) 18/24,27/25,27/29 in Period 1, Period 2, and Period 3, respectively (total 150).

	SIT (n = 12)			CT (n = 10)		
	Period 1	Period 2	Period 3	Period 1	Period 2	Period 3
HR, beats/min						
AT	68.2 ± 2.6	66.5 ± 2.5	67.6 ± 2.4	67.3 ± 2.3	65.4 ± 2.3	65.0 ± 3.0
AR	63.4 ± 1.8^{*at}	64.8 ± 2.2	67.8 ± 2.7^{p1}	65.9 ± 2.7	63.6 ± 2.3	65.0 ± 2.5
SDNN, ms						
AT	86.1 ± 8.0	85.9 ± 9.4	83.1 ± 6.6	88.7 ± 13.5	88.4 ± 13.9	87.7 ± 13.0
AR	109.4 ± 5.0^{*at}	100.5 ± 8.2	91.2 ± 10.2	94.8 ± 15.2	93.8 ± 12.2	89.9 ± 14.9
SD1, ms						
AT	33.4 ± 3.1	36.4 ± 5.3	35.4 ± 4.1	42.7 ± 7.0	42.3 ± 7.5	42.6 ± 7.6
AR	50.7 ± 6.1^{*at}	44.6 ± 5.1	41.6 ± 6.7	45.1 ± 7.6	44.9 ± 7.0	44.2 ± 8.6
SD2, ms						
AT	116.4 ± 11.0	115.0 ± 12.3	111.2 ± 8.6	116.5 ± 18.3	116.6 ± 18.5	115.1 ± 17.2
AR	144.3 ± 17.0^{*at}	133.2 ± 10.9	121.2 ± 13.0	124.5 ± 20.8	124.1 ± 15.8	118.2 ± 19.3
HF, ln ms^2						
AT	6.4 ± 0.2	6.5 ± 0.2	6.6 ± 0.2	6.8 ± 0.4	6.6 ± 0.6	6.7 ± 0.4
AR	7.2 ± 0.3^{*at}	7.0 ± 0.3	6.7 ± 0.2	6.7 ± 0.6	6.8 ± 0.5	6.7 ± 0.4
LF, ln ms^2						
AT	7.8 ± 0.2	7.7 ± 0.2	7.6 ± 0.2	7.5 ± 0.4	7.2 ± 0.6	7.4 ± 0.3
AR	8.1 ± 0.2^{*at}	7.9 ± 0.2	7.8 ± 0.2	7.6 ± 0.6	7.6 ± 0.4	7.4 ± 0.4
LF/HF ratio						
AT	5.3 ± 0.7	4.6 ± 0.9	3.4 ± 0.5^{p1}	2.8 ± 0.6$^{\#}$	3.1 ± 1.0	2.7 ± 0.6
AR	3.5 ± 0.5^{*at}	3.4 ± 0.6	3.8 ± 0.6	3.8 ± 1.0	3.4 ± 1.0	3.1 ± 0.8
APEN						
AT	0.7 ± 0.02	0.6 ± 0.03^{p1}	0.7 ± 0.04	0.6 ± 0.04	0.7 ± 0.04	0.6 ± 0.04
AR	0.6 ± 0.04	0.6 ± 0.03	0.6 ± 0.04	0.6 ± 0.03	0.6 ± 0.04	0.6 ± 0.03
α_1						
AT	1.4 ± 0.06	1.3 ± 0.07	1.3 ± 0.06	1.2 ± 0.07$^{\#}$	1.2 ± 0.08	1.2 ± 0.08
AR	1.2 ± 0.07	1.2 ± 0.05	1.2 ± 0.04	1.2 ± 0.09	1.2 ± 0.06	1.2 ± 0.07

Values are expressed as means ± SEM. SDNN, standard deviation of all R-R intervals; SD1, standard deviation of data points perpendicular to the line of identity (Poincare plot) ;SD2, standard deviation of data points along the line of identity (Poincare plot); HF, high frequency power; LF, low frequency power; ln, natural logarithm; LF/HF ratio, absolute LF power divided by absolute HF power; APEN, approximate entropy; α_1, fractal scaling exponent in detrended fluctuation analysis.

^{*at} $P < 0.05$; comparisons between AR and AT,

^{p2} $P < 0.05$; comparisons between period 3 and period 2

^{p1} $P < 0.05$; comparisons between period 3 and period 1 or period 2 and period 1.

# $P < 0.05$; comparisons between CT and SIT

Table 4-3-2: Short-term HRV in standing posture

Table 4-3-2: Standing short-term HRV during eight weeks of sprint interval (SIT) and continuously moderate intensity training (CT). R-R intervals were recorded for 10 minutes in the morning (5 min sitting followed by 5 min standing) and sitting HRV was calculated between 1.5-4.5 min of sitting. Subjects did two recordings per week; one after a training day (AT) and one after a resting day (AR) Data in Period 1 represents means of registrations during week 1 and 2; data in Period 2 represents means of registration in week 3-5; data in Period 3 represents means of registration during week 6-8. Number of days with HRV analysis for SIT were (AT/AR) 28/26,27/32,30/30 in Period 1, Period 2, and Period 3, respectively (total 173). Number of days with HRV analysis for CT were (AT/AR) 18/24,27/25,27/29 in Period 1, Period 2, and Period 3, respectively (total 150).

	SIT (n = 12)			CT (n = 10)		
	Period 1	Period 2	Period 3	Period 1	Period 2	Period 3
HR, beats/min						
AT	81.6 ± 3.3	80.1 ± 3.4	82.2 ± 3.0	86.7 ± 3.1	87.4 ± 3.5	86.3 ± 3.9
AR	77.1 ± 2.9*^{at}	79.4 ± 3.4	82.5 ± 3.6^{p1}	86.7 ± 3.9	85.4 ± 3.5	85.2 ± 3.4
SDNN, ms						
AT	63.2 ± 6.0	68.6 ± 6.6	61.4 ± 4.4	64.5 ± 6.9	60.3 ± 5.8	63.4 ± 8.5
AR	70.2 ± 5.0*^{at}	71.6 ± 7.4	63.6 ± 5.2	62.1 ± 5.9	64.6 ± 7.4	69.8 ± 8.0
SD1, ms						
AT	18.2 ± 2.0	21.0 ± 5.3	17.3 ± 1.5	19.2 ± 3.1	16.9 ± 2.0	20.6 ± 4.0
AR	23.4 ± 1.8*^{at}	21.3 ± 2.8	17.5 ± 1.8^{p1}	19.6 ± 3.2	18.6 ± 2.7	20.1 ± 3.0
SD2, ms						
AT	87.1 ± 8.3	94.3 ± 8.9	111.2 ± 8.5	88.7 ± 9.2	83.2 ± 7.6	86.9 ± 11.4
AR	96.0 ± 7.1	98.5 ± 10.1	87.9 ± 7.1	90.1 ± 9.7	89.0 ± 10.0	96.2 ± 10.9
HF, ln ms^2						
AT	5.1 ± 0.2	5.4 ± 0.2	5.1 ± 0.1	5.4 ± 0.3	5.2 ± 0.2	5.3 ± 0.4
AR	5.7 ± 0.2*^{at}	5.4 ± 0.3	5.2 ± 0.2^{p1}	5.3 ±0.3	5.2 ± 0.3	5.4 ± 0.3
LF, ln ms^2						
AT	7.4 ± 0.2	7.4 ± 0.2	7.3 ± 0.2	7.3 ± 0.3	7.1 ± 0.2	7.2 ± 0.3
AR	7.6 ± 0.2	7.4 ± 0.2	7.3 ± 0.9^{p1}	7.2 ± 0.2	7.1 ± 0.3	7.4 ± 0.2
LF/HF ratio						
AT	10.5 ± 1.6	9.6 ± 1.5	10.1 ± 1.5	8.4 ± 1.6	7.5 ± 0.9	7.5 ± 0.9
AR	8.3 ± 1.3*^{at}	8.4 ± 1.1	8.9 ± 1.0	8.1 ± 1.6	8.3 ± 1.3	8.2 ± 1.4
APEN						
AT	0.7 ± 0.03	0.7 ± 0.03	0.7 ± 0.03	0.7 ± 0.04	0.6 ± 0.04	0.7 ± 0.04
AR	0.7 ± 0.04	0.7 ± 0.03	0.6 ± 0.04*^{at}	0.7 ± 0.03	0.6 ± 0.04	0.7 ± 0.04
α_1						
AT	1.6 ± 0.03	1.6 ± 0.04	1.6 ± 0.03	1.5 ± 0.04	1.6 ± 0.03	1.5 ± 0.03
AR	1.5 ± 0.05*^{at}	1.5 ± 0.04	1.6 ± 0.02^{p1}	1.6 ± 0.04	1.6 ± 0.03	1.6 ± 0.02

Values are expressed as means ± SEM. SDNN, standard deviation of all R-R intervals; SD1, standard deviation of data points perpendicular to the line of identity (Poincare plot) ;SD2, standard deviation of data points along the line of identity (Poincare plot); HF, high frequency power; LF, low frequency power; ln, natural logarithm; LF/HF ratio, absolute LF power divided by absolute HF power; APEN, approximate entropy; α_1, fractal scaling exponent in detrended fluctuation analysis.
*^{at} P < 0.05; comparisons between AR and AT,
^{p2} P < 0.05; comparisons between period 3 and period 2
^{p1} P < 0.05; comparisons between period 3 and period 1 or period 2 and period 1.
# P < 0.05; comparisons between CT and SIT
(**/^{pp} P < 0.001)

Table 4-3-3: Changes in short-term HRV during eight weeks of training

Table 4.3.3: Changes in short-term HRV variables during eight weeks of training; difference between period 1 (p1) and period 3 (p3) in absolute units (Δp3-p1). Data are presented for "sitting" and "standing" posture separately. P-values are from t-tests between period 1 and period 3 for each day and parameter. See table 4.3.1 and 4.3.2 for original values.

	SIT (n = 12)				CT (n = 10)			
	SITTING		STANDING		SITTING		STANDING	
	Δ p3-p1	*P-value*	Δ p3-p1	*P-value*	Δ p3-p1	*P-value*	Δ p3-p1	*P-value*
HR, beats/min								
AT	-0.7 ± 1.7	*0.703*	0.7 ± 1.9	*0.736*	-2.3 ± 1.5	*0.163*	-0.3 ± 1.7	*0.845*
AR	4.4 ± 1.8	*0.036*	5.4 ± 2.0	*0.019*	-0.8 ± 2.0	*0.697*	-1.5 ± 2.7#	*0.595*
SDNN, ms								
AT	-3.0 ± 6.8	*0.670*	-1.8 ± 3.5	*0.625*	-0.9 ± 5.7	*0.879*	-1.1 ± 4.0	*0.797*
AR	-18.2 ± 10.1	*0.097*	-6.7 ± 4.2	*0.141*	-4.9 ± 8.1	*0.557*	7.7 ± 6.5	*0.265*
SD1, ms								
AT	2.0 ± 4.0	*0.621*	-15.2 ± 2.5	*0.593*	-0.1 ± 4.3	*0.974*	1.5 ± 1.7	*0.406*
AR	-9.1 ± 6.1	*0.163*	-5.9 ± 1.6	*0.003*	-0.9 ± 7.3	*0.903*	0.5 ± 2.4#	*0.850*
SD2, ms								
AT	-5.1 ± 9.5	*0.600*	-2.3 ± 4.9	*0.646*	-1.4 ± 7.6	*0.857*	-1.8 ± 5.5	*0.754*
AR	-23.1 ± 13.7	*0.120*	-23.1 ± 13.7	*0.120*	-6.3 ± 9.9	*0.538*	6.1 ± 6.8	*0.397*
HF, ln ms^2								
AT	0.2 ± 0.2	*0.321*	0.0 ± 0.1	*0.853*	-0.1 ± 0.2	*0.682*	-0.1 ± 0.2	*0.702*
AR	-0.4 ± 0.2	*0.100*	-0.5 ± 0.2	*0.010*	0.1 ± 0.4	*0.851*	0.1 ± 0.3#	*0.651*
LF, ln ms^2								
AT	-0.2 ± 0.1	*0.106*	-0.0 ± 0.0	*0.788*	-0.1 ± 0.2	*0.702*	-0.0 ± 0.2	*0.822*
AR	-0.3 ± 0.2	*0.070*	-0.3 ± 0.2	*0.132*	-0.1 ± 0.2	*0.667*	0.2 ± 0.2	*0.418*
LF/HF ratio								
AT	-1.9 ± 0.7	*0.016*	-0.4 ± 1.4	*0.778*	-0.1 ± 0.4#	*0.767*	-0.9 ± 1.1	*0.420*
AR	0.3 ± 0.7	*0.689*	0.6 ± 0.7	*0.386*	-0.7 ± 0.7	*0.300*	0.2 ± 1.0	*0.881*
APEN								
AT	-0.01 ± 0.03	*0.747*	0.03 ± 0.01	*0.121*	0.00 ± 0.02	*0.992*	0.00 ± 0.04	*0.902*
AR	0.04 ± 0.04	*0.275*	-0.03 ± 0.03	*0.324*	0.04 ± 0.05	*0.483*	0.01 ± 0.03	*0.767*
α_1								
AT	-0.09 ± 0.07	*0.200*	0.02 ± 0.04	*0.703*	0.02 ± 0.07	*0.810*	0.03 ± 0.04	*0.492*
AR	0.02 ± 0.05	*0.650*	0.10 ± 0.03	*0.012*	-0.07 ± 0.07	*0.381*	0.02 ± 0.04	*0.730*

Values are expressed as means ± SEM. SDNN, standard deviation of all R-R intervals; SD1, standard deviation of data points perpendicular to the line of identity (Poincare plot) ;SD2, standard deviation of data points along the line of identity (Poincare plot); HF, high frequency power; LF, low frequency power; ln, natural logarithm; LF/HF ratio, absolute LF power divided by absolute HF power; APEN, approximate entropy; α_1, fractal scaling exponent in detrended fluctuation analysis.P-value; from the difference between values period 1 and period 3 (table 4-3-2 and 4-3-2). # $P < 0.05$; difference between CT and SIT.

4.3.3 Differences in changes from period 1 to period 3

Between groups there were significant differences in ΔHR p1-p3 on AR days in standing posture (table 4-3-3). CT had a 1.5 ± 2.7 beats·min^{-1} decrease compared to an increase of 5.4 ± 2.0 beats·min^{-1} for SIT ($p < 0.05$). For SD1 and HF, the changes between periods differed significantly between groups (standing posture), showing an increase in HF power for CT (0.5 ± 2.4 ln, ms) versus a decrease (-0.5 ± 0.2 ln, ms^2) for SIT ($p < 0.05$). The same appeared for changes in SD1 ($p < 0.05$), see table 4-3-3 for values.

4.3.3 Difference between sitting and standing posture

For SIT, there was a significant change between sitting and standing posture in HR, SDNN, SD1, LF/HF ratio and α1 on both AR and AT days in all periods ($p < 0.05$). Only in LF (ln, ms^2) there was no change between sitting and standing posture in any period (table 4.3.4). For APEN there was no difference between sitting and standing posture, except on AT days in period 2. For SD2 it was significant changes in both days in all three periods, except on AT days in period 2 ($p = 0.184$)

CT was similar as SIT, except no significant changes for SDNN. In addition they had no significant changes in APEN values and SD2 values. For HF power there was no significant change in period 1 on AR days ($p= 0.058$).

Between groups there was observed a larger increase for CT in HR from sitting to standing posture. On average in period 2, CT had an increase of 22.0 ± 2.6 beats·min^{-1} on AT days, whereas SIT had an increase of 13.5 ± 1.6 beats·min^{-1} ($p= 0.009$ for AT, difference between SIT and CT)

Table 4-3-4: Difference in HRV between sitting and standing posture

Table 4.3.4: Changes in short-term HRV variables between sitting and standing position (Sitting subtracted by standing) during 8 weeks of training. Data are presented as absolute values (Δstanding-sitting). For sitting and standing values see table 4.3.1 and 4.3.2.

	SIT (n = 12)			CT (n = 10)		
Stand - sitting	Period 1	Period 2	Period 3	Period 1	Period 2	Period 3
HR, beats/min						
AT, Δ standing-sitting	13.3 ± 2.1*	13.5 ± 1.6*	14.7 ± 1.6*	19.4 ± 2.0*	22.0 ± 2.6*#	21.3 ± 2.9*#
AR, Δ standing-sitting	13.7 ± 2.0*	14.7 ± 1.7*	14.8 ± 1.7*	20.8 ± 2.7*#	21.8 ± 3.0*#	20.2 ± 2.6*
SDNN, ms						
AT, Δ standing-sitting	-22.7 ± 5.7*	-17.4 ± 7.4	-21.6 ±6.4*	-24.2 ± 9.1	-28.1 ± 12.3	-24.3 ± 9.8
AR, Δ standing-sitting	-39.2 ± 9.8*	-28.9 ± 6.9*	-27.6 ± 9.0*	-32.7 ± 16.1	-29.2 ± 10.0	-20.1 ± 12.0
SD1, ms						
AT, Δ standing-sitting	-15.2 ± 2.5*	-15.4 ± 3.1*	-18.1 ± 3.5*	-23.5 ± 5.3*	-25.4 ± 7.2*	-22.0 ± 6.1*
AR, Δ standing-sitting	-27.3 ± 4.9*	-23.3 ± 3.6*	-24.1 ± 6.1*	-25.5 ± 7.7*	-26.2 ± 6.4*	-24.1 ± 6.8*
SD2, ms						
AT, Δ standing-sitting	-29.2 ± 7.8*	-20.8 ± 10.1	-26.4 ± 8.6*	-27.8 ± 12.4	-33.5 ± 17.9	-28.2 ± 12.8
AR, Δ standing-sitting	-48.3 ± 13.3*	-34.7 ± 10.0*	-33.2 ± 11.52*	-34.4 ± 18.8	-35.1 ± 13.0	-22.0 ± 15.8
HF, ln ms^2						
AT, Δ standing-sitting	-1.2 ± 0.2*	-1.1 ± 0.2	-1.4 ± 0.2*	-1.4 ± 0.4*	-1.4 ± 0.7*	-1.4 ± 0.4*
AR, Δ standing-sitting	-1.5 ± 0.1*	-1.5 ± 0.2*	-1.6 ± 0.2*	-1.3 ± 0.7	-1.7 ± 0.7*	-1.3 ± 0.4*
LF, ln ms^2						
AT, Δ standing-sitting	-0.5 ± 0.2	-0.3 ± 0.2	-0.3 ± 0.2	-0.2 ± 0.3	-0.1 ± 0.6	-0.2 ± 0.3
AR, Δ standing-sitting	-0.5 ± 0.2	-0.5 ± 0.1	-0.5 ± 0.2	-0.3 ± 0.6	-0.5 ± 0.4	-0.03 ± 0.3
LF/HF ratio						
AT, Δ standing-sitting	5.2 ± 1.5*	5.0 ± 1.6*	6.7 ± 1.5*	5.6 ± 1.4*	4.4 ± 0.7*	4.8 ± 0.6*
AR, Δ standing-sitting	4.8 ± 1.1*	5.0 ± 1.1*	5.1 ± 0.9*	4.3 ± 1.1*	4.9 ± 1.7*	5.1 ± 1.2*
APEN						
AT, Δ standing-sitting	0.01 ± 0.04	0.10 ± 0.06*	0.05 ± 0.05	0.03 ± 0.06	-0.02 ± 0.07	0.04 ± 0.07
AR, Δ standing-sitting	0.08 ± 0.04	0.08 ± 0.04	0.01 ± 0.05	0.06 ± 0.06	0.03 ± 0.04	0.03 ± 0.04
α_1						
AT, Δ standing-sitting	0.23 ± 0.05*	0.26 ± 0.06*	0.34 ± 0.06*	0.35 ± 0.05*	0.41 ± 0.08*	0.37 ± 0.06*
AR, Δ standing-sitting	0.26 ± 0.06*	0.29 ± 0.05*	0.34 ± 0.03*	0.32 ± 0.08*	0.37 ± 0.06*	0.41 ± 0.07*

Values are expressed as means ± SEM. (SDNN, standard deviation of all R-R intervals; SD1, standard deviation of data points perpendicular to the line of identity (Poincare plot) ;SD2, standard deviation of data points along the line of identity (Poincare plot); HF, high frequency power; LF, low frequency power; ln, natural logarithm; LF/HF ratio, absolute LF power divided by absolute HF power; APEN, approximate entropy; α_1, fractal scaling exponent in detrended fluctuation analysis.) * $P < 0.05$, difference between standing and sitting value. Only delta value is shown. *P<0.05, difference between sitting and standing value for AR and AT separate.

4.4 Correlation between short-term HRV and response in VO_{2max}

4.4.1 Correlation between ΔHF power and ΔVO_{2max}

A moderate, but significant correlation was found between change in VO_{2max} and change in short-term HF power on AR days (sitting posture) for both groups together (r= 0.531, p=0.011) and for SIT separate (r=0.615, p=0.033). For CT there was a moderate, but non-significant correlation (r=0.617, p=0.057).

Table 4-4-1: Correlation data for ΔHF power (p1-p3) and ΔVO_{2max} (pre-post) in SIT, CT and both groups together.

Correlation ΔHF power and ΔVO_{2max}	Type of correlation	Correlation Coefficient	Significance
SIT (n = 12)	Spearman rho	0.615*	0.033
CT (n = 10)	Pearson	0.617	0.057
Both groups (n =22)	Spearman rho	0.531*	0.011

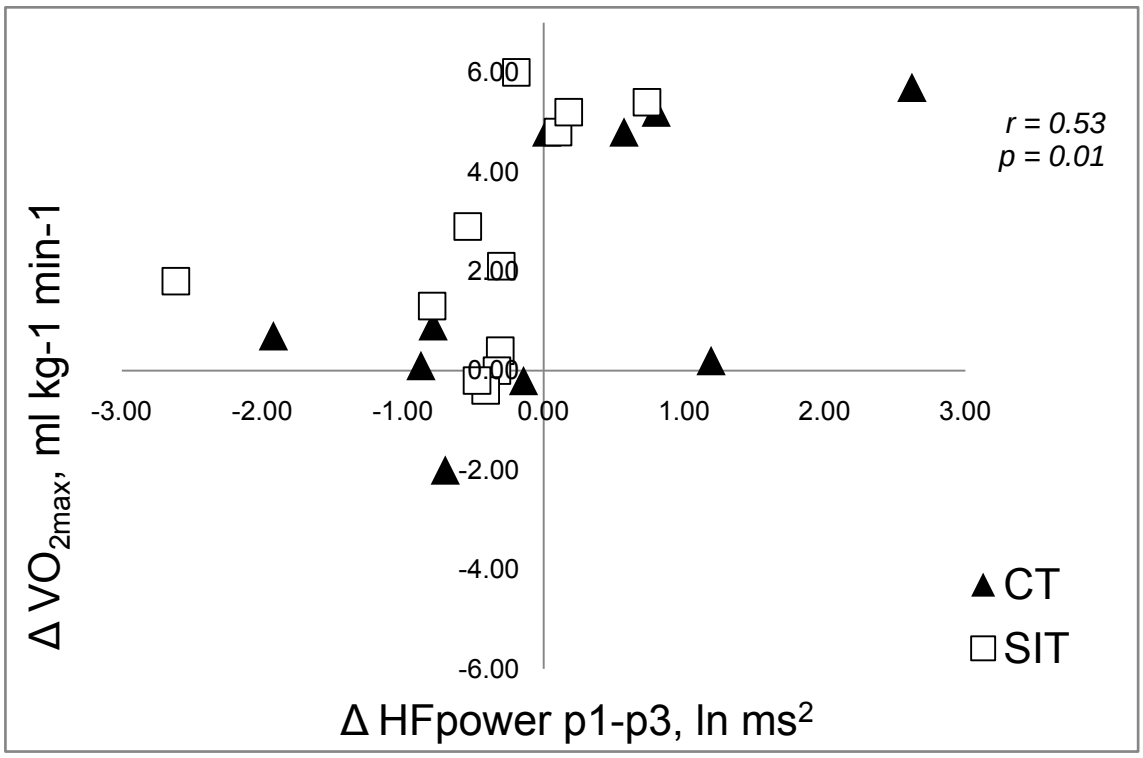


Figure 4-4-1: Correlation between the individual change (period 1 – period 3) in HF power (ln, ms^2) and the individual training response (ΔVO_{2max}). SIT (triangles) and CT (squares). The HF power values are from sitting posture.

4.4.2 Correlation between HF power in period 1 and ΔVO_{2max}

There were no correlation between HF power on AR days (sitting posture) in period 1(both groups) and changes in VO_{2max} (r= -0.22, p=0.32). HF power in period 1 was 7.2 ± 0.3 and 6.7 ± 0.6 ln, ms^2 for SIT and CT respectively.

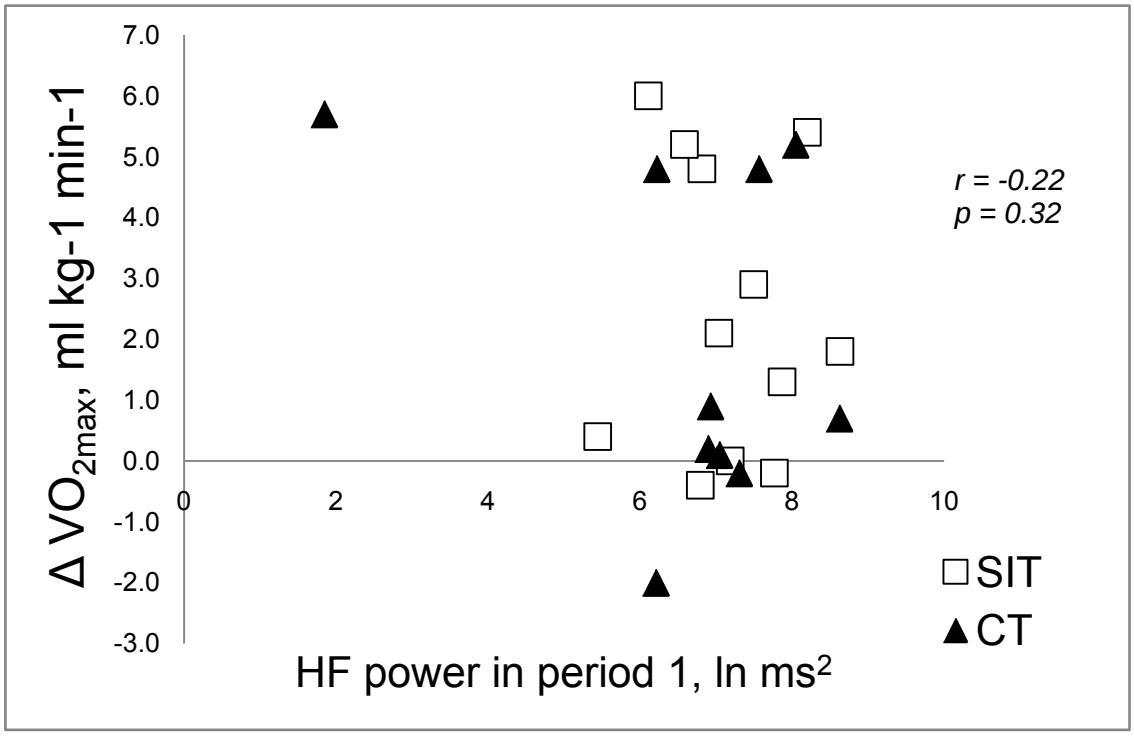


Figure 4-4-2: Correlation between the individual HF power (ln, ms^2) in the beginning of the training period (p1), and the individual training response (ΔVO_{2max}). The HF power values are means of sitting values on AR days in period 1.

4.5 Long-term HRV

For long-term 24-hours measurements there is a subgroup of 17 subjects, having both pre- and post recordings. Data are shown in table 4-5. There were no significant changes between pre, mid and post for either CT or SIT; except a decrease in SDNN for SIT from pre – to mid, and from pre – to post ($p < 0.05$). There were no differences between groups.

Table 4-5 Long-term 24 hours HRV

Table 4-5: Long-term HRV before (Pre), after 4 weeks (Mid) and after (Post) 8 weeks training in either SIT or CT. Data are presented from total 24 hours, and also separate for night hours (1 am – 5 am).

	SIT (n = 9)			CT (n = 8)		
	Pre	Mid[1]	Post	Pre	Mid[1]	Post
HR, beats/min						
24h	70.00 ± 2.47	67.14 ± 2.14	68.89 ± 2.12	73.50 ± 1.50	73.88 ± 2.29	73.88 ± 2.29
1 AM - 5 AM	54.22 ± 1.88	56.00 ± 1.88	55.67 ± 1.77	56.88 ± 2.03	59.43 ± 4.42	59.25 ± 2.96
SDNN, ms						
24h	205.9 ± 11.6	183.6 ± 15.1*	184.9 ± 12.1*	191.0 ± 12.6	191.7 ± 12.02	185.13 ± 12.3
SD1, ms						
24h	49.70 ± 4.85	51.01 ± 7.94	51.13 ± 6.98	44.54 ± 2.33	48.41 ± 2.65	43.95 ± 3.93
1 AM – 5 AM	74.30 ± 7.18	70.70 ± 7.77	72.87 ± 9.16	61.51 ± 6.91	71.73 ± 5.60	52.96 ± 6.62
SD2, ms						
24h	177.4 ± 14.1	172.6 ± 16.8	176.1 ± 14.0	163.8 ± 8.5	165.1 ± 8.0	166.00 ± 9.4
1 AM – 5 AM	168.1 ± 11.6	168.0 ± 12.7	164.1 ± 12.3	149.9 ± 11.6	170.2 ± 12.5	151.2 ± 13.6
HF, ln ms^2						
24h	7.44 ± 0.21	7.48 ± 0.32	7.39 ± 0.30	7.27 ± 0.12	7.45 ± 0.12	7.13 ± 0.22
1 AM – 5 AM	8.14 ± 0.21	8.04 ± 0.27	8.05 ± 0.29	7.71 ± 0.36	8.18 ± 0.17	7.38 ± 0.30
LF, ln ms^2						
24h	7.67 ± 0.17	7.76 ± 0.21	7.60 ± 0.17	7.73 ± 0.09	7.88 ± 0.07	7.74 ± 0.09
1 AM – 5 AM	8.05 ± 0.16	8.06 ± 0.19	7.91 ± 0.19	7.75 ± 0.22	8.07 ± 0.09	7.63 ± 0.18
LF/HF ratio						
24h	1.37 ± 0.21	1.49 ± 0.26	1.42 ± 0.28	1.64 ± 0.15	1.57 ± 0.15	2.04 ± 0.43
1 AM – 5 AM	1.00 ± 0.14	1.13 ± 0.22	0.96 ± 0.14	1.19 ± 0.26	1.00 ± 0.24	1.53 ± 0.37
VLF, ln ms^2						
24h	7.82 ± 0.16	8.08 ± 0.20	7.87 ± 0.21	7.74 ± 0.09	7.90 ± 0.11	7.87 ± 0.12
ULF, ln ms^2						
24h	9.99 ± 0.15	9.61 ± 0.17	9.67 ± 0.13	9.66 ± 0.18	9.64 ± 0.14	9.79 ± 0.14
APEN						
24h	1.18 ± 0.02	1.20 ± 0.05	1.21 ± 0.04	1.09 ± 0.09	1.13 ± 0.07	1.09 ± 0.08
1 AM – 5 AM	1.50 ± 0.04	1.44 ± 0.02	1.44 ± 0.04	1.32 ± 0.13	1.34 ± 0.09	1.25 ± 0.15
α_1						
24h	1.13 ± 0.04	1.13 ± 0.06	1.09 ± 0.06	1.17 ±0.03	1.16 ± 0.02	1.18 ± 0.04
1 AM – 5 AM	0.93 ± 0.05	0.95 ± 0.06	0.91 ± 0.06	0.98 ± 0.06	0.95 ± 0.05	1.06 ± 0.07

Values are expressed as means ± SEM. SDNN, standard deviation of all R-R intervals; SD1, standard deviation of data points perpendicular to the line of identity (Poincare plot) ;SD2, standard deviation of data points along the line of identity (Poincare plot); HF, high frequency power; LF, low frequency power; ln, natural logarithm; LF/HF ratio, absolute LF power divided by absolute HF power; APEN, approximate entropy; α_1, fractal scaling exponent in detrended fluctuation analysis.
* P < 0.05; comparisons between pre- and post and mid- an pre
# P < 0.05; comparisons between CT and SIT
[1] n = 7 for SIT and n = 7 for CT

4.5.1 Correlation between HF power during night hours and ΔVO_{2max}

There was no correlation between individual HF power during night hours at baseline (both groups) and individual change in VO_{2max} (r= 0.28, p=0.28). The mean HF power (1-5 am) were 8.1 ± 0.2 and 7.7 ± 0.4 (ln, ms^2) for SIT and CT respectively.

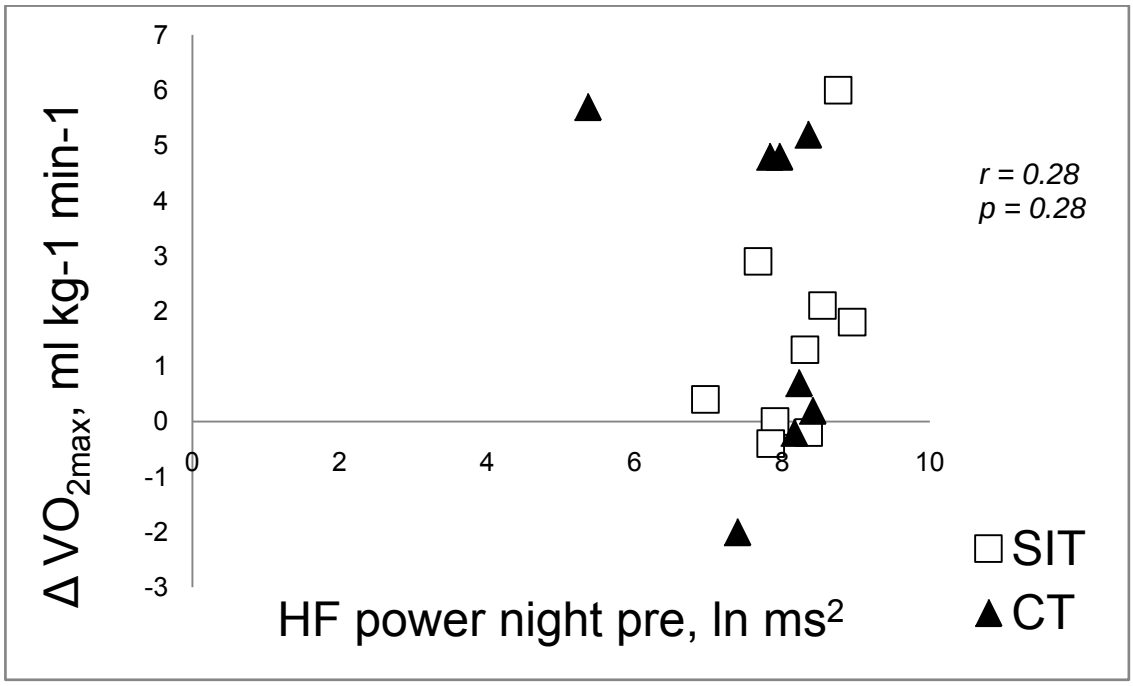


Figure 4-5-1: Correlation between HF power (ln, ms^2) during night hours (1 a.m. to 5 a.m.) before the training period (pre), and the individual training response (ΔVO_{2max}).

5.0 Discussion

5.1 Main findings

The first main finding in this study was that SIT showed a lower HRV after training days compared to AR days during the first two training weeks (p1), illustrated in figure 4-3 for both sitting and standing posture. For CT, no change in HRV after training days was observed in any period. The finding indicates a reduced cardiac vagal activity for SIT, but not for CT, which may reflect a delayed exercise recovery for SIT. However, during the last six weeks no differences in HRV was observed between AT and A R for SIT, despite a progressive increase in training load (number of sprint intervals per session). Instead, from period 1 to period 3 an actual decrease in HRV was observed for SIT, marked significantly in standing posture ($p < 0.05$ for SD1, HF- and LF power). It can be ar gued that the training load for SIT was too high, with insufficient recovery, compared to CT who maintained a similar HRV during the training intervention.

Furthermore we found that eight weeks of SIT or CT did not increase any HRV parameter in 24-hours HRV. High HRV is considered healthy, and previous studies have shown an increase in HRV after endurance training (Carter et al., 2003; Tulppo et al., 2003)., For CT, all HRV parameters remained similar after eight weeks of training with three sessions per week.

Although SIT and C T did not increase HRV, both groups increased VO_{2max} significantly. A moderate correlation was found between response in VO_{2max} and response in HRV (figure 4-4-1). Finally, baseline HRV did not correlate with training response as previously found by Hautala et al (2003).

The main findings will be further discussed.

5.2 Decrease in HRV for SIT after training days, but not for CT

The present study showed a significant lower HRV for SIT after training days compared to AR days in period 1. For CT, no difference was found between AR and AT. Five to six 30 seconds sprint intervals seemed to be enough to decrease all over HRV, showing an alteration of the autonomic nervous system towards a withdrawal of vagal activity and further a delay in exercise recovery. Compared to SIT, CT performed continuous running for 30-35 minutes at 70-80% of HR_{peak} during the two first training weeks, which did not influence HRV. Compared to CT, the mean HR of SIT was in the target exercise zone for CT, but the duration was shorter, pointing out the underestimation of high intensity exercise from HR monitoring (Seiler, 2010). In addition, regarding that none of the subjects in the present study had performed SIT before, the findings are not surprising, and it proves the valuable meaning of sufficient recovery in a training program for "beginners".

There are few other studies comparing effects of high intensity exercise bouts and moderate intensity exercise bouts on long-term HRV recovery. Most studies investigate the acute recovery, ranging from five minutes to four hours (Kaikkonen et al., 2008; Martinmaki & Rusko, 2008; Kaikkonen et al., 2007; Seiler et al., 2007). In addition most of the studies are done in laboratory conditions (Millar et al., 2009; Mourot, Bouhaddi, Tordi, Rouillon, & Regnard, 2004). No studies have investigated the day after-effect of short-duration sprint interval exercise. However, there are some interesting and relevant findings in the abovementioned studies which can support the present results.

The study of Millar and colleagues (2009) showed prolonged alterations in HRV after multiple Wingate tests (four 30 seconds all-out on a cycleergometer). In agreement with the present study, they found a significant increase in LF/HF ratio after exercise. Although Millar and colleagues measured only two hours after completion of the exercise in contrast to our day-after measurements, both findings indicate that sympathovagal balance is still altered, and a decrease in cardiac vagal activity.

It has been hypothesized that HRV recovery will be slower after interval exercise compared to continuous exercise (Kaikkonen et al., 2008; Kaikkonen

et al., 2007; Mourot et al., 2004). Our data could confirm this hypothesis, but others have rejected it.

In the study of Mourot et al. (2004), they found that 24- and 48 hours following a single bout of either constant training or interval training, HRV parameters was similar between exercise modes (Mourot et al., 2004). In the present study, altered HR and HRV were only found after interval training. However, the continuous exercise of Mourot et al. (2004) was performed at higher intensity compared to CT, and the interval training was mainly aerobic. Furthermore the same amount of total work was achieved by the two modes in the latter study. In the present study we did not measure the total work, and it is inaccurate to calculate from the HR distribution.

Kiviniemi and colleagues (2010) found a significant decrease in HRV (SD1) for female subjects after high intensity exercise, but not for men. The high intensity exercise session was aerobic, and subjects performed 30 minutes at ~85% of HR_{peak}. The decrease in SD1 observed for female subjects is in agreement with our study, as the majority of our subjects were females. However, when looking at the four male subjects in SIT alone, they also had a decrease in SD1 AT ($p < 0.05$) for both sitting and standing posture, showing a decrease from 26.1 ± 3.4 ms to 19.8 ± 4.0. Taken this together it may seem that sprint interval exercise promotes greater disturbances of the autonomic nervous system compared to high intensity aerobic exercise in a moderately fit population, irrespective of sex.

Hynynen and colleagues (2010) observed a significant lower HRV the night after a marathon compared to a control night (night following a resting day). In the same study they found that moderate exercise also gave a decrease in HRV the following night. The moderate endurance exercise in the latter study were ~50 minutes at light intensity (3±1 on scale 0-10) having an average HR of 72% of HR_{max}. This bout is quite similar to those used in our training study regarding duration and intensity; in fact the intensity was is a bit lower in the study of Hynynen et al,.(2010). They found a three beats higher HR, and a decrease in SDNN from 142 ± 16 to 126 ± 15 the night after moderate exercise. Regarding the fact that there are huge interindividual differences in HRV, the findings are perhaps not that meaningful at an individual level. However, the finding is

contradictory to the present study. CT showed no decrease in HRV the morning after a training day, compared to the morning after a resting day, not even during the last training weeks when the exercise duration were doubled from the first week (p3). It should be emphasized that lack of standardization and randomization of the interventions by Hynynen et al. (2010), can overestimate the finding of decreased HRV after moderate intensity exercise.

5.3 Changes in HRV from p1 to p3: decrease in HRV for SIT, but not CT.

The second main finding in this study, which we found surprising, was that the fluctuation in HRV for SIT between AT and AR days was gone during the last six weeks of training. Instead we observed a decrease in almost all HRV parameters in standing posture for SIT between period 1 and period 3. This decrease in HRV could be due to several factors, but it`s reasonable to believe that the increased training load and thereby insufficient recovery is the main cause. For CT, no changes were observed between AR and AT in any period, nor between periods, despite an increase in training load (e.g. duration). It can be argued that CT had a proper progression in exercise duration and a sufficient recovery between training sessions, shown as unaltered HRV.

To date, no other study has measured HRV during an eight weeks training intervention for moderately fit subjects. However, Pichot and colleagues (2000) followed HRV (nightly) during a four weeks training period for middle-distance runners. They found a progressive decrease in HRV during three weeks of training followed by a significant increase the fourth week. The training period was a part of their training cycle (a heavy period), and the fourth week was a recovery week (only light training sessions). The finding is in agreement with our observations, showing a tendency towards lower parasympathetic drive, and opposite higher sympathetic drive during and after a heavy training period. However, when adding a recovery week the autonomic balance returned towards vagal predominance (Pichot et al., 2000). Recovery weeks was also used by Carter and colleagues (2003) in recreational runners showing an increase in HRV and a decrease in HR at supine rest after 12 weeks of training. They used the fifth and the 12th week as taper periods. In the present study, SIT

did not have these recovery periods, and in addition they did not do any low intensity exercise except daily activities.

The effects of a large training volume and training load on HRV has also been studied in relation to symptoms of overtraining (Hynynen, Uusitalo, Konttinen, & Rusko, 2008; Hynynen, Uusitalo, Konttinen, & Rusko, 2006; Mourot et al., 2004; Uusitalo, Uusitalo, & Rusko, 2000). Mourot et al. (2004) compared a HRV profile between overtrained athletes, healthy athletes and sedentary subjects. They found the overtrained subjects had a marked predominance of sympathetic activity, both in supine and upright position (60 degrees) represented by lower LF/HF ratio (Mourot et al., 2004) In relation to our study, overtrained subjects had higher ratio in supine position (3.96 ± 5.7 SD) compared to SIT in both post recording during night hours (0.96 ± 0.14) and during sitting position in period 3 (3.4 ± 0.5, 3.8 ± 0.6 for AT and AR respectively).

5.4 Response in VO_{2max}

The training protocol was designed to improve performance for both SIT and CT. After eight weeks of training, both groups increased their VO_{2max} expressed as ml kg-1 min-1 by 4.6 and 4.1 %, for SIT and CT, respectively. However, since VO_{2max} is a widely discussed measure in terms of training response, it can be added to this thesis that subjects had a greater improvement in a shuttle run test (see Sigbjørn Litleskares master thesis, 2011).

Compared to other similar training studies, a greater increase in performance have been observed both after SIT (Macpherson, Hazell, Olver, Paterson, & Lemon, 2011; Mohr et al., 2007) and CT (Leicht et al., 2003; Tulppo et al., 2003; Hautala et al., 2003) However, in these studies the number of sessions per week was higher compared to the present protocol. The variation in initial fitness level can also influence the observed differences in training response. In the study of Hautala et al. (2003), untrained subjects exercised at moderate intensity six times per week, and had an average increase of 11% in VO_{2peak} after eight weeks of training, Training response is highly individualized, and other factors, such as genetics plays an important role (Bouchard & Rankinen, 2001). Our data supports this, and we observed a wide range in ΔVO_{2max},

having some subjects completing the training intervention, without any response at all (- 4%), while others received a great response (~15%).

In the training study of Kiviniemi and colleagues (2010), it was concluded that female subjects may benefit in fitness having less high intensity exercise. On average the female subjects in that study performed 1.8 ± 0.3 high intensity exercises per week, and a total of five sessions per week (Kiviniemi et al., 2010). However, there was no progression in the different sessions, in contrast to the present study, where the number of the sprint intervals increased progressively throughout the intervention period. In the last two weeks of our study, the training load was doubled compared to the first. The finding of Kiviniemi et al (2010) is an interesting finding compared to the present study, as we had most female subjects. Surprisingly then, when leaving males out of the analysis of VO_{2max}, the mean response values were the same for SIT, but not for CT. However, as we had too less subjects to detect sex differences, the results should not be interpreted as a significant finding.

5.5 Correlation between HRV and response in VO_{2max}

In the present study a moderate correlation (r= 0.531, p = 0.011) was found between training response, measured as ΔVO_{2max} and HRV response Δ(HF power ms^2, ln) in both groups together, and for SIT alone (r=0.615, p=0.033). For CT alone there was a near significant correlation (r=0.617, p= 0.057).

This latter is in agreement to the finding of Nummela et al (2010), showing a correlation between nocturnal ΔHF power (ln, ms^2) and change in maximal aerobic speed after four weeks of endurance training (r=0.482, p =0.042). As HF power is proposed to be a good measure of cardiac vagal activity, it can be assumed that an increase in the activity parasympathetic division to the heart may be related to improved performance.

In our study we found no correlation between nocturnal HRV and training response. For HF power (ln, ms^2) during night hours, the correlation coefficient was 0.280 and a p-value of 0.276. However, this can be due to a lower number of subjects and less pronounced responses.

5.6 HRV as a predictor for training response

The interest of HRV as a predictor for exercise and training response has fascinated physicians for many years, but the relationship has mostly been reported from cross-sectional studies (Borresen & Lambert, 2008; Buchheit et al., 2005; Aubert et al., 2001). These studies shows a higher resting HRV in trained subjects compared to untrained subjects. However, these findings can also be due to just random differences between to populations rather than an effect of exercise. On the contrary, no differences between athletes and non-athletes are also reported (Sacknoff, Gleim, Stachenfeld, & Coplan, 1994)

The question of whether cardiac autonomic function, measured as HRV correlates with response of aerobic training were investigated in an experimental study by Hautala et al (2003). In sedentary subjects they found a significant, moderate correlation between baseline HF power (ln, ms^2), measured during night hours, and ΔVO_{2peak} (r=0.52, p=0.001). In the present study no correlation was found between pre-training HRV (HF power during night hours and HF power in sitting posture on AR days) and response in VO_{2max} (figure 4-4-2 and 4-5-1). Compared to our study, Hautalas` subjects had the same duration of the training intervention, but they had a larger number of sessions per week (i.e. six sessions). In the study of Hautala, subjects were divided in quartiles based on the individual training response, having ten subjects in each quartile. When comparing HF power from these data to our data the initial night hours values in the present study were high (8.1 ± 0.2 for SIT and 7.7 ± 0.4 ln, ms^2 for CT) which indicate high cardiac vagal activity at rest (high HRV). In the study of Hautala et al. (2003), the high-responders showed a HF power (ln, ms^2) of 7.6 ± 0.5 during night hours whereas low-responders had 6.0 ± 0.7 ln, ms^2. To our knowledge, we are the first who investigated this relationship in moderately fit subjects (VO_{2max} > 45). It can be assumed that by using a larger (for CT) and individualized training volume (for SIT) we could have seen the observed findings by Hautala et al. (2003)

5.7 Practical implications of HRV analysis methods

In the present study, a wide spectrum of HRV analysis methods was used, and

in addition we both used short- and long-term recordings. For the practical availability of HRV, the use of simple short-term measurements is valuable. In addition, it can be believed that the use of standing posture is as appropriate as both sitting- and supine posture (Kiviniemi et al., 2010). In the present study, we found more marked results in standing posture compared to sitting posture, especially regarding changes from period 1 to period 3 (figure 4-3-1 and 4-3-2). The use of standing posture in HRV recordings may diminish or exclude the possible effects of saturation in HF oscillations of R-R intervals (Kiviniemi et al., 2004). In this matter, saturation reflects the inability of HRV measures to detect cardiac vagal influence at low HR levels (Kiviniemi et al., 2004).

In terms of evaluating exercise- and training response, the use of the Poincarè Plot is emphasized as an applicable method (Kiviniemi et al., 2010; Mourot et al., 2004). The visual shape of the Poincarè Plot can easily be understood by athletes and other training enthusiasts, where the “width” of the scattergram indicates HRV (SD1). A wide scattergram indicates high HRV, whereas a narrow shape indicates low HRV (see theory chapter, figure 2-6).

The day-to-day distribution of training intensity is proposed to be an important variable to balance positive and negative adapted stress and to avoid stagnation and overtraining (Achten & Jeukendrup, 2003). Today, tailored HR monitors can measure HRV, and simple HRV analysis can be done in commercial and free software (Niskanen & Tarvainen, 2008). It can be added to the literature that HRV may be a valuable tool for evaluating recovery during a period of sprint interval training in moderately fit subjects.

5.8 Methodological considerations

5.8.1 Sample size

There was a small sample size in this study (n=22), which may limit the ability to generalize results and interpreting findings to the science of HRV. The sample size were estimated using the response in VO_{2max} (the smallest possible change 1%, SEM 3%), from a formula of William Hopkins; “Sample Size for Magnitude-Based interferences” showing a number of 24 subjects as sufficient (Hopkins, Marshall, Batterham, & Hanin, 2009). The original plan was to include 32

subjects (in thoughts of possible drop-out), but due to a strict time schedule in the start up of the research project and having high fit subjects signing up, 29 subjects were included. Furthermore, seven subjects didn`t satisfy the HRV measurement criteria.

Despite these assumptions and facts, the small sample size can be strengthened by the use of both short- and long-term measurements and the inclusion of a large number of analysis methods.

5.8.2 Intraindividual variation in HRV

We found no change in resting HR measured during night hours, in which had not expected. However, many other factors are affecting HR, and the subjects had low initial values of HR indicating a high fitness level. From a methodological point of view it has been observed a five beats intra subject day-to-day variation in sleeping HR (Borresen & Lambert, 2008). Therefore, more than one night recording should be included for analysis, although this is not normal. For short-term results, it is even more intra individual day-to-day variation, and means of several days should be used as reference values (Kiviniemi et al., 2010; Kiviniemi et al., 2007).

5.8.3 Timing between training sessions short-term HRV measurements

One large limitation in this study is the time between the training sessions and the short-term HRV measurement. The time of the short-term measurements were standardized in the terms of awakening, and that it should be taken before noon. However, a large part of our subjects were students, and some of them didn`t have the routine to wake up at 7 a.m. every morning. So, changes in HRV could be due to differences in sleeping time.

As these measurements should be a part of their daily living, it would have been unnatural and impossible for the subjects to sleep the same amount of hours each night (and also the same hours). The second limitation in this matter is the time of preceding training session, performed either it the morning (between 8.30 and 11 a.m.) or evening (between 5 and 7 p.m.).

5.8.4 Number of short-term measurements

We had to arrange the data in periods, instead of comparing week by week. This was done because somebody had problems, or forgot to do the recordings, or did them the wrong day. This could have been avoided if the subjects had transferred their data each day of recording and sent them to us by email. However, we considered this as too time-consuming for the subjects. Instead we decided to collect all data, from both short-term recordings and training sessions. Because of large storage space on the Polar monitors, we did not transfer the data systematically every second week. We tried to control it by having the subjects writing a diary of their activity, both arranged training sessions and other activity. In the same scheme we asked the subjects to write down the days of short-term recordings. However, we did not manage to follow this up.

5.8.5 Design and training protocol

The study was designed as a parallel longitudinal intervention study with two different training groups. One limitation in this matter is the missing control group, in which would have isolated the possible training effects. We decided to drop it, mostly due to practical and ethical considerations. As we searched for those who wanted to start with regular endurance training, it would have felt bad to say to those subjects that they should not start regular exercise. However, by offering supervision after the end of training intervention, we could have justified the use of a control group.

When designing the training protocol, we did not manage to do a proper pilot study in the target population. However, we arranged a few sessions of SIT for fellow students, and thereby decided the final protocol. We discussed widely the warm up. For high intensity exercise, a long and progressive warm up is recommended (Aasen et al., 2005). In our study we used a short warm up protocol, but we added three strides to meet the demands of SIT. However, these strides were not included for CT, and furthermore the intensity of warm up for CT was lower. Then, we do not know the possible effect of warm up alone. In other studies, this is often unreported, as pointing out SIT as a time-efficient

training protocol (Macpherson et al., 2011). However, Mohr and colleagues (2007) reported the use of 20 minutes warm up.

It was hard to give instruction on intensity for SIT. As we were afraid that "all-out" exercise would have provoked injuries, we chose near maximal effort. The subjects rated the intervals as very- to extremely hard on the Borg scale. For CT, it was hard to run at the target intensity. As used in other studies, the intensity of 70-80% of $HR_{max.}$ For CT subjects it was hard to not precede the target exercise zone, and thereby having the mean intensity of 79% of $HR_{peak.}$ It can be questioned whether HR intensity zones are suitable for sedentary and moderately fit subjects, or if other limits should be considered. According to ACSM, exercising at 70-80% of HR_{max} is defined as "hard". The subjects in the present study described the exercise as light- to somewhat hard, and our observations supported this rating.

Finally we chose to increase training load (duration for CT, number of sprint intervals for SIT) rather than training volume. We wanted to control the sessions as far as it was possible, and therefore we thought that more than three sessions per week would have increased the dropout rate. In addition we arranged the sessions outside, in order to make the finding more transferable to real life conditions.

6.0 Conclusions

1. During eight weeks of SIT, short-term HRV is lower in mornings after training days, compared to mornings after resting days in the first two training weeks, but not during eight weeks of CT, indicating a withdrawal of vagal activity after sprint interval exercise, but not after moderate intensity exercise. As the number of sprint intervals in each session increases weekly, HRV decreases when measured after resting days. In CT, increasing exercise duration in the training period does not affect short-term HRV after training or resting days.

2. Eight weeks of SIT or CT with three sessions per week do not increase long-term 24-hours HRV in moderately fit young women and men.

3.

a) Baseline HRV, measured during night hours before training interventions or measured in short-term on AR days in the beginning, do not correlate with the response in VO_{2max}.

b) Both SIT and CT improves VO_{2max} significantly, and this response correlates with the change in short-term HRV from the beginning to the end of an eight weeks training period.

It can be concluded that during the first period of an eight weeks sprint interval training intervention, heart rate variability is lower after training days compared to after resting days. Furthermore, a lower heart rate variability after resting days appears at the end of the sprint interval training period, indicating a training overload. On the contrary, an eight weeks period of aerobic continuously moderate intensity training does decrease HRV after training days compared to after resting days. Although sprint interval training and continuously moderate intensity training with three sessions per week are efficient to increase maximal oxygen consumption, our data do not support responses in heart rate variability in moderately fit young women and men.

Norwegian summary

Hensikt: Denne studien ble designet for å undersøke effekten sprintintervalltrening (SIT) og langkjøring med moderat intensitet (CT) på hjertefrekvensvariasjon (HRV) hos friske, moderat trente unge menn og kvinner; før, underveis og etter en åtte ukers intervensjonsperiode.

Metode: 22 forsøkspersoner ble randomisert i to grupper, matchet på kjønn, vekt og maksimalt oksygenopptak (VO_{2maks}). Begge grupper, SIT (fire menn, åtte kvinner) og CT (tre menn, syv kvinner), gjennomførte tre løpeøkter per uke med minimum en dags hvile mellom øktene. Alle økter ble gjennomført ute, i nærområdet ved Norges idrettshøgskole. SIT startet i uke 1 med fem sprintintervaller a 30-sekunder, og økte med et intervall per uke (10 i uke 8). Sprintintervallene ble gjennomført på en liten grusvei med svak stigning, estimert til 5-8%. CT gjennomførte 30-60 minutters langkjøringsøkter på en intensitet mellom 70 og 80% av maksimal hjertefrekvens (HR_{peak})med fem minuttets økning i varighet per uke.

Underveis i treningsperioden målte forsøkspersonene korttids-HRV (fem minutter sittende etterfulgt av fem minutter stående) to ganger per uke, en gang etter en treningsdag (AT) og en gang etter en hviledag (AR). Målingene ble gjennomført tidlig om morgenen (før frokost), og ble gjort ved bruk av Polar pulsklokke og pulsbånd (Polar RS800CX, Polar Electro Oy, Kempele, Finland). Pulsbåndet fanger opp elektriske signaler ned mot 1 millisekund (ms), og måler tiden mellom hvert hjerteslag (tiden mellom hver R-topp i QRS-komplekset i et EKG i ms). Før, midtveis og etter treningsperioden ble det gjennomført en test av VO_{2maks} og 24-timers måling av HRV. Målingene av HRV (R-R data) ble lagret i pulsklokka for videre analyse i et HRV-program (Hearts software). HRV ble analysert i tids-domene (mean HR/R-R OG SDNN) frekvens-domene (LF power, HF power og LF/HF ratio) og med ikke-lineær analyse (SD1 og SD2 fra Poincaré Plot, α_1 og ApEn). Data er presentert som gjennomsnitt ± SEM. Korttidsdata ble analysert for AR og AT separat, og gjennomsnittsverdiene ble videre samlet i tre underperioder (Periode 1: uke 0-2, periode 2: uke 3-5, periode 3: uke 6-8). 24-timersmålingene ble analysert for hele døgnet, og separat for nattetimer (klokka 01.00 til 05.00).

Resultater: For SIT i periode 1 så vi en signifikant reduksjon i HRV etter en treningsdag, sammenlignet med etter en hviledag, men ikke for CT. I de seks siste treningsukene ble det ikke funnet forskjell mellom HRV etter trening og etter hvile for noen av gruppene. For SIT ble det observert en gradvis reduksjon i HRV utover i treningsperioden, markert signifikant i stående stilling. 24-timers (n=17) data støtter dette funnet, og en signifikant reduksjon i SDNN ble funnet etter endt treningsperiode. For CT så vi ingen forandring i HRV fra før til etter intervensjonen. Videre fant vi en signifikant korrelasjon mellom forandring i HRV på AR-dager og fremgang i $VO_{2maks,}$ for begge grupper samlet og for SIT separat.

Konklusjon: Det ser ut til at en underveis i en åtte ukers periode med SIT, er HRV lavere morgenen etter en treningsdag sammenlignet med etter en hviledag, og lavere etter hviledager på slutten av treningsperioden sammenlignet med i starten. Dette kan ikke sies om CT, som ikke gir noe utslag i HRV, selv ikke etter at varigheten på øktene er doblet opp til en time de to siste treningsukene. Til tross for at både SIT og CT viste seg å være effektive treningsformer for å øke VO_{2maks}, så støtter ikke våre data en økning i HRV hos moderat trente kvinner og menn.

Nøkkelord: hjertefrekvensvariasjon, sprintintervalltrening, moderat intensitet, kontinuerlig løpetrening, langkjøring

References

Aasen, S. B., Frøyd, C., Madsen, Ø., Sæterdal, R., Tønnesen, E., & Wisnes, A. (2005). *Utholdenhetstrening - trening som gir resultater*. Olympiatoppen, Akilles.

Achten, J. & Jeukendrup, A. E. (2003). Heart rate monitoring: applications and limitations. *Sports Med., 33,* 517-538.

ACSM (1998). American College of Sports Medicine Position Stand. The recommended quantity and quality of exercise for developing and maintaining cardiorespiratory and muscular fitness, and flexibility in healthy adults. *Med.Sci.Sports Exerc., 30,* 975-991.

Akselrod, S., Gordon, D., Madwed, J. B., Snidman, N. C., Shannon, D. C., & Cohen, R. J. (1985). Hemodynamic regulation: investigation by spectral analysis. *Am.J.Physiol, 249,* H867-H875.

Akselrod, S., Gordon, D., Ubel, F. A., Shannon, D. C., Berger, A. C., & Cohen, R. J. (1981). Power spectrum analysis of heart rate fluctuation: a quantitative probe of beat-to-beat cardiovascular control. *Science, 213,* 220-222.

al-Ani, M., Munir, S. M., White, M., Townend, J., & Coote, J. H. (1996). Changes in R-R variability before and after endurance training measured by power spectral analysis and by the effect of isometric muscle contraction. *Eur.J.Appl.Physiol Occup.Physiol, 74,* 397-403.

Aubert, A. E., Beckers, F., & Ramaekers, D. (2001). Short-term heart rate variability in young athletes. *J.Cardiol., 37 Suppl 1,* 85-88.

Aubert, A. E., Seps, B., & Beckers, F. (2003). Heart rate variability in athletes. *Sports Med., 33,* 889-919.

Berntson, G. G., Bigger, J. T., Jr., Eckberg, D. L., Grossman, P., Kaufmann, P. G., Malik, M. et al. (1997). Heart rate variability: origins, methods, and interpretive caveats. *Psychophysiology, 34,* 623-648.

Berntson, G. G., Cacioppo, J. T., & Quigley, K. S. (1993). Cardiac psychophysiology and autonomic space in humans: empirical perspectives and conceptual implications. *Psychol.Bull., 114,* 296-322.

Borg, G. A. (1982). Psychophysical bases of perceived exertion. *Med.Sci.Sports Exerc., 14,* 377-381.

Borresen, J. & Lambert, M. I. (2008). Autonomic control of heart rate during and after exercise : measurements and implications for monitoring training status. *Sports Med., 38,* 633-646.

Bouchard, C. & Rankinen, T. (2001). Individual differences in response to regular physical activity. *Med.Sci.Sports Exerc., 33,* S446-S451.

Buchheit, M., Simon, C., Charloux, A., Doutreleau, S., Piquard, F., & Brandenberger, G. (2005). Heart rate variability and intensity of habitual physical activity in middle-aged persons. *Med.Sci.Sports Exerc., 37,* 1530-1534.

Burgomaster, K. A., Heigenhauser, G. J., & Gibala, M. J. (2006). Effect of short-term sprint interval training on human skeletal muscle carbohydrate metabolism during exercise and time-trial performance. *J.Appl.Physiol, 100,* 2041-2047.

Burgomaster, K. A., Howarth, K. R., Phillips, S. M., Rakobowchuk, M., MacDonald, M. J., McGee, S. L. et al. (2008). Similar metabolic adaptations during exercise after low volume sprint interval and traditional endurance training in humans. *J.Physiol, 586,* 151-160.

Burgomaster, K. A., Hughes, S. C., Heigenhauser, G. J., Bradwell, S. N., & Gibala, M. J. (2005). Six sessions of sprint interval training increases muscle oxidative potential and cycle endurance capacity in humans. *J.Appl.Physiol, 98,* 1985-1990.

Carter, J. B., Banister, E. W., & Blaber, A. P. (2003). The effect of age and gender on heart rate variability after endurance training. *Med.Sci.Sports Exerc., 35,* 1333-1340.

Catai, A. M., Chacon-Mikahil, M. P., Martinelli, F. S., Forti, V. A., Silva, E., Golfetti, R. et al. (2002). Effects of aerobic exercise training on heart rate variability during wakefulness and sleep and cardiorespiratory responses of young and middle-aged healthy men. *Braz.J.Med.Biol.Res., 35,* 741-752.

Eckberg, D. L. (1997). Sympathovagal balance: a critical appraisal. *Circulation, 96,* 3224-3232.

Eckberg, D. L. (2003). The human respiratory gate. *J.Physiol, 548,* 339-352.

Ewing, D. J., Borsey, D. Q., Bellavere, F., & Clarke, B. F. (1981). Cardiac autonomic neuropathy in diabetes: comparison of measures of R-R interval variation. *Diabetologia, 21,* 18-24.

Ewing, D. J., Campbell, I. W., Murray, A., Neilson, J. M., & Clarke, B. F. (1978). Immediate heart-rate response to standing: simple test for autonomic neuropathy in diabetes. *Br.Med.J., 1,* 145-147.

Gamelin, F. X., Berthoin, S., & Bosquet, L. (2006). Validity of the polar S810 heart rate monitor to measure R-R intervals at rest. *Med.Sci.Sports Exerc., 38,* 887-893.

Gibala, M. J., Little, J. P., van, E. M., Wilkin, G. P., Burgomaster, K. A., Safdar, A. et al. (2006). Short-term sprint interval versus traditional endurance training: similar initial adaptations in human skeletal muscle and exercise performance. *J.Physiol, 575,* 901-911.

Goldberger, A. L. (1992). Fractal mechanisms in the electrophysiology of the heart. *IEEE Eng Med.Biol.Mag., 11,* 47-52.

Goldberger, A. L. (1996). Non-linear dynamics for clinicians: chaos theory, fractals, and complexity at the bedside. *Lancet, 347,* 1312-1314.

Hall, J. E. & Guyton, A. C. (2011). *Textbook of Medical Physiology*. (12 ed.) Saunders Elsevier.

Hautala, A., Tulppo, M. P., Makikallio, T. H., Laukkanen, R., Nissila, S., & Huikuri, H. V. (2001). Changes in cardiac autonomic regulation after prolonged maximal exercise. *Clin.Physiol, 21,* 238-245.

Hautala, A. J., Makikallio, T. H., Kiviniemi, A., Laukkanen, R. T., Nissila, S., Huikuri, H. V. et al. (2003). Cardiovascular autonomic function correlates with the response to aerobic training in healthy sedentary subjects. *Am.J.Physiol Heart Circ.Physiol, 285,* H1747-H1752.

Hautala, A. J., Makikallio, T. H., Kiviniemi, A., Laukkanen, R. T., Nissila, S., Huikuri, H. V. et al. (2004). Heart rate dynamics after controlled training followed by a home-based exercise program. *Eur.J.Appl.Physiol, 92,* 289-297.

Hayano, J., Yasuma, F., Okada, A., Mukai, S., & Fujinami, T. (1996). Respiratory sinus arrhythmia. A phenomenon improving pulmonary gas exchange and circulatory efficiency. *Circulation, 94,* 842-847.

Hopkins, W. G., Marshall, S. W., Batterham, A. M., & Hanin, J. (2009). Progressive statistics for studies in sports medicine and exercise science. *Med.Sci.Sports Exerc., 41,* 3-13.

Huikuri, H. V., Makikallio, T. H., Peng, C. K., Goldberger, A. L., Hintze, U., & Moller, M. (2000). Fractal correlation properties of R-R interval dynamics and mortality in patients with depressed left ventricular function after an acute myocardial infarction. *Circulation, 101,* 47-53.

Huikuri, H. V., Niemela, M. J., Ojala, S., Rantala, A., Ikaheimo, M. J., & Airaksinen, K. E. (1994). Circadian rhythms of frequency domain measures of heart rate variability in healthy subjects and patients with coronary artery disease. Effects of arousal and upright posture. *Circulation, 90,* 121-126.

Huikuri, H. V., Seppanen, T., Koistinen, M. J., Airaksinen, J., Ikaheimo, M. J., Castellanos, A. et al. (1996). Abnormalities in beat-to-beat dynamics of heart rate before the spontaneous onset of life-threatening ventricular tachyarrhythmias in patients with prior myocardial infarction. *Circulation, 93,* 1836-1844.

Huikuri, H. V., Valkama, J. O., Airaksinen, K. E., Seppanen, T., Kessler, K. M., Takkunen, J. T. et al. (1993). Frequency domain measures of heart rate variability before the onset of nonsustained and sustained ventricular tachycardia in patients with coronary artery disease. *Circulation, 87,* 1220-1228.

Hynynen, E., Uusitalo, A., Konttinen, N., & Rusko, H. (2006). Heart rate variability during night sleep and after awakening in overtrained athletes. *Med.Sci.Sports Exerc., 38,* 313-317.

Hynynen, E., Uusitalo, A., Konttinen, N., & Rusko, H. (2008). Cardiac autonomic responses to standing up and cognitive task in overtrained athletes. *Int.J.Sports Med., 29,* 552-558.

Hynynen, E., Vesterinen, V., Rusko, H., & Nummela, A. (2010). Effects of Moderate and Heavy Endurance Exercise on Nocturnal HRV. *Int.J.Sports Med.*.

Jensen-Urstad, K., Storck, N., Bouvier, F., Ericson, M., Lindblad, L. E., & Jensen-Urstad, M. (1997). Heart rate variability in healthy subjects is related to age and gender. *Acta Physiol Scand., 160,* 235-241.

Kaikkonen, P., Hynynen, E., Mann, T., Rusko, H., & Nummela, A. (2010). Can HRV be used to evaluate training load in constant load exercises? *Eur.J.Appl.Physiol, 108,* 435-442.

Kaikkonen, P., Nummela, A., & Rusko, H. (2007). Heart rate variability dynamics during early recovery after different endurance exercises. *Eur.J.Appl.Physiol, 102,* 79-86.

Kaikkonen, P., Rusko, H., & Martinmaki, K. (2008). Post-exercise heart rate variability of endurance athletes after different high-intensity exercise interventions. *Scand.J.Med.Sci.Sports, 18,* 511-519.

Kiviniemi, A. M., Hautala, A. J., Kinnunen, H., Nissila, J., Virtanen, P., Karjalainen, J. et al. (2010). Daily exercise prescription on the basis of HR variability among men and women. *Med.Sci.Sports Exerc., 42,* 1355-1363.

Kiviniemi, A. M., Hautala, A. J., Kinnunen, H., & Tulppo, M. P. (2007). Endurance training guided individually by daily heart rate variability measurements. *Eur.J.Appl.Physiol, 101,* 743-751.

Kiviniemi, A. M., Hautala, A. J., Seppanen, T., Makikallio, T. H., Huikuri, H. V., & Tulppo, M. P. (2004). Saturation of high-frequency oscillations of R-R intervals in healthy subjects and patients after acute myocardial infarction during ambulatory conditions. *Am.J.Physiol Heart Circ.Physiol, 287,* H1921-H1927.

Kleiger, R. E., Miller, J. P., Bigger, J. T., Jr., & Moss, A. J. (1987). Decreased heart rate variability and its association with increased mortality after acute myocardial infarction. *Am.J.Cardiol., 59,* 256-262.

Leicht, A. S., Allen, G. D., & Hoey, A. J. (2003). Influence of intensive cycling training on heart rate variability during rest and exercise. *Can.J.Appl.Physiol, 28,* 898-909.

Levine, B. D. (2008). .VO2max: what do we know, and what do we still need to know? *J.Physiol, 586,* 25-34.

Loimaala, A., Huikuri, H., Oja, P., Pasanen, M., & Vuori, I. (2000). Controlled 5-mo aerobic training improves heart rate but not heart rate variability or baroreflex sensitivity. *J.Appl.Physiol, 89,* 1825-1829.

MacDougall, J. D., Hicks, A. L., MacDonald, J. R., McKelvie, R. S., Green, H. J., & Smith, K. M. (1998). Muscle performance and enzymatic adaptations to sprint interval training. *J.Appl.Physiol, 84,* 2138-2142.

Macpherson, R. E., Hazell, T. J., Olver, T. D., Paterson, D. H., & Lemon, P. W. (2011). Run sprint interval training improves aerobic performance but not maximal cardiac output. *Med.Sci.Sports Exerc., 43,* 115-122.

Malliani, A., Pagani, M., Lombardi, F., & Cerutti, S. (1991). Cardiovascular neural regulation explored in the frequency domain. *Circulation, 84,* 482-492.

Martinmaki, K. & Rusko, H. (2008). Time-frequency analysis of heart rate variability during immediate recovery from low and high intensity exercise. *Eur.J.Appl.Physiol, 102,* 353-360.

McArdle, W. D., Katch, F. I., & Katch, V. L. (2007). *Exercise physiology: Energy, Nutrition and Human Performance.* (6th ed.) Baltimore: Lippincott Wiliams & Wilkins.

Melanson, E. L. & Freedson, P. S. (2001). The effect of endurance training on resting heart rate variability in sedentary adult males. *Eur.J.Appl.Physiol, 85,* 442-449.

Millar, P. J., Rakobowchuk, M., McCartney, N., & MacDonald, M. J. (2009). Heart rate variability and nonlinear analysis of heart rate dynamics following single and multiple Wingate bouts. *Appl.Physiol Nutr.Metab, 34,* 875-883.

Mohr, M., Krustrup, P., Nielsen, J. J., Nybo, L., Rasmussen, M. K., Juel, C. et al. (2007). Effect of two different intense training regimens on skeletal muscle ion transport proteins and fatigue development. *Am.J.Physiol Regul.Integr.Comp Physiol, 292,* R1594-R1602.

Mourot, L., Bouhaddi, M., Perrey, S., Cappelle, S., Henriet, M. T., Wolf, J. P. et al. (2004). Decrease in heart rate variability with overtraining: assessment by the Poincare plot analysis. *Clin.Physiol Funct.Imaging, 24,* 10-18.

Mourot, L., Bouhaddi, M., Tordi, N., Rouillon, J. D., & Regnard, J. (2004). Short- and long-term effects of a single bout of exercise on heart rate variability: comparison between constant and interval training exercises. *Eur.J.Appl.Physiol, 92,* 508-517.

Nakamura, Y., Yamamoto, Y., & Muraoka, I. (1993). Autonomic control of heart rate during physical exercise and fractal dimension of heart rate variability. *J.Appl.Physiol, 74,* 875-881.

Niemela, T. H., Kiviniemi, A. M., Hautala, A. J., Salmi, J. A., Linnamo, V., & Tulppo, M. P. (2008). Recovery pattern of baroreflex sensitivity after exercise. *Med.Sci.Sports Exerc., 40,* 864-870.

Niskanen, J-P and Tarvainen, M. P (2008). Kubios HRV version 2.0: User's guide. http://kubios.uku.fi

Nummela, A., Hynynen, E., Kaikkonen, P., & Rusko, H. (2010). Endurance performance and nocturnal HRV indices. *Int.J.Sports Med., 31,* 154-159.

O'Leary, D. D., Kimmerly, D. S., Cechetto, A. D., & Shoemaker, J. K. (2003). Differential effect of head-up tilt on cardiovagal and sympathetic baroreflex sensitivity in humans. *Exp.Physiol, 88,* 769-774.

Peltola, M. (2010). *Analysis of heart rate variability from 24-hour ambulatory electrocardiographic recordings.* Academic dissertaion University of Oulu.

Peng, C. K., Havlin, S., Stanley, H. E., & Goldberger, A. L. (1995). Quantification of scaling exponents and crossover phenomena in nonstationary heartbeat time series. *Chaos., 5,* 82-87.

Perini, R., Fisher, N., Veicsteinas, A., & Pendergast, D. R. (2002). Aerobic training and cardiovascular responses at rest and during exercise in older men and women. *Med.Sci.Sports Exerc., 34,* 700-708.

Pichot, V., Roche, F., Denis, C., Garet, M., Duverney, D., Costes, F. et al. (2005). Interval training in elderly men increases both heart rate variability and baroreflex activity. *Clin.Auton.Res., 15,* 107-115.

Pichot, V., Roche, F., Gaspoz, J. M., Enjolras, F., Antoniadis, A., Minini, P. et al. (2000). Relation between heart rate variability and training load in middle-distance runners. *Med.Sci.Sports Exerc., 32,* 1729-1736.

Pikkujamsa, S. M., Makikallio, T. H., Airaksinen, K. E., & Huikuri, H. V. (2001). Determinants and interindividual variation of R-R interval dynamics in healthy middle-aged subjects. *Am.J.Physiol Heart Circ.Physiol, 280,* H1400-H1406.

Polar Electro (2011). Polar sport zones for running. Retrieved 27-4-0011, from http://www.polar.fi/en/training_with_polar/training_articles/maximize_performance/running/polar_sport_zones_for_running

Ramaekers, D., Ector, H., Aubert, A. E., Rubens, A., & Van de Werf, F. (1998). Heart rate variability and heart rate in healthy volunteers. Is the female autonomic nervous system cardioprotective? *Eur.Heart J., 19,* 1334-1341.

Robinson, B. F., Epstein, S. E., Beiser, G. D., & Braunwald, E. (1966). Control of heart rate by the autonomic nervous system. Studies in man on the interrelation between baroreceptor mechanisms and exercise. *Circ.Res., 19,* 400-411.

Ruha, A., Sallinen, S., & Nissila, S. (1997). A real-time microprocessor QRS detector system with a 1-ms timing accuracy for the measurement of ambulatory HRV. *IEEE Trans.Biomed.Eng, 44,* 159-167.

Sacknoff, D. M., Gleim, G. W., Stachenfeld, N., & Coplan, N. L. (1994). Effect of athletic training on heart rate variability. *Am.Heart J., 127,* 1275-1278.

Salo, M. A., Huikuri, H. V., & Seppanen, T. (2001). Ectopic beats in heart rate variability analysis: effects of editing on time and frequency domain measures. *Ann.Noninvasive.Electrocardiol., 6,* 5-17.

Savin, W. M., Davidson, D. M., & Haskell, W. L. (1982). Autonomic contribution to heart rate recovery from exercise in humans. *J.Appl.Physiol, 53,* 1572-1575.

Schmidt, G., Malik, M., Barthel, P., Schneider, R., Ulm, K., Rolnitzky, L. et al. (1999). Heart-rate turbulence after ventricular premature beats as a predictor of mortality after acute myocardial infarction. *Lancet, 353,* 1390-1396.

Seals, D. R. & Chase, P. B. (1989). Influence of physical training on heart rate variability and baroreflex circulatory control. *J.Appl.Physiol, 66,* 1886-1895.

Seiler, S. (2010). What is best practice for training intensity and duration distribution in endurance athletes? *Int.J.Sports Physiol Perform., 5,* 276-291.

Seiler, S., Haugen, O., & Kuffel, E. (2007). Autonomic recovery after exercise in trained athletes: intensity and duration effects. *Med.Sci.Sports Exerc., 39,* 1366-1373.

Stein, P. K., Ehsani, A. A., Domitrovich, P. P., Kleiger, R. E., & Rottman, J. N. (1999). Effect of exercise training on heart rate variability in healthy older adults. *Am.Heart J., 138,* 567-576.

Task Force of the ESC and NASP (1996). Heart rate variability. Standards of measurement, physiological interpretation, and clinical use. Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology. *Eur.Heart J., 17,* 354-381.

Taylor, J. A., Myers, C. W., Halliwill, J. R., Seidel, H., & Eckberg, D. L. (2001). Sympathetic restraint of respiratory sinus arrhythmia: implications for vagal-cardiac tone assessment in humans. *Am.J.Physiol Heart Circ.Physiol, 280,* H2804-H2814.

Tulppo, M. P., Hautala, A. J., Makikallio, T. H., Laukkanen, R. T., Nissila, S., Hughson, R. L. et al. (2003). Effects of aerobic training on heart rate dynamics in sedentary subjects. *J.Appl.Physiol, 95,* 364-372.

Tulppo, M. P., Makikallio, T. H., Seppanen, T., Laukkanen, R. T., & Huikuri, H. V. (1998). Vagal modulation of heart rate during exercise: effects of age and physical fitness. *Am.J.Physiol, 274,* H424-H429.

Tulppo, M. P., Makikallio, T. H., Takala, T. E., Seppanen, T., & Huikuri, H. V. (1996). Quantitative beat-to-beat analysis of heart rate dynamics during exercise. *Am.J.Physiol, 271,* H244-H252.

Umetani, K., Singer, D. H., McCraty, R., & Atkinson, M. (1998). Twenty-four hour time domain heart rate variability and heart rate: relations to age and gender over nine decades. *J.Am.Coll.Cardiol., 31,* 593-601.

Uusitalo, A. L., Uusitalo, A. J., & Rusko, H. K. (2000). Heart rate and blood pressure variability during heavy training and overtraining in the female athlete. *Int.J.Sports Med., 21,* 45-53.

Verheyden, B., Eijnde, B. O., Beckers, F., Vanhees, L., & Aubert, A. E. (2006). Low-dose exercise training does not influence cardiac autonomic control in healthy sedentary men aged 55-75 years. *J.Sports Sci., 24,* 1137-1147.

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

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Dato: 12.07.10
Deres ref.:
Vår ref.: 2010/1567-1

2010/1567-1 Sammenligning av effekten av langkjøring og sprint-intervalltrening på insulinsensitivitet, metabolisme og hjertefrekvens

Forskningsansvarlig: Norges Idrettshøgskole
Prosjektleder: Jørgen Jensen

Formålet med studien er å sammenligne effekten av sprint-intervall og kontinuerlig løpetrening på flere fysiologiske parametre, samt prestasjon. Disse parametrene er maksimalt oksygenopptak, prestasjon, metabolisme, insulinsensitivitet og variasjon i hjertefrekvens. Det skal inkluderes 32 forsøkspersoner i prosjektet, fortrinnsvis 16 menn og 16 damer. Videre søkes det om opprettelse av en spesifikk forskningsbiobank for oppbevaring av blodprøver.

Vi viser til søknad om forhåndsgodkjenning av ovennevnte forskningsprosjekt. Søknaden ble behandlet av Regional forskningsetisk komité for medisinsk og helsefaglig forskningsetikk (REK sør-øst D) i møtet 17.06.2010. Søknaden er vurdert i henhold til lov av 20. juni 2008 nr. 44, om medisinsk og helsefaglig forskning (helseforskningsloven), med tilhørende forskrift om organisering av medisinsk og helsefaglig forskning av 1. juli 2009 nr 0955.

Komiteens vurdering

Komiteen konstaterer at deltagelse i prosjektet forutsetter betydelig bruk av tid for den enkelte. I dette tilfellet er imidlertid forskningsdeltakerne antatt friske personer og komiteen har ingen forskningsetiske innvendinger mot gjennomføringen av prosjektet.

Det søkes om opprettelse av forskningsbiobank for blodprøver som tas i forskningsprosjektet. Komiteen ønsker en tilbakemelding på om det i dette tilfellet søkes om opprettelse av en generell forskningsbiobank.

Vedtak

Med hjemmel i helseforskningsloven § 10, jfr. forskingsetikkloven § 4 godkjenner komiteen at prosjektet gjennomføres i samsvar med det som framgår av søknaden.

REK godkjenner opprettelse av forskningsbiobank "NIH-JJ-NSES1-2010". Melding om godkjenningen er sendt Biobankregisteret.

- Ansvarshavende er professor Jørgen Jensen.
- Materiale som inngår i forskningsbiobanken er blodprøver.
- Bruk av det humant biologiske materialet kan bare skje etter prosjektdeltakernes samtykke og er begrenset til hva som fremgår av informasjonsskrivet.
- Forskningsbiobankens varighet er satt til 31.12.2015.

Dersom forskningsbiobanken opphører, nedlegges eller overtas av andre, skal det søkes REK om tillatelse, jfr. helseforskningsloven § 30.

Godkjenningen av prosjektet gjelder til 31.12.2011. Av dokumentasjonshensyn skal opplysningene og det humant biologiske materialet likevel bevares inntil 31.12.2016. Opplysningene og det humant biologiske materialet skal deretter slettes eller anonymiseres, senest innen 30.06.2017.

Dersom forskningsbiobanken opphører, nedlegges eller overtas av andre, skal det søkes REK om tillatelse, jfr. Helseforskningsloven § 30.

Opplysningene skal lagres avidentifisert, det vil si adskilt i en nøkkel- og en opplysningsfil.

Forskningsprosjektets data skal oppbevares forsvarlig, se personopplysningsforskriften kapittel 2, og Helsedirektoratets veileder for «Personvern og informasjonssikkerhet i forskningsprosjekter innenfor helse- og omsorgssektoren».

Prosjektet skal sende sluttmelding til REK sør-øst D på fastsatt skjema senest 30.06.2012.

Tillatelsen er gitt under forutsetning av at prosjektet gjennomføres slik det er beskrevet i søknaden og protokollen, og de bestemmelser som følger av helseforskningsloven med forskrifter.

Dersom det skal gjøres endringer i prosjektet i forhold til de opplysninger som er gitt i søknaden, må prosjektleder sende endringsmelding til REK. Vi gjør oppmerksom på at hvis endringene er "vesentlige", må prosjektleder sende ny søknad, eller REK kan pålegge at det sendes ny søknad.

Vi ber om at alle henvendelser sendes inn via vår saksportal:
http://helseforskning.etikkom.no eller på e-post til: post@helseforskning.etikkom.no

Vennligst oppgi vårt referansenummer i korrespondansen.

Med vennlig hilsen,

Stein A. Evensen (sign.)
prof. dr.med.
leder

Øyvind Grønlie Olsen
jurist
fungerende komitésekretær

Kopi til: Norges Idrettshøgskole, ved øverste adm. ledelse
Biobankregisteret

NORGES IDRETTSHØGSKOLE

APPENDIX II

DELTA PÅ GRATIS TRENING I HØST!*

*NIH søker forsøkspersoner til løpestudie høsten 2010!

Vi søker kvinner og menn i alderen **18-35 år** som kunne tenke seg å delta i et forskningsprosjekt på Norges Idrettshøgskole.

Hensikten med studien er å se på effekt av sprintintervalltrening sammenlignet med langkjøring på moderat intensitet.

Vi søker friske forsøkspersoner som **ikke har drevet regelmessig utholdenhetstrening** (løping, sykling, ski etc) de siste 2 årene. Det er en fordel at du bor i Oslo, da all trening og testing vil foregå ved Norges idrettshøgskole, ved Sognsvann.

Du vil som forsøksperson få testet din fysiske form på tredemølle, og lære å bruke pulsklokke under løpetrening. Du må ha tid til å trene **3 timer i uka.**

Oppstart av forsøket vil være i i uke 34/35.

Høres dette interessant ut for deg? Ta kontakt, eller gå inn på **www.nih.no/utholdenhetstrening**

Kontakt Line Støen
Telefon: 476 41 418 eller epost:
forskningsprosjekt.nih@gmail.com
Forsøket er godkjent av regional komité for medisinsk forskningsetikk Sør-Norge (REK sør)

NORGES IDRETTSHØGSKOLE

NORGES IDRETTSHØGSKOLE

Forespørsel om deltakelse i forskningsprosjekt.

Utholdenhetstrening: langkjøring vs. sprintintervall?

Sammenligning av effekten av langkjøring og sprintintervall på insulinsensitivitet, metabolisme og hjertefrekvens.

Bakgrunn og hensikt
Tidligere forskning på utholdenhetstrening har vist at både trening med lav intensitet, moderat intensitet og trening med høy intensitet kan ha gunstig effekt på fysisk form og helse. I de senere år har også forskere undersøkt effekten av anaerobe kortintervaller, av typen 6-30 sekunders svært intensivt arbeid på ergometersykkel. Det er også her blitt funnet forbedringer i fysisk form og utholdenhetsprestasjon. Det er imidlertid gjort lite forskning på denne typen trening ved løping.

Hensikten med dette treningsforsøket er å sammenligne effekten av sprintintervaller (fem til ti 30-sekunders løp med høy fart, pause 4 min) med kontinuerlig løping på moderat intensitet (30-60 minutters løping på ca. 70 - 80 % av maksimal hjertefrekvens). Selve treningsperioden skal vare i 8 uker, med tre treningsøkter pr. uke. Prosjektdeltakere skal registrere hjertefrekvens (puls) med bruk av pulsklokke på alle treningene.

Dette skrivet er til alle som ønsker å delta som forsøkspersoner i prosjektet. For å kunne delta må du oppfylle følgende kriterier: Du må være frisk og i alderen 18-35 år og du kan ikke ha drevet organisert og/eller systematisk utholdenhetstrening de siste 2 årene. Videre må du kunne trene tre ganger i uken i 8 uker (fortrinnsvis utendørs), og kunne gjennomføre flere fysiske tester før og etter treningsperioden.

Hva innebærer studien?
I tillegg til utholdenhetsprestasjon ønsker vi å undersøke metabolske forandringer, insulinsensitivitet, hjertefrekvens under trening og hjertefrekvensvariasjon. Disse variablene henger sammen med din utholdenhetskapasitet.

Studien innebærer oppmøte på 6 testdager (eksl. pretest) både i forkant og etterkant av studien, i tillegg til selve treningsperioden hvor du skal trene tre ganger i uken. Før du kan starte opp med treningsøktene skal du møte opp på testlaboratoriet ved Norges Idrettshøgskole for informasjon og tilvenning til ustyr. Selve testingen består av måling av maksimalt oksygenopptak ($VO2_{maks}$,) submaksimal løpetest, anaerob prestasjonstest, glukosetoleransetest, beep-test, lungefunksjonsmålinger, blodprøver og standardisert registrering av hjertefrekvensvariasjon.

Du vil få tilgang på en pulsklokke som skal brukes under hele prosjektet for å registrere hjertefrekvens og hjertefrekvensvariasjon. I løpet av de første dagene vil du få opplæring i bruk av pulsklokken, og du må foreta en 24-timers registrering av hjertefrekvensvariasjon med kompatible EKG-elektroder. Under selve treningsperioden skal du foreta en 10-minutts registrering av hjertefrekvensvariasjon to ganger i uka før frokost. Dette gjøres med pulsklokken og pulsbåndet. Prosedyrene er beskrevet i detalj i kapittel A.

Fordeling i grupper foregår ved loddtrekning.

Treningsprotokoll for gruppe 1: Sprintintervall
Den ene gruppen skal gjennomføre sprintintervaller. På hvert intervall skal du løpe så raskt du kan i 30 sekund. De to første ukene vil de gjennomføre 5 30-sekunders sprintløp pr. økt. Hvileperioden mellom hvert sprintløp er fire minutter. Det gjennomføres alltid 10 min oppvarming, som avsluttes med 3 stigningsløp. Det vil være en gradvis progresjon på antall sprintløp og for hver uke vil antall intervaller økes med 1. Det betyr at på den siste uken skal du løpe 10 x 30 sekunder pr. økt. På hver økt bruker du din pulsklokke for å loggføre økten. .. Etter ukens siste økt leveres pulsklokken til din instruktør. Data vil da bli lagt inn, og du får tilbake klokken på den første økta uken etter.

Treningsprotokoll for gruppe 2: Langkjøring
Den andre gruppen vil gjennomføre treningen med en intensitet tilsvarende 65 % av VO_{2maks}. Dette tilsvarer ca. 70-80 % av din maksimale hjertefrekvens. Det oppleves ”litt anstrengende”, men du skal ikke kjenne tegn til melkesyre. Det gjennomføres 10 min oppvarming. Den første uka vil arbeidstiden tilsvare 40 min per økt. For å sikre progresjon i treningen økes varigheten med 5 min pr. uke. Det betyr at i den siste uken skal du løpe 60 minutter, i tillegg til oppvarmingen. På hver økt bruker du din pulsklokke for å styre intensiteten og logge økta. Etter ukens siste økt leveres pulsklokken til din instruktør. Data vil da bli lagt inn, og du får tilbake klokken på den første økta uken etter.

Mulige fordeler og ulemper

Fordeler
Du har mulighet til å delta i et forskningsprosjekt, og du får anledning til å teste din utholdenhet før, under og etter treningsforsøket. Dette gir deg innsikt i hvordan en type trening over tid kan påvirke fysiologiske faktorer i kroppen din. Du vil få kunnskap om utholdenhetstrening, og sannsynligheten for at du kommer i bedre fysisk form er stor, basert på kunnskap og praksis fra andre treningsstudier. Videre vil du lære om hvordan du kan bruke pulsklokke under trening og registre inn pulskurver fra dine treningsøkter på datamaskin. Alle forsøkspersoner som deltar vil få informasjon om resultatene fra treningsforsøket, dersom det er ønskelig.

Ulemper
Deltakelse i treningsforsøket vil kreve mye tid. Det må påberegnes at du møter opp 6 separate dager (1-4 timer pr. dag) for testing i forkant og etterkant av selve treningsperioden. I tillegg må du beregne 10 min ekstra tid 2 ganger i uka på morgenen, for pulsregistrering.

Gjennomføring av treningen vil kunne medføre en viss risiko for skader som ved all løpetrening. Følelse av stølhet, som følge av uvant trening, kan i starten av treningsperiode oppleves som ubehagelig. Noen av de fysiske testene kan oppleves meget anstrengende. Dette er i midlertidig en kortvarig følelse. Når det gjelder risiko for skader under selve testingen, så er den liten.

Hva skjer med prøvene og informasjonen om deg?

Prøvene tatt av deg og informasjonen som registreres om deg skal kun brukes slik som beskrevet i hensikten med studien. Alle opplysningene og prøvene vil bli behandlet uten navn og fødselsnummer eller andre direkte gjenkjennende opplysninger. En kode knytter deg til dine opplysninger og prøver gjennom en navneliste. Det betyr at opplysningene er avidentifisert.

Det er kun autorisert personell knyttet til prosjektet som har adgang til navnelisten og som kan finne tilbake til deg.

Det vil ikke være mulig å identifisere deg i resultatene av studien når disse publiseres.

Frivillig deltakelse

Det er frivillig å delta i studien. Du kan når som helst og uten å oppgi noen grunn trekke ditt samtykke til å delta i studien. Dette vil ikke få konsekvenser for din videre behandling. Dersom du ønsker å delta, undertegner du samtykkeerklæringen på siste side. Om du nå sier ja til å delta, kan du senere trekke tilbake ditt samtykke uten at det påvirker din øvrige behandling. Dersom du senere ønsker å trekke deg eller har spørsmål til studien, kan du kontakte en av følgende prosjektmedarbeidere:

Line Støen - 47641418,
Marit Sandvei - 90795938,
Sigbjørn Litleskare, - 93433946.
E-post:
forskningsprosjekt.nih@gmail.com

Eller
Prosjektleder Jørgen Jensen på telefonnummer 23 26 22 49, eller e-post:
jorgen.jensen@nih.no

Nettside:
http://www.nih.no/utholdenhetstrening

Ytterligere informasjon om studien finnes i kapittel *A*
– utdypende forklaring av hva studien innebærer.

Ytterligere informasjon om biobank, personvern og forsikring finnes i kapittel B –
Personvern, biobank, økonomi og forsikring.

Samtykkeerklæring følger etter kapittel B.

Kapittel A - utdypende forklaring av hva studien innebærer

Kriterier for deltakelse
Du må være frisk og i alderen 18-35 år og du kan ikke ha drevet organisert og/eller systematisk utholdenhetstrening de siste 2 årene. Videre må du kunne trene tre ganger i uken i 8 uker (løping på tredemølle eller vei/bane) og kunne gjennomføre flere fysiske tester før og etter treningsperioden.

A. Inklusjonskriterier:
- Mellom 18-35 år
- BMI 20 - 27 kg/m^2
- Ikke trent systematisk utholdenhetstrening de siste to årene

B. Eksklusjonskriterier:
- Røykere
- Alvorlige lidelser (vedlegg: egenerklæring helseundersøkelse)

Bakgrunn og hensikt med studien – utdyping av variabler som skal måles

Det er vist at utholdenhetstrening bedrer fysisk form og prestasjon, både for idrettsutøvere og øvrige. De kroppslige endringene som skjer er komplekse, men av stor interesse for forskere. I dette prosjektet skal vi undersøke om sprintintervalltrening har samme effekt som kontinuerlig løping på følgende variabler:

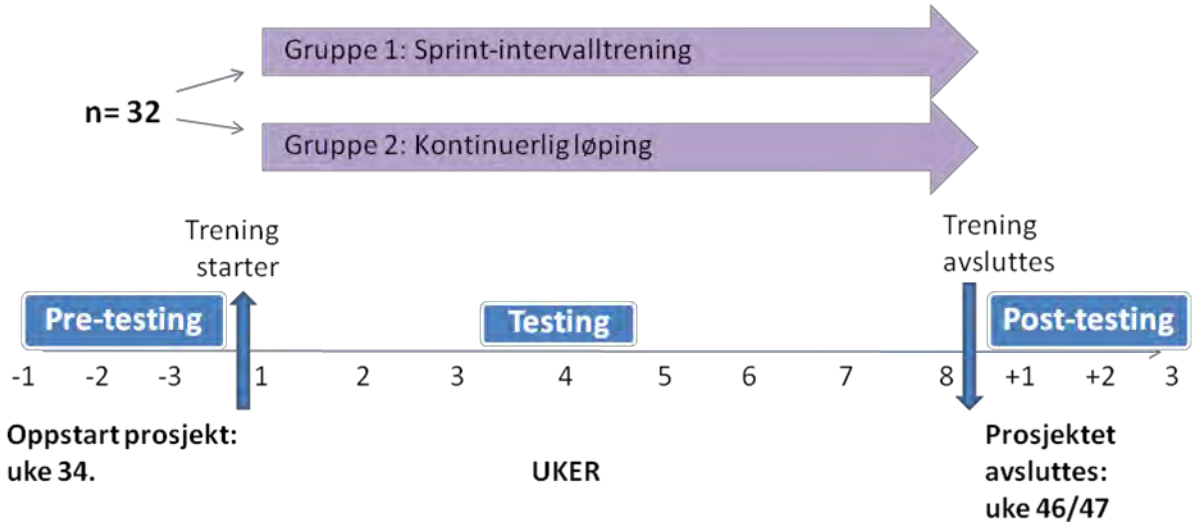
Utholdenhetsprestasjon
Det maksimale oksygenopptaket ($VO2_{maks}$) brukes som mål for organismens maksimale opptak av oksygen per tidsenhet, og beskriver utholdenhetskapasitet. I forskningen er dette gullstandard for måling av aerob kapasitet, men det gir imidlertid ikke det fulle svaret på utholdenhetsprestasjon. Derfor inkluderes ofte også andre tester, som kan sammenlignes med VO_{2maks} og utfylle denne testen.

Fettmetabolisme
Med fettmetabolisme menes kroppens evne til å forbruke fett som energi. Bruk av fett som energikilde under trening fører til at karbohydratlagrene (glykogenlagrene) spares. Fettmetabolisme måles ut i fra respiratorisk utvekslingskvotient, og ut i fra dette tallet kan en si om det er karbohydrat eller fett som forbrukes.

Insulinsensitivitet
Insulinsensitiviteten vil bli målt ved en oral glukose toleranse test (OGTT). Dette sier noe om kroppens evne til karbohydratforbenning, og det er vist at insulinsensitiviteten bedres ved fysisk aktivitet og trening.

Hjertefrekvensvariasjon (e.g Heart rate variability)
Selv om hjertefrekvensen kan virke stabil, kan tiden mellom to slag variere. Tiden mellom to slag er definert som hjertefrekvensvariasjon(HRV). Variasjoner i hjertefrekvensen blir brukt som indeks på autonome nervesystemets responsitivitet. Høy HRV er assosiert med god fysisk form.

Tidsskjema
Rekruttering av forsøkspersoner foregår i begynnelsen av august. Inklusjon av forsøkspersoner, basert på egenerklæringsskjema og tilvenningsdager, foregår medio august. Forsøkspersoner må være forberedt på å møte til 6 forskjellige dager på testlaboratoriet ved Norges Idrettshøgskole. Se detaljer nedenfor. Oppmøtetid for testing vil bli avtalt mellom testledere og forsøksperson, men det er ønskelig at vi får samlet opp til gruppevise oppmøter. Videre vil treningsøktene foregå utendørs ved Norges idrettshøgskole med erfarne treningsveiledere. Treningsveilederne er fleksible på tidspunkt for treningsøkter, men det er ønskelig at forsøkspersoner kan benytte seg enten av morgenøkt eller ettermiddagsøkt. Det vil kunne bli gjort unntak på enkelte dager og det vil bli mulighet for at en eller to av ukens økter kan utføres hjemme eller mer sentralt.



Undersøkelser, tester og målinger
Nedenfor følger et eksempel på hvordan de 6 testdagene fordeler seg. Rekkefølgen kan variere fra person til person og informeres om ved prosjektstart. Samme prosedyre vil foregå etter treningsperioden, med unntak av informasjon- og tilvenningsdagene som da vil forsvinne. Det vil alltid være minst 1 dag mellom hver fysiske test, og alltid minst en hviledag før OGTT-testen.

Dag 1: Informasjon om prosjektet, tilvenning på tredemølle og opplæring i bruk av pulsklokke (varighet: ca. 4 timer)
På den første testdagen vil du få tilstrekkelig med informasjon om prosjektet, og du vil få opplæring i løping på tredemølle og tilvenning til utstyr. Du kommer til å få utdelt en pulsklokke (Polar RS800CX) med tilhørende kodet pulsbånd (Polar WearLink W.I.N.D), som du skal bruke på hver treningsøkt for å styre intensiteten og registrere data. Det vil bli gitt individuell opplæring i bruk og lagring av data som registreres på klokken. Hvis forsøkspersoner og testleder føler det ikke er tilstrekkelig med en dags opplæring, vil det bli tilbudt en dag ekstra.

Dag 2: Intro til 24-timers registrering av hjertefrekvensvariasjon (varighet: 30 min)
På dag 2 skal du først gjennomføre måling av hjertefrekvens i hvile. Det foregår med bruk av pulsbånd og klokke, og du skal først sitte i 5 minutt, for så å stå i 5 minutt. Dette er tilsvarende målingene du skal gjøre hjemme 2 ganger i uken. Deretter vil du få hjelp til å feste på EKG-elektroder rett under brystet, som er kompatible med pulsklokken. Disse skal brukes for å registrere hjertefrekvensvariasjonen i 24 timer. Du skal leve som normalt under denne registreringen, og elektrodene skal også være på hele natten mens du sover. I

forbindelse med registreringen vil du få med deg en aktivitetsmonitor, slik at vi kan registrere all fysisk aktivitet i de 24-timene du skal ha på utstyret. Aktivitetsmonitoren ser ut som en vanlig klokke, og skal bæres på motsatt arm av den andre klokken (pulsklokken). Du vil få opplæring i bruk av utstyret, og det er viktig at du ikke tar det av deg. Det vil bli satt på tastelås, slik at man ikke plutselig bare trykker på stopp.

Dag 3: Måling av oral glukosetoleranse/insulinsensitivitet (varighet: ca. 3 timer)
På den tredje testdagen må du komme "fastende" om morgenen på NIH. Det betyr at du ikke må spise eller drikke noe, bortsett fra vann, etter klokken 22.00 dagen før testen. Dagen starter med at du får hjelp til å ta av utstyret fra 24-timers målingen du startet med dagen før. Det er viktig at du kommer på NIH før kl 08.00 siden en ny gruppe trenger utstyret. Videre følger måling av kroppssammensetning (InBody-vekt) før glukosetoleransetesten starter. Der skal du drikke en sukkerholdig drikk som inneholder 75 gram glukose oppløst i 3-4 dl vann. Det vil bli tatt blodprøver etter 0, 30, 60, 90 og 120 minutter. Under testingen vil også respiratoriske målinger og hjertefrekvensregistrering bli gjort. Etter testslutt vil du få utdelt en enkelt frokost.

Dag 4: Måling av maksimalt oksygenopptak på tredemølle (varighet: ca. 30 min)
På dag 4 skal du teste ditt maksimale oksygenopptak. For å måle hvor mye oksygen du puster inn og CO2 du puster ut, brukes et munnstykke som er koblet til en slange og en oksygenanalysator. Det vil også bli brukt neseklype for å få all luft inn i analysatoren. Det er kvalifisert testpersonell som utfører testingen. Selve testingen starter med at du varmer opp tredemølle i 15-20 min, før belastningen gradvis økes helt til du ikke orker mer. Dette vil totalt ta ca. 30 min. Umiddelbart etter avsluttet test vil det bli tatt en fingerstikkprøve for måling av melkesyre i blodet.

Dag 5: Submaksimal løpetest og anaerob prestasjonstest (varighet: ca. 45 min)
Deretter skal du løpe på tredemølle for å kartlegge metabolisme (RER-verdi) og løpsøkonomi. Du løper på tredemølle på fire submaksimale belastninger (50, 60, 70 og 80 % av maksimalt oksygenopptak) i fem minutter på hver belastning. Den første belastningen er meget rolig, mens den siste vil oppleves som anstrengende. Etter hver belastning får du 1 minutt pause hvor det vil det bli tatt blodprøve fra fingerstikk. Dette fordi vi skal analysere innholdet i blodet ditt (laktat) Oksygenopptaket måles også på hver belastning for å vurdere løpsøkonomi. Umiddelbart etter siste belastning er ferdig får du en liten pause, før du blir fulgt inn i en idrettshall. Her skal du løpe 5x60 meter, med 30 sekunders pause i mellom hver 60-meter. Denne testen tar kort tid, men oppleves svært anstrengende underveis. Testen blir brukt som mål på anaerob prestasjon.

Dag 6: Beep-test (varighet: ca. 30 min)
På dag 6 skal du gjennomføre den siste fysiske testen før treningsforsøket starter. Du skal løpe mellom to markeringer på 20 m med økende hastighet innen et gitt lydsignal. Dersom du ikke når markeringen på andre siden innen lydsignalet vil testen bli avsluttet for den enkelte, og antall lydsignal/gjennomført lengde vil bli registrert (nivået du nådde og hvilken runde på det nivået du har gjennomført). Denne testen gjennomfører du sammen med andre forsøkspersoner, og varigheten på testen er avhengig av din utholdenhetskapasitet. Testen vil for de fleste ta mellom 10 og 20 minutter. Total varighet inkludert oppvarming vil være ca. 30 minutter.

Mulige bivirkninger ved testing og målinger
Deltakelse i prosjektet vil kreve mye tid til tester og målinger, og noen av testene vil oppleves anstrengende. Følelse av stølhet og tretthet i muskulatur vil forekomme, men dette er forbigående.Det er minimal fare for at noe skal gå galt ved blodprøvetakingen, og det er kvalifisert helsepersonell som vil foreta alle disse prøvene.

Økonomi og honorar
Du som forsøksperson får ingen økonomisk honorar for å delta i denne studien, men vi vil dekke eventuelle ekstrautgifter i forbindelse med reise til og fra NIH, og frokost/lunsj på enkelte testdager. Videre vil du som forsøksperson etter endt studie få tips, råd og kunnskap om hvordan du kan legge opp egen utholdenhetstrening etter prosjektets slutt.

Studiedeltakerens ansvar

- Møte opp til avtalte tider, evt. avlyse i god tid i forveien om oppsatt dato/tid for møtet ikke passer.
- Ta vare på, og ta med pulsklokke og pulsbånd til hver test/treningsøkt.
- Registrere hjertefrekvensvariasjon med pulsklokke og pulsbånd 2 ganger i uka, før frokost.

Kapittel B - Personvern, biobank, økonomi og forsikring

Personvern
I dette prosjektet vil ikke navnet ditt være knyttet til noen forsøksdata. Norges idrettshøgskole ved administrerende direktør er ansvarlig for behandling av data. Hvis du sier ja til å delta i studien, har du rett til å få innsyn i hvilke opplysninger som er registrert om deg. Du har videre rett til å få korrigert eventuelle feil i de opplysningene vi har registrert.

Analysene av hjertefrekvensvariasjon vil utføres i samarbeid med dr.Scient Mikko Tulppo fra Finland. Han vil kun få tilgang til datamaterialet knyttet til hjertefrekvensmålinger, og har ikke tilgang til personopplysninger.

Biobank
Blodprøvene som blir tatt og informasjonen utledet av dette materialet vil bli lagret i en forskningsbiobank ved Norges idrettshøgskole. Hvis du sier ja til å delta i studien, gir du også samtykke til at det biologiske materialet og analyseresultater inngår i biobanken. Jørgen Jensen er ansvarshavende for forskningsbiobanken. Biobanken planlegges å vare til 2015 Etter dette vil materiale og opplysninger bli destruert og slettet etter interne retningslinjer.

Utlevering av materiale og opplysninger til andre
Hvis du sier ja til å delta i studien, gir du også ditt samtykke til at prøver og avidentifiserte opplysninger utleveres til bruk i vitenskapelige publikasjoner.

Rett til innsyn og sletting av opplysninger om deg og sletting av prøver
Hvis du sier ja til å delta i studien, har du rett til å få innsyn i hvilke opplysninger som er registrert om deg. Du har videre rett til å få korrigert eventuelle feil i de opplysningene vi har registrert. Dersom du trekker deg fra studien, kan du kreve å få slettet innsamlede prøver og opplysninger, med mindre opplysningene allerede er inngått i analyser eller brukt i vitenskapelige publikasjoner.

Økonomi
Studien er finansiert av Norges idrettshøgskole. Det er ingen sponsorer tilknyttet til prosjektet, og ingen interessekonflikter.

Forsikring
Du er som forsøksperson forsikret via særskilt forsikring ved Norges idrettshøgskole.

Informasjon om utfallet av studien
Som forsøksperson har du rett til informasjon om utfallet av studien.

Frivillig å delta
Det er frivillig å delta og du kan når som helst trekke deg fra prosjektet uten å oppgi noen begrunnelse for valget. Alle data vil bli anonymisert.

Dersom du skulle ønske å trekke tilbake samtykke om deltakelse i studien kan du kreve at det biologiske materialet blir destruert, og at innsamlet helse- og personopplysninger blir slettet eller utlevert. Muligheten til å tilbakekalle samtykket eller kreve destruksjon, sletting eller utlevering gjelder ikke dersom opplysningene alt har gått inn vitenskapelige arbeid, jfr. biobankloven § 14 tredje ledd. Dersom du ønsker flere opplysninger angående prosjektet kan du kontakte prosjektmedarbeidere:

Line Støen - 47641418,
Marit Sandvei - 90795938,
Sigbjørn Litleskare, - 93433946.

E-post:
forskningsprosjekt.nih@gmail.com

Eller
Prosjektleder Jørgen Jensen på telefonnummer 23 26 22 49, eller e-post:
jorgen.jensen@nih.no

Samtykke til deltakelse i studien

"Sammenligning av effekten av langkjøring og sprintintervall på insulinsensitivitet, metabolisme og hjertefrekvens"

Jeg er villig til å delta i studien

(Signert av prosjektdeltaker, dato)

Stedfortredende samtykke når berettiget, enten i tillegg til personen selv eller istedenfor

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(Signert av nærstående, dato)

Jeg bekrefter å ha gitt informasjon om studien

(Signert, rolle i studien, dato)

APPENDIX IV

Etternavn:	Fornavn:	Født:
Studentadresse:		
Hjemmeadresse:		
Tlf.:	E-mailadresse:	
Idrettsbakgrunn (angi idrettsgrener og omtrent hvor mange timer du trener pr. uke):		

EGENERKLÆRING FOR FORSØKSPERSONER

Takk for at du vurderer å delta som forsøksperson ved Norges idrettshøgskole! Før du kan delta, må vi imidlertid kartlegge om din deltakelse kan medføre noen form for helserisiko. Vær snill å lese gjennom alle spørsmålene nøye og svar ærlig ved å krysse av for JA eller NEI. Hvis du er i tvil, bør du be om å få snakke med legen som er ansvarlig for forsøket.

Hvis du krysser av for JA på ett eller flere av disse spørsmålene, må du gjennomgå en legeundersøkelse før forsøksstart. Ved enkelte typer forsøk vil du uansett bli innkalt til legeundersøkelse.

APPENDIX IV

JA	NEI	
☐	☐	1. Kjenner du til at du har en hjertesykdom?
☐	☐	2. Hender det du får brystsmerter i hvile eller i forbindelse med fysisk aktivitet?
☐	☐	3. Kjenner du til at du har høyt blodtrykk?
☐	☐	4. Bruker du for tiden medisiner for høyt blodtrykk eller hjertesykdom (f.eks. vanndrivende tabletter)?
☐	☐	5. Har noen av dine foreldre, søsken eller barn fått hjerteinfarkt eller dødd plutselig (før fylte 55 år for menn og 65 for kvinner)?
☐	☐	6. Røyker du?
☐	☐	7. Kjenner du til om du har høyt kolesterolnivå i blodet?
☐	☐	8. Har du besvimt i løpet av de siste 6 måneder?
☐	☐	9. Hender det du mister balansen på grunn av svimmelhet?
☐	☐	10. Har du sukkersyke (diabetes)?
☐	☐	11. Kjenner du til noen annen grunn til at din deltakelse i prosjektet kan medføre helse- eller skaderisiko?

Gi beskjed straks dersom din helsesituasjon forandrer seg fra nå og til undersøkelsen er ferdig, f.eks. ved at du blir forkjølet, får feber, eller blir gravid.

_____

Sted - dato

_____

Underskrift

APPENDIX V

Spørreskjema for forsøkspersoner

ID-nr (testansvarlige fyller ut):

Mann ☐ Kvinne ☐

Vekt (kg):

Høyde (cm):

1. Er du interessert i fysisk aktivitet og trening?

Nei ☐ Ja ☐ Litt ☐

2. Hvordan kommer du deg til jobb/skole?

Går ☐ Sykler ☐ Bil ☐ Kollektivt ☐ Annet ☐

3. Har du tidligere deltatt/ deltar i organisert idrett?

Ja ☐ Nei ☐

Hvis **ja**, svar på de påfølgende spørsmålene (Hvis nei, hopp til spørsmål 7)

4. Hvilken type idrett?

5. Hvor mye trente/trener du?

< 1 gang i mnd ☐ 1-2 ganger i mnd ☐ 1-2 ganger i uka ☐

3-4 ganger i uka ☐ > 4 ganger i uka ☐

6. Når sluttet du, eller holder du på fortsatt?

7. Hvor fysisk aktiv er du i hverdagen (gange, sykling, hagearbeid, etc)?

< ½ time/dag ☐ ½-1 time/dag ☐ 1-3 timer/dag ☐ >3 timer/dag ☐

8. Har du drevet systematisk trening de siste to årene (inkl. både uorganisert og organisert idrett)?

Ja ☐ Nei ☐

APPENDIX V

Hvis **ja**, svar på de påfølgende spørsmålene. Hvis **nei**, er du ferdig med spørreskjema.

9. Hvordan type trening (styrke, spenst, utholdenhet etc.)?

10. Hvor ofte trente/trener du?

< 1 gang i mnd ☐ 1-2 ganger i mnd ☐ 1-2 ganger i uka ☐

3-4 ganger i uka ☐ > 4 ganger i uka ☐

11. Når trente du sist?

De siste 3 dagene ☐ Siste uken ☐ Siste 2 ukene ☐

Siste måneden ☐ Siste 3 mnd ☐ Siste 6 mnd ☐

Takk for hjelpen! ☺

Definisjonen på FA er all aktivitet skapt av din muskulatur. Det betyr at det å gå opp trappen eller klippe plen er FA. Altså aktiviteter som gjør at du puster litt tyngre.

APPENDIX VI

Rutiner for bruk av Polar pulsklokke og aktivitetsmåler

For deltakere i forskningsprosjekt ved Norges Idrettshøgskole høsten 2010.

Hvis du har problemer, ikke nøl med å ringe eller sende e-post.

Line Støen: 47641418
forskningsprosjekt.nih@gmail.com

NORGES IDRETTSHØGSKOLE

1.0 Forklaring av knapper på klokken.

2.0 Prosedyre for kortidsmåling av HRV

Du skal ukentlig gjennomføre to kortidsmålinger av HRV (hjertefrekvensvariasjon). Dette gjorde du i testperioden i forkant av glukosetoleransetesten, og under testen. Målingen skal gjennomføres tidlig morgen, før frokost, etter første toalettbesøk.

1. måling: hviledag
2. måling: treningsdag

Eks: du trener mandag – onsdag – fredag, 1.måling gjennomføres tirsdag, 2. måling gjennomføres onsdag. Alternativt – torsdag(1) og fredag(2).

Prosedyre for selve målingen:
5 minutter sittende, etterfulgt av 5 minutter stående

1. Gå inn på ”settings” på klokken, trykk videre inn på ”features”, trykk deg ned til ”RR data”, sjekk at eller sørg for at RR data er ON! (ved å trykk ok – rød knapp). Trykk deretter stopp for å komme tilbake til utgangspunktet.
2. Fukt pulsbåndet med vann på sidene av senderen.
3. Fest pulsbåndet rundt overkroppen, rett under brystet. Sjekk at brikken/senderen sitter rett vei.
4. Finn deg en stol, prøv å unngå å sitte i nærheten av annet elektronisk utstyr, av type tv, pc, mobil osv. Slapp av og nyt morgenroen.
5. Trykk på start (rød knapp) **2 ganger!!**
6. Bli sittende 5 min, trykk deretter rød knapp (runde/lap) og reis deg.
7. Stå i 5 min. Trykk deretter stopp (knapp nederst på venstre side). Du er nå ferdig.
8. Gjenta punkt 1, men skru av ”OFF” RR-data. Klokken er nå klar til bruk på trening. (5s rec time

3.0 Bruk av pulsklokken på trening

1. Sjekk at RR-data er OFF om du er usikker, se 2.0.
2. Forandre/stille inn HR-view (slik at du kan se % av din makspuls) – velg %HRmax (settings – features – HR view – HR%)
3. Trykk på start (rød knapp), se at puls kommer opp, trykk en gang til – du er nå i gang med registreringen.
4. Velg visningsmodus med velgeknappene (se punkt 1.0), for langkjøringsgruppen kan det være greit å ha ”heart rate” eller ”lap time”. For sprintintervallgruppen anbefales ”lap time”. På denne måten kan du ha kontroll på tiden selv, og vite at du løper akkurat 30 sekund, og har akkurat 4 minutt pause. Dette forutsetter at for hver sprint må du trykke på den rød både før og etter. Eks. lap 1; 30sek, lap 2; 4 minutt.
5. Trykk på rød knapp etter oppvarmingen (for å sette skille/lap), og før nedtrapping.
6. Trykk stopp når du er helt ferdig, NB! Knapp nederst til venstre. Se punkt 1.0.

TARGET ZONE	INTENSITY % OF HRmax	EXAMPLE INTERVAL DURATIONS	PHYSIOLOGICAL BENEFIT/ TRAINING EFFECT
5 MAXIMUM	90-100%	0-2 minutes	>Tones the neuromuscular system >Increases maximum sprint race speed
4 HARD	80-90%	2-10 minutes	>Increases anaerobic tolerance >Improves high speed endurance
3 MODERATE	70-80%	10-40 minutes	>Enhances aerobic power >Improves blood circulation
2 LIGHT	60-70%	40-80 minutes	>Increases aerobic endurance >Strengthens body to tolerate higher intensity training >Increases fat metabolism
1 VERY LIGHT	50-60%	20-40 minutes	>Helps and speeds up recovery after heavier exercises

Bildet viser Polars intensitetsskala (mest til hjelp for langkjøringsgruppen).Les mer om denne på www.pulsklokke.no.

4.0 Bruk av aktivitetsmåler – Polar FA20

Du låner aktivitetsmåleren av Norges Idrettshøgskole, og det er kun DU som skal bruke den under prosjektperioden. Aktivitetsmåleren skal brukes under hele treningsperioden, og den skal leveres inn på dataavlesning hver 1 gang pr. andre uke, på samme tid som pulsklokken. Aktivitetsmåleren tåler litt vann, men fungerer ikke så godt til svømming. Aktivitetsmåleren kan tas av om natta, og tas på igjen tidlig på morgen. Aktivitetsmåleren fanger ikke opp at du sykler, så hvis du sykler mye (eks. til og fra jobb) er det fint om du rapporterer dette. Aktivitetsmåleren skal brukes på ikke-dominant hånd, dvs. motsatt av det du hadde på 24-timers registreringen, altså der du pleier å bruke klokke. På treningsøktene kan du flytte aktivitetsmåleren oppover på armen, slik at du får plass til pulsklokka. Evnt. bruke motsatt arm.

5.0 Oppbevaring og vedlikehold av klokken og pulsbåndet

Du låner klokken av Norges Idrettshøgskole, og det er kun DU som skal bruke den under prosjektperioden. Sørg for at den er innelåst i bolig da den ikke er i bruk. Legg den gjerne i boksen da du ikke bruker den, da vet du hvor du har den.

Rutine for skylling og vasking av pulsbåndet. (se side 17 i bruksanvisning):

1. Koble kontakten (delen som kan kneppes av foran) fra båndet, og skyll stroppen etter hver gang du har brukt den på trening.
2. Vask stroppen 1 gang i uka (NB! 40 grader i vaskepose, NB! Kun stroppen/båndet, ikke kontakten)
3. Stroppen må ikke bløtlegges, sentrifugeres, strykes, tørrenses eller blekes. Ikke bruk vaskepulver med blekemiddel eller skyllemiddel.
4. Knepp alltid AV kontakten før vasking/skylling.

Bilde viser pulsbåndet og avtagbar sender/mottaker. Denne skal kobles av ved skylling og vasking.

6.0 Innlevering/utlevering av klokken og aktivitetsmåleren

For at klokken og aktivitetsmåleren ikke skal gå tom for lagringsplass må du levere de inn en gang pr. andre uke. Dette vil du få nærmere beskjed om fra uke til uke, men det blir mest sannsynlig på ukens siste økt. Det vil si for de fleste, fredag eller lørdag.

Utstyret skal leveres i utdelt boks enten til Line, Sigbjørn eller Marit. Hvis du ikke får levert utstyret til en av oss, kan du levere det i resepsjonen på Norges idrettshøgskole. Du får tilbake utstyret på første økt, eller til avtalt tid. Du kan maks ha utstyret i 20 dager før du må levere inn.

For spørsmål, kontakt oss på telefon eller mail!

APPENDIX VII

Utholdenhetstrening: sprintintervall vs langkjøring

SPØRRESKJEMA FOR FORSØKSPERSONER

FP NR **SETT HAKE ELLER RING RUNDT DITT/DINE SVAR**

KOSTHOLD UNDER TRENINGSPERIODEN

1. Har du endret spisevaner under treningsperioden?

JA **NEI** **USIKKKER**

2. Hvis JA, hva slags endringer? (flere kryss mulig)

Spist mer mat generelt
Spist sunnere
Spist mindre
Spist mer snop og usunn mat
Annet

Kommentar

3. Følte du deg mer sulten etter at du kom i gang med treningen?

JA **NEI** **USIKKER**

4. Ble du bevisst på å spise i god tid før trening?

JA **NEI** **BÅDE OG (slurvet litt)**

Kommentar:

5. Dagen før OGTT og samme dag; fikk du i deg noe annet en vann?
(eks. te, kaffe, tyggis, repsils, røyk, snus, pastiller, slurk saft eller lignende)

6. Har du røyket eller snuset under treningsperioden? Hvis JA, hvor mye?

ANDRE TRENINGSVANER

1. Hvor ofte trente du utenom de oppstatte treningene i prosjektet?

2. Hva slags type trening/aktivitet har du gjort utenom?

3. Startet du opp med noen nye aktiviteter (som du ikke holdt på med før prosjektstart)?
I såfall hva?

APPENDIX VII

SELVE TRENINGEN

1. Føler du at du har kommet i bedre form?

JA **NEI** **USIKKER**

Kommentar:

2. Likte du treningsformen?

JA **NEI** **USIKKER**

Kommentar

3. Hvordan opplevde du intensiteten på treningsøktene?
(subjektiv følelse i kroppen; bein og pust/puls)
På en skala fra 6-20, hvor 6 er svært lett og 20 er maksimalt anstrengende (se vedlegg)

Sprintgruppa:	**Langkjøring:**
Oppvarming	Oppvarming
Stigningsløp	Hoveddel
Hver sprint (- første)	Nedtrapping

Kommentar:

SKADE OG SYKDOM

Spesifiser hva slags skade eller sykdom, svar kort, stikkord!
1. Har du vært syk eller skadet under treningsperioden som følge av selve treningen?

2. Har du att forkjølelse/influensa/annen sykdom som har holdt deg borte fra treningen?

3. Hvis du har vært syk: hvordan følte du at det påvirket treningen og formen?

4. Hvis du har vært skadet: hvordan følte du at det har påvirket treningen og formen?

VEIEN VIDERE

Vi kommer til å tilby individuell veiledning, hva er mest interessant for deg?
(frivillig, sett kryss/hake)
Veiledning i utholdenhetstrening - optimalisering
Veiledning styrketrening
Veiledning om endring i kroppssammensetning/vekt
Kostholdsveiledning
Bruk av pulsklokken

Kommentar:

Ønsket dato/tidspunkt for veiledning:

NORGES IDRETTSHØGSKOLE

APPENDIX VII

NORGES IDRETTSHØGSKOLE

BORGS SKALA FOR OPPLEVD ANSTRENGELSE

*Borg, Gunnar 1972

6	**Ingen anstrengelse**
7	**Svært lett**
8	
9	**Meget lett**
10	
11	**Lett**
12	
13	**Litt anstrengende**
14	
15	**Anstrengende**
16	
17	**Meget anstrengende**
18	
19	**Svært anstrengende**
20	**Maksimalt anstrengende**