STUDIES IN SPORT, PHYSICAL EDUCATION AND HEALTH
224

Piia Kaikkonen

Post-exercise Heart Rate Variability

A New Approach to Evaluation of Exercise-Induced Physiological Training Load

JYVÄSKYLÄN YLIOPISTO

STUDIES IN SPORT, PHYSICAL EDUCATION AND HEALTH 224

Piia Kaikkonen

Post-exercise Heart Rate Variability

A New Approach to Evaluation of Exercise-Induced Physiological Training Load

Esitetään Jyväskylän yliopiston liikuntatieteellisen tiedekunnan suostumuksella
julkisesti tarkastettavaksi yliopiston Historica-rakennuksen salissa H320
kesäkuun 13. päivänä 2015 kello 12.

Academic dissertation to be publicly discussed, by permission of
the Faculty of Sport and Health Sciences of the University of Jyväskylä,
in building Historica, auditorium H320, on June 13, 2015 at 12 o'clock noon.

UNIVERSITY OF JYVÄSKYLÄ

JYVÄSKYLÄ 2015

Post-exercise Heart Rate Variability

A New Approach to Evaluation of Exercise-Induced Physiological Training Load

STUDIES IN SPORT, PHYSICAL EDUCATION AND HEALTH 224

Piia Kaikkonen

Post-exercise Heart Rate Variability

A New Approach to Evaluation of Exercise-Induced Physiological Training Load

UNIVERSITY OF JYVÄSKYLÄ

JYVÄSKYLÄ 2015

Editors
Taija Juutinen
Department of Biology of Physical Activity, University of Jyväskylä
Pekka Olsbo, Timo Hautala
Publishing Unit, University Library of Jyväskylä

URN:ISBN:978-951-39-6218-0
ISBN 978-951-39-6218-0 (PDF)

ISBN 978-951-39-6217-3 (nid.)
ISSN 0356-1070

Jyväskylä University Printing House, Jyväskylä 2015

ABSTRACT

Kaikkonen, Piia
Post-exercise heart rate variability: a new approach to evaluation of exercise-induced physiological training load
Jyväskylä: University of Jyväskylä, 2015, 88 p.
(Studies in Sport, Physical Education and Health
ISSN 0356-1070; 224)
ISBN 978-951-39-6217-3 (nid.)
ISBN 978-951-39-6218-0 (PDF)

To improve maximal endurance performance, an optimal physiological training load, i.e. the balance between exercise and recovery, is required. In general, the goal of a single endurance exercise session is to transiently disturb body homeostasis after which a so-called supercompensation, an improvement in performance, can occur if the recovery period is adequate. The physical training load of a single exercise session consists of the combination of exercise intensity and duration. At the present time, there is no single tool to quantify the amount of disturbance of homeostasis that is equal to physiological training load of a single exercise session. Heart rate variability (HRV), the changes in time between consecutive R-R –intervals, has widely been used as a non-invasive tool to estimate changes in cardiac autonomic modulation in different physiological conditions. Methodological limitations have usually inhibited the investigation of HRV during changes in autonomic modulation, for example during immediate recovery after exercise. In the present study, a time-frequency analysis of HRV was used to exceed this limitation. The main aim of the present study was to find out if HRV could be used to estimate the exercise-induced physiological training load of single endurance exercise sessions. The results of the present study indicated that the differences in physical training load of endurance exercise sessions, either by changes in exercise intensity or duration, could be detected in immediate post-exercise HRV already during the first recovery minutes. The main factor determining post-exercise HRV seemed to be exercise intensity, but increases in exercise duration could be detected in post-exercise HRV as well, if the exercise intensity was at least moderate. There were negative relationships between post-exercise HRV and so-called traditional training load parameters, for example blood lactate, rating of perceived exertion, excess post-exercise oxygen consumption and training impulse. The results of the present study suggest that immediate post-exercise HRV may be used to estimate physiological training load of single exercise sessions, and it seems to give additional information when compared to traditional parameters used to quantify training load.

Keywords: training load; cardiac autonomic modulation; endurance exercise; HRV recovery dynamics

Author's address

Piia Kaikkonen
Tampere Research Center of Sports Medicine
PO Box 30 - Kaupinpuistonkatu 1
FI-33501 Tampere
Finland
piia.kaikkonen@uta.fi

Supervisors

Professor Emeritus Heikki Rusko, PhD
Department of Biology of Physical Activity
University of Jyväskylä, Finland
KIHU-Research Institute for Olympic Sports
Jyväskylä, Finland

Professor Heikki Kyröläinen, PhD
Department of Biology of Physical Activity
University of Jyväskylä, Finland

Reviewers

Laurent Mourot, PhD
Franche-Comté University
Besançon, France

Professor Mikko Tulppo, PhD
School of Applied Science
London South Bank University
London, UK

Opponent

Professor Robert P. Lamberts, PhD
Division of Orthopedic Surgery
Stellenbosch University
Stellenbosch, South Africa
and
Division of Exercise Science and Sports Medicine
University of Cape Town
Cape Town, South Africa

ACKNOWLEDGEMENTS

This study was carried out at KIHU – Reseach Institute for Olympic Sports, Jyväskylä, Finland, at the Department of Biology of Physical Activity, University of Jyväskylä and at the Tampere Research Center of Sports Medicine, UKK Institute. There are several persons without who this thesis could not been done, and to whom I wish to express my gratitude.

First, I express my deepest gratitude to my supervisor Professor Emeritus Heikki Rusko, PhD, the former Scientific and Managing Director of KIHU. Heikki, thank you for introducing to me this interesting research topic, and thank you for your invaluable and excellent guidance and supportive feedback during all the years of my study. It has been a privilege to work in your research group. I also express my gratitude to my other supervisor Professor Heikki Kyröläinen, PhD. Heikki, I am grateful for your guidance during the final steps of my thesis. It was a privilege to have you both as my supervisors.

I had the great honor to have Laurent Mourot, PhD (Franche-Comté University, Besançon, France) and Professor Mikko Tulppo, PhD (London South Bank University, London, UK) as the reviewers of my thesis. I wish to address my sincere thanks for them for valuable comments that helped me in finishing the manuscript.

I owe my sincere thanks to my co-authors Ari Nummela, PhD, Esa Hynynen, PhD, Theresa Mann, PhD and Kaisu Martinmäki, PhD, for their valuable contribution to this study. Ari, I am grateful for your guidance and encouragement during the time I worked at KIHU. Kaisu, I am greatful for your friendship as well as valuable advice and help during the early years of my study. I also owe my sincere thanks to Sirpa Vänttinen for valuable assistance in the laboratory and for being a great colleague.

I would like to thank the entire personnel of KIHU -- Research Institute for Olympic Sports, it was a pleasure to work with you during my first years as a researcher. I would also like to thank the entire personnel of Tampere Reseach Center of Sports Medicine and UKK Institute for being great colleagues during the last years. Especially I would like to thank Jari Parkkari, MD, PhD and Tommi Vasankari, MD, PhD, for enabling the accomplishment of my thesis along with my other work.

I owe my most sincere thanks to my family and to all my great friends; I have been lucky to have you by my side supporting and giving me a perspective. Finally, I owe my deepest gratitude to my husband Tero and to our lovely children Oona, Oliver and Iida for all love and support. You remind me what really is important in life.

This study was financially supported by TEKES – National Technology Agency of Finland, the Foundation of Sports Institute, KIHU – Research Institute for Olympic Sports, the Department of Biology of Physical Activity, the Emil Aaltonen Foundation, Suunto Ltd. and Firstbeat Technologies, Ltd. I also wish to acknowledge Ritva Taipale for revision of the English language of this thesis.

Tampere, April 21st, 2015

Piia Kaikkonen

ABBREVIATIONS

BLa	blood lactate concentration
ECG	electrocardiography
EPOC	excess post-exercise oxygen consumption
FFT	fast Fourier transform
HFP	high frequency power
HFP_{ln}	natural logarithm of HFP
HR	heart rate
HR_{max}	maximal heart rate
HRR	heart rate recovery
$HR_{reserve}$	heart rate reserve
HRV	heart rate variability
LFP	low frequency power
LFP_{ln}	natural logarithm of LFP
PSD	power spectral density
RMSSD	square root of the mean of the sum of the squares of the differences between adjacent R-to-R peak intervals
RPE	rating of perceived exertion
RRI	R-to-R interval
RSA	respiratory sinus arrhythmia
SD	standard deviation
SDNN	standard deviation of normal-to-normal RR-intervals
SPWT	Smoothed Pseudo Wigner transformation
STFT	short-time Fourier transform
TFRD	time-frequency distribution
TL	training load
TP	total power
TP_{ln}	natural logarithm of TP
TRIMP	Training Impulse
$TRIMP_i$	individualized TRIMP
VLFP	very low frequency power
VO_2	oxygen uptake
VO_{2max}	maximal oxygen uptake
vVO_{2max}	velocity at which maximal oxygen uptake is reached
V_{max}	maximal velocity
W_{RCT}	work at respiratory compensation threshold

LIST OF ORIGINAL PUBLICATIONS

This thesis is based on the following original articles, which are referred to in the text by their Roman numerals.

I Kaikkonen P, Martinmäki K & Rusko H (2008) Post-exercise heart rate variability of endurance athletes after different high-intensity exercise interventions. Scandinavian Journal of Medicine and Science in Sports 18: 511–519.

II Kaikkonen P, Nummela A & Rusko H (2007) Heart rate variability dynamics during early recovery after different endurance exercises. European Journal of Applied Physiology 102, 79–86.

III Kaikkonen P, Hynynen E, Mann T, Rusko H & Nummela A (2010) Can HRV be used to evaluate training load in constant load exercises? European Journal of Applied Physiology 108, 435–442.

IV Kaikkonen P, Hynynen E, Mann T, Rusko H & Nummela A (2012) Heart rate variability is related to training load variables in interval running exercises. European Journal of Applied Physiology 112, 829-838.

CONTENTS

INTRODUCTION

Physical exercise induces several acute and long-term physiological alterations in the human body. A complex co-operation of the central and peripheral nervous systems as well as hormonal factors regulate cardiopulmonary and neuromuscular function during and after exercise. Exercise causes both rapid and reversible changes at the beginning of a single exercise session when muscles are activated as well as long-lasting adaptations to training appearing only after months or years of regular training. Some of the most well-known, important physiological adaptations to exercise include improved function of the heart and working muscles. Many of the physiological responses during exercise are regulated at the brain level, a phenomenon called "central command" (Åstrand & Rodahl, 1986; Mitchell et al. 1983). In addition, neural pathways and peripheral reflexes as well as hormonal functions take part in this regulation (Rowell & O'Leary, 1990; Strange et al. 1993). Both neural and endocrine system attempt to maintain equilibrium, so called homeostasis, of the human body during different internal or external challenges, for example physical exercise. The term homeostasis has been introduced already in the early 1900s, and later "the general adaptation syndrome" was introduced as the mechanism by which the human body reacts in the case of different challenges (Cannon, 1914). The autonomic nervous system (ANS), consisting of vagal and sympathetic subsystems, represents neural control of homeostasis. In other words, homeostasis may be described as the autonomic state that is characterized by high level of vagal tone (Porges, 1995).

The way in which the ANS responds to stress, caused by e.g. physical exercise, has been under investigation during the past century. It has been hypothesized that neurophysiological mechanisms, regulated at brainstem, either promote or limit physiological reactivity to stressful situations. Usually, procedures disturbing homeostasis result in decreases in vagal tone. If vagal tone is depressed chronically, it may lead to poor homeostasis and increased vulnerability to stress (Porges, 1992).

The function of the ANS, especially of the vagal component, may be noninvasively estimated via cardiac functions. Vagal control of the sinoatrial node

(S-A node) may be evaluated by quantifying respiratory sinus arrhythmia (RSA), a phenomenon which is characterized by a rhythmic increase and decrease of heart rate according to phases of inspiration and expiration (Porges, 1995; Task Force, 1996). The term heart rate variability (HRV) has been established to describe this fluctuation in ANS activity, estimated via the time difference between consecutive R to R intervals of an electrocardiogram (ECG).

According to the overload principle of training, the goal of training is to overload the body and cause a disturbance in homeostasis after which performance can be improved if adequate period of recovery is accomplished. Sometimes physical training load exceeds the capacity of the body to adapt, which can cause short- or long-term overload to the body. Physical training load (TL) can be identified as the total load caused either by single exercise session or longer training period, depending on exercise intensity and duration. Measurement of physiological TL has been challenging, and no practical, validated physiological method is available so far to reliably detect changes in the body as a result of physical TL.

Exercise-induced modifications in the ANS, and more specifically in the control of the cardiovascular system, have been investigated noninvasively by HRV. HRV has been found to indicate fluctuations in the ANS during different conditions, such as rest, physical exercise, and daily activities (Akselrod et al. 1985; Martinmäki et al. 2006a; Task Force, 1996). Decreased levels of HRV have been found in overtrained athletes (Baumert et al. 2006; Hynynen et al. 2007; Uusitalo et al. 1996) as well as in several diseases, e.g. increased risk for death after myocardial infarction (La Rovere et al. 1998).

Methodological issues have restricted investigations of HRV during transiently changing ANS conditions, therefore knowledge of HRV dynamics during immediate recovery after exercise sessions have remained unclear. This thesis focuses on HRV during immediate recovery after different endurance exercise sessions and attempts to find out if HRV might be used as an indicator of physiological training load, in addition to traditional training load indicators or even as an individual variable.

1 REVIEW OF THE LITERATURE

1.1 Physiological training load and body homeostasis

1.1.1 Body homeostasis and stress

The term homeostasis was introduced by Cannon already in early 1900s. Stress, in turn, can be broadly defined as an actual or anticipated disturbance of homeostasis. When the internal needs of organs are no longer being adequately responded to by the vagal system (decreased vagal tone), an organism is experiencing stress. Furthermore, if vagal tone is decreased chronically, homeostasis is poor and stress vulnerability increased. Clinically, a lack of endogenous variability in neurally mediated peripheral systems, such as heart rate, can be seen a sign of severe physiological compromise (Porges, 1992).

The neural and endocrine systems of the human body help in integrating and controlling bodily functions, providing stability to the internal environment of the body for example during different internal or external demands. Hormones take part in regulating growth and reproduction and enhance the ability of the body to adapt to psychological and physical stress. The maintenance of internal equilibrium, homeostasis, is also regulated via hormonal functions and the autonomic nervous system (ANS), which provide the most immediate response to stress exposure. Sympathetic and vagal branches provoke rapid alterations in physiological states through neural innervation of end organs. Compared to the rapid responses of the ANS, a more gradual regulation of homeostasis occurs via the hypothalamic-pituitary-adrenocortical axis (HPA), which takes part for example in controlling circulating glucocorticoids (Ulrich-Lai & Herman, 2009). In the present study, the main focus is the ANS as a regulator of body homeostasis.

1.1.2 Physiological training load in sports and exercise

The main goal of sport coaches and athletes is to facilitate biological adaptations to achieve the best possible performance at the right time. To achieve the optimal training effect and to improve performance, the overload principle of training must be carried out. This means that when the homeostasis of cells, tissues, and organs are appropriately, that is more than they are used to, disturbed and overloaded, training adaptations occur and enable the body to function more efficiently. Physical exercise or training may be interpreted as a stimulus that causes this disturbance of homeostasis, which should be restored during recovery after a given training session (Borresen & Lambert, 2009). This leads to supercompensation, which is improved resistance to stress, and to improved performance if sufficient recovery after exercise is accomplished. Thus, the principle of training may also be thought as a "dose-response" relationship between the physiological stress associated with the load of exercise training (dose) and the training adaptations (response), where the latter one may be measured quite easily as an improvement in performance. So far, there is no single, practical, validated parameter to measure the physiological training load (TL) of exercise (Borresen & Lambert, 2008b). Physical TL can be estimated for example with diaries or calculating the total volume of training; sessions, intensities and durations of exercises. In the present study, the focus is in estimating the physiological TL of single exercise. Physiological TL could be defined as the exercise-induced modifications in the internal environment of the body (homeostasis), which might be detected in cardiac autonomic modulation with HRV.

It is generally believed that increased physical TL results in an improvement in sports performance. However, in many cases the approach of unsystematic increases in training intensity, duration, and/or frequency might increase the likelihood for injury and symptoms of overreaching or overtraining (Borresen & Lambert, 2009), which can be detected in the function of the ANS (Hynynen et al. 2006; Uusitalo, 2001). In order to produce optimal adaptations, physical TL and recovery must be balanced so that neither under- nor overtraining exists. In other words, overreaching/overtraining can be identified as underrecovery. The symptoms of overreaching/overtraining are often miscellaneous, and can include impaired performance, increased resting heart rate, hormonal changes, restlessness, irritability, disturbed sleep, weight loss, loss of appetite and depressed mood (Kellmann, 2010; Uusitalo et al. 1996). Functional overreaching (FOR) may be defined as a "normal" status of an athlete during a short-term intensified training period, after which performance can improve if the subsequent resting period is adequate. The difference between the definitions of non-functional overreaching (NFOR) and more severe overtraining syndrome (OTS) are usually considered as the time needed for recovery. NFOR is considered as a more short-term condition, from which recovery may take several days or weeks, whereas recovery after OTS may take years, at worst (Meeusen et al. 2012; Uusitalo, 2001).

1.1.3 Measures for evaluating physiological training load

For now, there is no single practical, validated physiological parameter that could be used as a "golden standard" to estimate physiological TL in field conditions. Several parameters have been used on different occasions to estimate physiological TL and some of these parameters are shortly introduced in the following chapters.

Heart rate (HR). Heart rate is the most popular method for measuring exercise intensity during exercise. It is based on the principle that there is a linear relationship between heart rate and steady state work rate; heart rate increases as a result of increased exercise intensity (Arts & Kuipers, 1994; Åstrand & Rodahl, 1986). Exercise intensity can be estimated, in addition to absolute values of HR, as a percentage of maximal HR, or as a percentage of heart rate reserve ($HR_{reserve}$), according to Karvonen et al. (1957) (% of $HR_{reserve}$ = ($HR_{exercise}$-HR_{rest}) × 100 / HR_{max}-HR_{rest}). HR level during exercise is affected mainly by exercise intensity. However, when a continuous submaximal exercise is continued for a prolonged time, cardiovascular drift, i.e. a fall in stroke volume, is known to occur. This is characterized as a rise in HR, which may be related to increased core temperature, increased sympathetic nervous system activation and dehydration (Coyle & González-Alonso, 2001).

In addition to HR during exercise, the energetic demands of an exercise, i.e. physiological TL, may be estimated by immediate HR recovery after exercise. HR recovery in healthy subjects may be affected by several factors, for example exercise intensity, training background, and training status. Lamberts & Lambert (2009) investigated the day-to-day variation in submaximal and recovery HR and reported that the daily variation in HR decreased as the exercise intensity increased, reaching the lowest variation (5 ± 2 bpm) at the highest (85-90 % of HR_{max}) intensity. During recovery, the lowest variation (8 ± 3 bpm) was found during the first recovery minute after exercise cessation. In addition, Lamberts et al. (2009a) found improved heart rate recovery after a high-intensity training period in well-trained athletes, concluding that it may be used as sensitive method for monitoring training. This improvement in HR recovery was more obvious in the group of subjects who improved their performance (Lamberts et al. 2009b). Borresen & Lambert (2007) also investigated the effects of increased physical TL on heart rate recovery immediately after exercise during a two-week training program, and found a negative correlation between heart rate recovery and physical TL.

Altogether, there are environmental (e.g. temperature and humidity), physiological (e.g. state of training, dehydration) and psychological factors that may affect the relationship between exercise intensity and heart rate during exercise. Despite these, heart rate is an easy way to measure intensity during exercise, but the confounding factors during exercise and also its fast recovery near resting levels after exercise may weaken its sensitivity in detecting differences in physiological TL between different exercises.

Excess post-exercise oxygen consumption (EPOC). During exercise, oxygen uptake increases as a response to the increased demand for energy. After exercise cessation, oxygen consumption does not recover to resting levels immediately, but remains elevated above the resting level for a period of time, from minutes to hours. This elevated oxygen uptake was former discussed as 'oxygen debt' and thought to be necessary for repayment of the oxygen deficit incurred after the start of exercise. Recently, this hypothesis has been modified. In addition to oxygen debt, mechanisms underlying the rapid component of excess post-exercise oxygen consumption (EPOC) consist of ATP/CP resynthesis, replenishment of oxygen stores, lactate removal, increased body temperature, ventilation, and circulation. The mechanisms behind the prolonged component are unclear, but increased sympathoadrenal activity may be one mechanism underlying prolonged EPOC (Børsheim & Bahr, 2003).

Exercise intensity has been shown to be the main determinant of EPOC, and its effect has been found to be curvilinear, whereas the effect of exercise duration is more linear. However, the interaction between intensity and duration is not completely understood, and their effects are difficult to separate from each other. It has been suggested that some "limit" of intensity exists, beyond which the duration of exercise linearly increases EPOC. Gore & Withers (1990) have suggested that this limit is somewhere near 50 % of VO_{2max}, as increased exercise duration at 50 % VO_{2max} increased EPOC but at 30 % of VO_{2max} did not affect EPOC. Furthermore, they found that at the intensity of 70 % of VO_{2max}, duration of exercise increased EPOC even more than at 50 %. When total work is similar, continuous and interval exercises have been found to produce similar effects on EPOC (McGarvey et al. 2005).

EPOC might be used as some kind of reference method in the estimation of physiological TL, due to its physiological background. The disadvantage of EPOC is that the measurements demand laboratory conditions or equipment and are therefore not suitable for field conditions. Also the definition of the baseline of EPOC is demanding, and may differ between studies, which also naturally affect the analysis of EPOC.

Blood lactate (BLa). Blood lactate concentration can be seen as an index of anaerobic metabolism during exercise. During light to moderate activity, aerobic processes play a major role in energy output with blood lactate rising only slightly above the resting levels. With more intense exercise, lasting longer than the capacity of high-energy phosphate compounds (ATP/CP), anaerobic processes contribute to energy output, which predominates during high-intensity exercise. During high-intensity exercise, the rate of blood lactate production exceeds the rate of removal, resulting in increasing concentration of lactate in the blood (Åstrand & Rodahl, 1986). There are many confounding factors affecting lactate response to exercise that do not support the use of lactate measurement as an estimate of total physiological TL, such as ambient temperature, amount of carbohydrate ingestion, muscle damage, preceding exercises, mode of the present

exercise, and status of the intrinsic buffering system. Also measurement errors due to earlobe or fingertip blood collections exceed the possible changes expected as a result of exercise intensity (Lambert & Borresen, 2010).

Rating of perceived exertion (RPE). A subjective estimation of perceived exertion (RPE) has been used to estimate exercise intensity and TL. RPE has been found to correlate with the level of cardio-respiratory and metabolic demands (Noble, 1982; Skinner et al. 1973), and to integrate information, like signals from the central nervous system (Borg, 1982). As a subjective parameter, in addition to exercise intensity, RPE also takes exercise duration into account. The physiological mechanisms behind the cognitive perception of effort are unclear, and so it is not clear what exactly RPE represents. Many factors affect the personal perception of physical effort, including hormone concentrations (e.g. catecholamines), substrate concentrations (glucose, glycogen and lactate), personality traits, ventilation rate, neurotransmitter levels, and psychological states. These factors limit the use of RPE in an accurate quantification of exercise intensity (Borresen & Lambert, 2009).

Session RPE (RPEs). Foster et al. (1998) introduced the use of session RPE in order to quantify TL. Session RPE is a rating of overall difficulty of a bout of exercise obtained 30 minutes after the completion of the exercise. Session load is calculated by multiplying session RPE by session duration of aerobic exercise in minutes (Foster, 1998). As duration is already taken for some part into account in RPE (Borg, 1982), session RPE might overestimate the effect of exercise duration in overall difficulty. Similar to RPE, differences in personal perception of physical effort attenuate the accuracy of RPEs in TL measurement. However, session RPE has been found to correlate well with heart-rate based methods of quantifying TL, although at this time they are not validated (Borresen & Lambert, 2008b).

Training Impulse (TRIMP). Banister (1991) introduced a method to quantify training session as a "dose" of physical effort. He suggested that heart rate response along with the exercise duration, called training impulse (TRIMP), may be a plausible measure of physical effort. This is based on the increase in heart rate between resting and maximal levels (Banister, 1991). A limitation of TRIMP is the demand of steady state heart rate measurement, thus limiting the accuracy of TRIMP during interval exercises. Manzi et al. (2009) tested the hypothesis that in long-distance athletes, changes in ANS parameters are dose-response related to individual volume/intensity TL. They used individualized TRIMP ($TRIMP_i$) to monitor TL of athletes. In $TRIMP_i$, in addition to heart rate, individual lactate production affects the estimation of TL. The use of standard weighting factor (Y) in TRIMP, based on a fixed lactate-workload relationship, might be inappropriate for quantifying TL for subjects with different training status as the relationship between lactate and work load may change with im-

provements in training status but also with overtraining (Borresen & Lambert, 2008b).

Retrospective questionnaires and diaries have been used to obtain data from athletes regarding their physical activity during the last weeks or months. The advantages of questionnaires and diaries are easy administration, cost effectiveness, and the fact that they do not disturb training. However, 24 % of athletes have been reported to overestimate and 17 % to underestimate the duration of their training (Borresen & Lambert, 2009; Hopkins, 1991), so the accuracy of this method in TL analysis is quite poor.

1.1.4 Individual differences in response to training

Exercise-induced physiological responses and adaptations vary significantly between individuals. The interindividual variance might be explained by several factors, such as age, sex, training history, psychological factors, initial training status, recovery potential, exercise capacity, non-training stress factors, stress tolerance, and genetics (Borresen & Lambert, 2009). For instance, a mean increase of 25 % in endurance performance during a standardized training program hides the fact that some individuals may have improved 50 % or even more, whereas others may not improve at all. Age, sex, and ethnic origin seem to have only minor effect on human responses to regular physical activity, whereas the initial training status and genetic factors are significant contributors to "trainability" of individuals (Bouchard & Rankinen, 2001). No single marker has been identified that can accurately assess how an athlete is adapting and responding to a training programme (Borresen & Lambert, 2008a), but the resting level of HRV has been found to correlate with response to aerobic training in healthy sedentary subjects (Hautala et al. 2003).

1.2 Heart rate variability (HRV) as a tool for estimating cardiac modulations in the autonomic nervous system

1.2.1 General function of the autonomic nervous system

The autonomic nervous system (ANS) controls most of the internal, visceral functions of the body. It regulates the internal environment of our body to maintain homeostasis by responding to external challenges (Porges, 1992). It is activated mainly by centers located in the spinal cord, brain stem, and hypothalamus. In addition, parts of the cerebral cortex and limbic systems affect autonomic control by transmitting impulses. Autonomic signals are transmitted to the organs through two subdivisions, the sympathetic and parasympathetic (vagal) systems (Hainsworth, 1998). In general, the vagal system promotes functions associated with growth and restoration, while the sympathetic system, in contrast, promotes increased metabolic output to deal with challenges from

outside the body (Porges, 1995). Both sympathetic and vagal pathways consist of two fibers, pre- and postganglionic neurons. Nerve endings secrete one of the two synaptic transmitter substances, acetylcholine or norepinephrine. Fibers that secrete acetylcholine are said to be cholinergic and fibers that secrete norepinehrine, adrenergic. All preganglionaric neurons of sympathetic and vagal branches, as well as postganglionic neurons in the vagal branch, are cholinergic. Most of the postganglionic sympathetic neurons are adrenergic. The sympathetic and vagal systems are continually active, and the basal rates of activity are known as sympathetic tone and vagal tone, respectively (Hainsworth, 1998).

1.2.2 Autonomic control of the heart

In addition to several other physiological functions, the autonomic nervous system also controls cardiovascular function. The heart is supplied with both sympathetic and vagal nerves, which affect cardiac pumping by changing the heart rate and the strength of contractions of the heart. Cardiac impulses are controlled by the membrane processes of sinoatrial (S-A) node, which is the pacemaker of the heart (Berntson et al. 1997; Hainsworth, 1998). The intrinsic heart rate of a human, modulated by the S-A node, varies between 110-120 bpm at rest (Hainsworth, 1998). In general, increased sympathetic activity increases the overall activity of the heart, and vagal activity causes mainly opposite effects. Thus, heart rates below the intrinsic rate indicate that there is vagal predominance, as is the case of resting in which the resting heart rate is normally between 60-80 bpm. The vagal nerves are distributed mainly to the S-A and atrioventricular (A-V) nodes, and to a lesser extent also to the atrial and ventricular muscles. Sympathetic nerves spread to all parts of the heart, also largely to the ventricles. Vagal nerve activation releases acetylcholine from the vagal nerve endings, decreasing the rate of S-A impulses, leading to a decrease in heart rate. Vagal stimulation has a very short latency, the maximum response occurring in only 400 ms. Therefore, changes in vagal activity can influence heart rate on a beat-to-beat basis, whereas the maximum sympathetic effect may not occur for as long as 20-30 s (Hainsworth, 1998).

Baroreceptors. In addition to atrial and ventricular receptors, which influence cardiac filling as well as heart rate and blood pressure, there are also other important reflexes that control cardiac autonomic nervous activity. Baroreceptors are located in the walls of arteries, for example in the carotid sinuses, aortic arch, and coronary arteries. They are very sensitive to changing pressures, and respond with a rapid discharge when the vessel is stretched by an increase in blood pressure (Hainsworth, 1998). This discharge has been found to occur within 0.75 s, indicating that baroreceptors control heart rate on a beat-by-beat basis, and that their function is vagally mediated (Eckberg, 1976). Thus, stimulation of baroreceptors increases vagal activity and inhibits sympathetic activity, while abnormalities in baroreceptor function in a variety of pathological conditions (e.g. hypertension, cardiac diseases) may lead to sympathetic excitation and vagal withdrawal.

Under resting conditions, an increase in blood pressure usually decreases heart rate through the baroreceptor reflex mechanism. In contrast, an increase in blood pressure during exercise is accompanied by a concomitant increase in HR, which is an important factor in the increase of blood pressure (Iellamo, 2001). As blood pressure levels change, as is the case during exercise, baroreceptors are suggested to reset so that their range of function adapts according to the prevailing level of pressure, indicating that baroreceptor function is more about stabilizing fluctuations in blood pressure rather than about determining an absolute level. There is also an interaction between the baroreceptor reflex and respiratory activity, as baroreceptor function is almost completely inhibited during early inspiration, especially during rapid breathing (Eckberg, 1980). Physical exercise and mental arousal are associated with a reduction in baroreflex control of heart rate, while control of blood pressure is left relatively unchanged (La Rovere et al. 1998). Some gender differences may also exist in the function of baroreflexes. For example Huikuri et al. (1996) found that women had significantly lower baroreflex sensitivity during the Valsalva maneuver than men.

Chemoreceptors. Peripheral chemoreceptors are found in the carotid bodies, located near the carotid sinuses, and in the aortic bodies adjacent to the arch of the aorta. Central chemoreceptors are located in the floor of medulla. Main function of chemoreceptors is to control respiration, and they become strongly stimulated during arterial hypoxia and hypercapnia. They also affect cardiovascular system, as stimulation of central chemoreceptors increases sympathetic activity to the heart and blood vessels. Stimulation of carotid and aortic chemoreceptors induces primary reflex bradycardia and increased sympathetic activity, respectively (Hainsworth, 1998).

1.2.3 Basis of HRV

Heartbeat interval, as well as blood pressure interval, varies secondary to respiration, a phenomenon called 'respiratory sinus arrhythmia' (RSA). According to Berntson et al. (1997), the first documented observation of heart rate variability (HRV) is credited to Hales, who observed a respiratory pattern in the blood pressure and pulse of a horse in 1733. Later in 1847, Ludwig found a regular increase in pulse with inspiration and a decrease with expiration in dog. Further, clinical research interest in RSA increased in the 1960s when Wolf (1967) and Hon & Lee (1963) emphasized the relationship between heart rate variability and nervous system status (Berntson et al. 1997). RSA is reported to be modulated by vagal activity, as it disappears after vagal blockade with atropine (Akselrod et al. 1985; Cacioppo et al. 1994; Hayano et al. 1991; Katona & Jih, 1975; Uusitalo et al. 1996). It is associated with several parasympathetic parameters, such as central vagal outflow to the heart, cardiac vagal tone, baroreflex activity, and the phasic respiratory modulation of vagal activity (Berntson et al. 1997).

The simplest way to demonstrate the effects of autonomic modulation on the heart is to monitor the function of the S-A node, i.e. the changes in heart rate. Originally, HRV was quantified as a time-domain, i.e. R-R intervals in milliseconds plotted against time (Figure 1). Increased vagal activation is characterized by decreased HR and increased variability of HR, whereas the effects of increased sympathetic activity increases HR and decreases variability of HR. The term heart rate variability has become the conventionally accepted term to describe the variation of both instantaneous HR and RR intervals. As HRV is based on the analysis of fluctuations in R-R-intervals (ventricular depolarisations) and not P-P-intervals (atrial depolarisations), it actually does not measure only the rhythms of S-A node, but also the fluctuations in atrioventricular conduction (PQ-interval). However, as a result of complex function of cardiac autonomic regulation, HRV assessed from RR-intervals during sinus rhythm very accurately reflects the variability of the rhythm of the S-A node (Hartikainen et al. 1998).

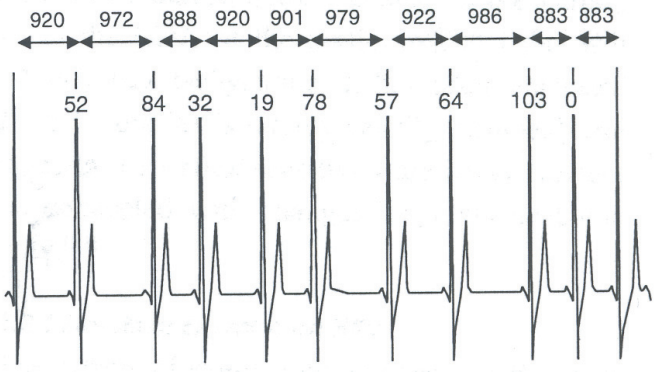

FIGURE 1 An example of an ECG signal. RR-interval times and differences between adjacent RR-intervals are displayed. (Achten & Jeukendrup, 2003).

1.2.4 Factors affecting HRV

HRV is a sensitive non-invasive method estimating modulations in the activity of autonomic nervous system, which occurs mostly without conscious actions of an individual. Therefore it is reasonable that numerous factors affect the individual magnitude of resting HRV. Some of the most important factors are shortly discussed in the following paragraphs.

A significant **genetic** contribution to HRV has been established in laboratory twin and family studies (Kupper et al. 2004; Singh et al. 1999; Uusitalo et al. 2007). A genetic contribution of 13-39 % of resting HRV has been reported, increasing up to 51 % during exposure to mentally and emotionally demanding tasks. An ambulatory 24 h recording revealed that the mean genetic contribution of SDNN and RMSSD varied from 35 to 47 % and from 40 to 48%, respec-

tively (Kupper et al. 2004). Martinmäki et al. (2006b) investigated the intraindividual variation of HRV indices to measure modulation of vagal activity and found a large variation in the quantitative relationship between vagal component (high frequency power, HFP) of HRV and vagal effects on the heart. Comparison of subjects with and without endurance training background indicated similar within subject linear increases in HRV variables after vagal blockade (Martinmäki et al. 2006b).

HRV is known to decline with **age** (Byrne et al. 1996; Fukusaki et al. 2000; Moodithaya & Avadhany, 2012). It has been concluded that age-related changes in HFP, reflecting vagal modulation, are primarily mediated by aging per se and not by physiological changes like physical fitness, body composition or blood pressure, which are characteristics of normal aging (Byrne et al. 1996; Fukusaki et al. 2000). Stiffening of the aortic wall is likely to decrease the sensitivity of aortic baroreceptors in the elderly, which probably increases the blood pressure and decreases heart rate variability. Vagal blockade results in a lesser increase in heart rate of the elderly when compared to young healthy individuals, and the age-related decline of heart rate variability is generally attributed to a decline in vagal activity (Semrád et al. 1998).

The differences in HRV between men and women have been investigated to some extent. Female **gender** seems to result in a lower total variability (Vandeput et al. 2012), whereas vagal modulation has been found to be similar (Moodithaya & Avadhany, 2012) or higher (Fukusaki et al. 2000; Huikuri et al. 1996) when compared to men. These differences in HRV are probably caused by lower sympathetic and higher vagal control of the heart in women (Carter et al. 2003).

During a supine rest, there is a vagal predominance, resulting in increased HRV when compared to a standing **posture.** In contrast to rest, the effect of body posture on HRV has been found to disappear during the immediate recovery after a high-intensity exercise session, when a time-varying vagal-related index ($RMSSD_{30}$) was used (Buchheit et al. 2009)**.**

Several **diseases** have been found to decrease HRV, and it is also an independent predictor of death among cardiac patients (La Rovere et al. 1998). Reduced 24-h HRV has been found as a risk factor for sudden cardiac death also in healthy individuals (Mølgaard et al. 1991).

In addition to previous mentioned factors, also several others, e.g. smoking, overweight, sleep stages, mental stress, breathing frequency and some medicines may affect the magnitude of HRV.

1.3 Effect of endurance exercise and training on cardiac functions

1.3.1 Cardiovascular function and HRV during endurance exercise

Endurance exercise may be defined as a conscious period of physical activity, where large groups of muscles are working continuously, usually from minutes to hours. Three mechanisms have been introduced as playing a role in neural cardiovascular regulation during exercise. The greatest control of the heart and blood vessels during exercise is provided by central command, originating from the medulla, activating parts of the brain responsible for the recruitment of motor units of skeletal muscles (Hainsworth, 1998; Iellamo, 2001). Secondly, exercise pressor reflex, i.e. reflex neural input from chemosensitive metaboreceptors of active muscles activate cardiovascular control areas in the medulla, regulating blood flow and blood pressure during exercise (Iellamo, 2001; Strange et al. 1993). Metaboreceptors of working muscles contribute to the sustained vagal inhibition as well as the sympathetic excitation during exercise. These receptors are also responsible for the inhibition of the baroreceptor reflex (Hainsworth, 1998), which is the third neural mechanism contributing to regulation of cardiovascular function during exercise (Iellamo, 2001).

Immediately within the first few seconds after the onset of exercise, coronary blood flow is increased due to vasodilatation of the arterioles. This is a result of oxygen deficiency of the heart as well as the effect of increased adenosine, an outcome of the breakdown of ATP, in the heart. The increase in the heart rate immediately after the onset of exercise is attributed mainly to vagal withdrawal. Furthermore, during progressive dynamic exercise, central input of the brain initiates a decrease in vagal activation that occurs simultaneously with an increase in sympathetic activity (Åstrand & Rodahl, 1986). In response to increased activation from sympathetic nervous system (SNS), adrenal glands secrete epinephrine and norepinephrine, which increases heart rate and contraction force. Circulating sympathetic catecholamines function both as neurotransmitters and as hormones, by increasing the pumping capacity of the heart and by constricting vessels (except in the working muscles) and increasing cardiac output as well as heart rate frequency to the high levels required during exercise (Mazzeo, 1991).

When expressed in either absolute frequency domain (HFP, LFP, TP) or time domain (e.g. SDNN) methods, HRV is greatly diminished during exercise when compared to rest (Arai et al. 1989; Casadei et al. 1995; Macor et al. 1996; Perini et al. 1990; Pichon et al. 2004). HRV decreases as a function of exercise intensity, almost disappearing at exercise intensities exceeding 50–60% of VO_{2max} (Hautala et al. 2003; Tulppo et al. 1998) or the ventilatory threshold (Yamamoto et al. 1991). Therefore, intensity seems to be the main factor affecting HRV during exercise.

As presented in the previous chapters, it seems likely that there are more than just autonomic mechanisms contributing to the modulation of heart rate

during exercise. Kamath et al. (1991) speculated that hormonal factors, such as circulating catecholamines, probably play a more dominant role than neural input in maintaining tachycardia during a steady-state exercise, because low frequency HRV was significantly diminished during exercise, which is in contrast to standing position during an orthostatic test. On the other hand, the origin of LPF may be different during exercise when compared to rest. Also during heavy exercise, heart rate variability may be influenced by other factors than the ANS. Mechanical or neurological coupling between the cardiac, locomotor and respiratory systems could play an important part in the observed changes (Bernardi et al. 1990; Casadei et al. 1996; Casties et al. 2006; Pichon et al. 2004).

1.3.2 Heart rate and HRV during recovery after exercise

Heart rate. An increase in vagal activation as well as a decrease in sympathetic activation has been detected already during the first minutes of the recovery (Savin et al. 1982). It has been suggested that the immediate first minute decline in HR after exercise cessation is mainly vagal in origin (Arai et al. 1989; Imai et al. 1994; Perini et al. 1989; Savin et al. 1982), although the relative role of sympathetic and vagal divisions seems to depend on exercise intensity (Imai et al. 1994; Perini et al. 1989). The recovery of heart rate has widely been investigated in clinical studies, and its prognostic value in different heart diseases has been confirmed. A delayed decrease in heart rate during the first minute after graded exercise was found to be a powerful predictor of overall mortality in the study of Cole et al. (1999). In addition, Nissinen et al. (2003) found that the slow recovery of heart rate during the first recovery minute after exhaustive exercise was related to increased amount of deaths among patients with former myocardial infarct.

Heart rate variability. The immediate recovery of HRV has been investigated in few studies during the past years. In many studies, the first five recovery minutes have been excluded from analysis due to methodological issues. The frequency domain methods are perhaps the most widely used in exercise/training studies investigating HRV, but they have methodological limitations that inhibit their use during changing autonomic conditions, like immediate recovery after exercise (Task Force, 1996). In the few studies that have investigated HRV recovery during the first minutes of recovery, HRV has been found to be significantly lower than resting values, and either starting to increase already during the first recovery minutes (Casties et al. 2006; Goldberger et al. 2006; Martinmäki & Rusko, 2008) or remaining blunted for several minutes before recovery begins (Buchheit et al. 2007a; Oliveira et al. 2013). Differences in the results may be, at least partly, explained with differences in the physical TL of the preceding exercises as well as in the training status of the subjects.

Casties et al. (2006) studied HRV of male cyclists before, during and 50 minutes after a graded 30 min exercise session at 40–90% VO_{2max}. According to

their results, the increase of HRV, $RMSSD_{30s}$ to be more specific, began already during the first 10 min of the recovery and further increase was observed during the succeeding 50 min of recovery. At the end of the recovery period, HRV still remained significantly lower than the resting values. Immediate post-exercise recovery has been studied also by Goldberger et al. (2006), who investigated HRV after a graded maximal exercise in normal subjects and subjects with coronary artery disease. They found an increase in the time domain parameters of HRV during the first 5 min of recovery and concluded that HRV (especially RMSSD and RMS, root mean square successive difference of the R–R intervals and root mean square residual, respectively) can be used in the assessment of parasympathetic reactivation immediately after maximal exercise.

Martinmäki & Rusko (2008) investigated the effects of low (10 min at 30 % VO_{2max}) and high (10 min at 60 % VO_{2max}) intensity exercises on immediate 10 min HRV recovery, and found a rapid increase in HFP and LFP during the first minute after low-intensity and during the first two minutes after high-intensity exercise. They concluded that restoration of autonomic control of HR is slower after exercise with a greater metabolic demand (Martinmäki & Rusko, 2008). In addition to Martinmäki & Rusko (2008), Buchheit et al. (2007a) concluded that parasympathetic reactivation appears to be mainly related to anaerobic process. They studied the effects of high-intensity sprint exercises on immediate 10 min HRV recovery of trained athletes, and found highly impaired vagal reactivation during the first minutes of recovery. According to previous studies (Hautala et al. 2001; Javorka et al. 2002; Oida et al. 1997; Seiler et al. 2007; Takahashi et al. 2000), HRV increases slowly towards resting values during recovery after single exercise session, but the effects of endurance exercise may last from minutes (Gladwell et al. 2010; Seiler et al. 2007) to even 48 hours (Mourot et al. 2004), depending on physical TL, i.e. the combined effect of exercise intensity and duration, of the exercise as well as the training status of the subjects.

In general, exercise intensity has been found to be the main factor determining the recovery of HRV. There is usually a negative relationship between exercise intensity and HRV during recovery after exercise. In addition to exercise intensity, exercise duration may have an effect on HRV recovery. However, the effects of prolonged endurance exercise on post-exercise HRV have barely been studied. Seiler et al. (2007) studied the effects of prolonged exercise on immediate post-exercise HRV, and found no effect of doubled exercise duration from 60 min to 120 min in highly trained subjects, when exercises were performed below the first ventilatory threshold (77 % of VO_{2max}). This result could have been different in subjects with different training status.

Nocturnal HRV has been studied during the night after endurance exercise in order to determine the long-term effects on autonomic functions. Hynynen et al. (2010) studied the effects of single moderate and heavy (marathon) endurance exercise sessions on nocturnal HRV, and found that both sessions affected nocturnal HR and HRV, HFP being only 77 % and 34 % of the nocturnal values of a resting day after moderate and heavy exercises, respectively. Hautala et al. (2001) also reported decreased HRV during the night fol-

lowing a 75 km cross-country ski-race, followed by a rebound phenomenon during the second night after the race. They also reported large interindividual variation in the recovery time of HFP to the pre-race level after the ski-race (0-14 h, mean 8.2 h). According to the results of Hautala et al. (2001), subjects with better cardiorespiratory fitness had a more rapid recovery.

Interpretation of HR and HRV during recovery. The origins of post-exercise HR and HRV recovery have been discussed, as the physiology of cardiac functions differ from resting. Possible mechanisms for the fast changes in cardiac autonomic modulation after exercise cessation include fast changes in cardiac preload, after-load and contractility of the heart (Miles et al. 1984; Plotnick et al. 1986). Together with the loss of central command and baroreflex activation, these mechanisms contribute to fast vagal reactivation after exercise cessation (Oida et al 1997; O'Leary, 1993). Despite the presumed vagal origin of both HR and HRV recovery, some studies have failed to find a relationship between the results of HR and HRV recovery (Buchheit et al. 2007b; Kannankeril et al. 2004). Buchheit et al. (2007b) suggested that HR recovery after submaximal exercise might describe the vagal tone per se, whereas post-exercise HRV reflects the magnitude of modulation in vagal outflow. Kannankeril et al. (2004) in turn suggests that sympathetic withdrawal is the major contributor to early HR recovery.

Pre-exercise HRV at rest has not been found to explain HR recovery after exercise (Javorka et al. 2002), but high modulation of cardiac vagal tone after exercise was associated with high baseline cardiac vagal tone and good aerobic fitness (Tulppo et al. 2011). In addition, modulation of cardiac vagal tone was found to be augmented in subjects who had higher peripheral sympathetic outflow in the recovery phase after exercise (Tulppo et al. 2011). Ng et al. (2009) validated both time- and frequency domain methods of HRV analysis with and without autonomic blockades during immediate recovery and found, similar to resting conditions, fluctuations both in high- and low-frequency bands. RMSSD was the only parameter showing opposite changes with β-adrenergic and vagal blockade. Both low- and high frequency fluctuations were significantly decreased after vagal blockade, but did not change after β-adrenergic blockade. In addition, LF/HF ratio was significantly decreased only by double blockade, and thus cannot be used as an indicator of sympathovagal balance during recovery. In addition to the expected findings after blockades, time- and frequency domain measures were highly correlated and could be used in HRV analysis during recovery conditions. During immediate recovery, both LFP and HFP seemed to indicate changes primarily in vagal activity (Ng et al. 2009).

1.3.3 HRV and endurance training

Training may lead both to anatomical and physiological adaptations of the cardiovascular system. Endurance training usually results in increased vagal control of the heart, but cardiovascular responses depend on the type and the intensity of exercise training (Scheuer & Tipton, 1977). Training may cause sinus

bradycardia, that is, a low resting heart rate as well as increases in left ventricular wall thickness and mass. These changes are often referred as "athlete's heart". Endurance exercise also reduces the metabolic load on the heart during rest and at submaximal exercise intensities, because of increased stroke volume that improves the heart's ability to pump blood. Increased stroke volume of the heart results primarily from an enlarged left ventricular chamber and also from increased end-diastolic volume (Cohen & Segal, 1985; Urhausen & Kindermann, 1999). In addition to anatomical changes, endurance training may also cause different functional changes, such as arrhythmias and abnormalities in atrioventricular conduction. Sinus bradycardia is often affiliated with increased vagal activity, but can also be related to intrinsic electrophysiological adaptations of sinus node automaticity (Stein et al. 2002).

The effects of endurance training on HRV have been investigated for some time. Usually training has been found to increase, but also decrease or have no effect on resting HRV (Hynynen et al. 2007; Pichot et al. 2000; Sandercock et al. 2005a; Uusitalo et al. 1996). Differences in the results are probably due to differences in the exercise modes and intensities, length of the training period, and training status of the subjects. Non-functional overreaching or overtraining syndrome has usually been found to decrease resting HRV (Uusitalo et al. 1996; Baumert et al. 2006; Hynynen et al. 2007; Pichot et al. 2000). Studies that have reported increase in VO_{2max} of more than 12 ml/kg/min after endurance training have also reported an increase in vagal control of the heart (Carter et al. 2003). Several studies have investigated the effects of training on nocturnal HRV, as sleep is an important period for both physiological and psychological recovery, and also offers reproducible, "controlled" measurement period for HRV, especially of high frequency power. Usually an overreaching, hard training period is found to decrease nocturnal HRV (Hynynen et al. 2007; Pichot et al. 2000). Also a link between endurance performance and nocturnal HRV has been found in the study of Nummela et al. (2010). The effects of a four-week endurance training period on post-exercise nocturnal HRV and on endurance performance were investigated and a link was found between the improved maximal running speed and increased nocturnal HRV when the subjects were divided into responders and non-responders according to the improvements in maximal treadmill test (Nummela et al. 2010).

Similar results to Nummela et al. (2010) were found by Buchheit et al. (2010) who studied the relationship between endurance performance and HRV by investigating the effects of an 8-week training period to HR-derived indices. They found increased resting vagal-related indices in those subjects who decreased their running time in a 10 km test run and were qualified as "responders" to the training program. Similar changes were found in post-exercise HRV results. They concluded that there is a potential of resting, exercise and post-exercise HR measurements for assessing the impact of aerobic training on endurance running performance. In addition to resting HRV, endurance training may also have an effect on the recovery of HR and HRV after exercise (Guerra et al. 2014; Seiler et al. 2007). Seiler et al. (2007) found delayed HRV recovery

after high-intensity exercises in trained subjects as compared to their highly trained counterparts. This finding is supported by similar findings between training status and recovery of HR, but the associations between training status and HRV needs to be studied further.

1.4 Methods for analyzing HRV

Before any calculations of HRV can be done, the RRI signal must be edited and corrected for ectopic beats, arrhythmias and noise. After that, normal intervals between successive QRS complexes (RR intervals, RRI) can be determined. RRI signal is commonly expressed in milliseconds (ms), from which the transformation to heart rate (bpm) is *bpm=60000/ms* (Saalasti, 2003). The nonlinear relationship between heart rate and RRI is presented in the Figure 2.

According to mathematical processing, HRV analysis methods can be divided into two methods; time-domain and frequency-domain methods. Further, time-domain methods can be divided into statistical and geometric methods, frequency-domain methods into parametric and nonparametric methods (Task Force, 1996).

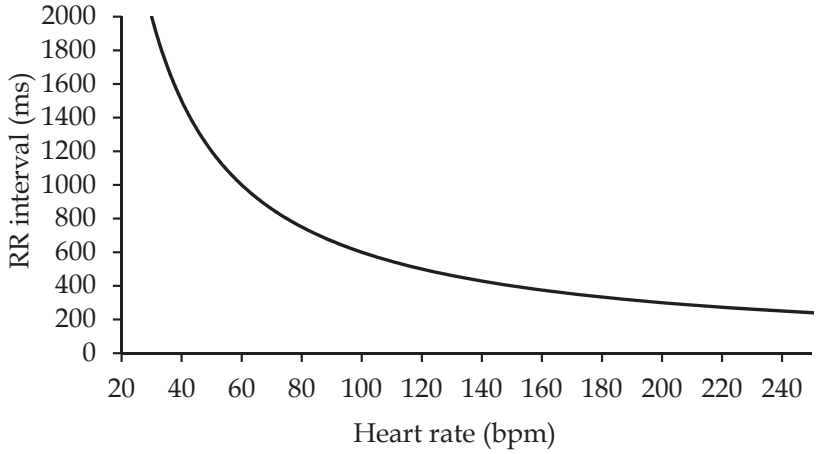

FIGURE 2 The nonlinear relationship between heart rate and RRI.

1.4.1 Time domain analysis

Perhaps the simplest methods to evaluate heart rate variability are time domain analyses. In general, the time domain methods are ideal for the longer HRV recordings, but they can be used in recordings of shorter duration, too. One part of time-domain methods is based on the determination of the intervals of the

successive normal R-R complexes (normal-to-normal, NN) resulting from sinus node repolarization. The standard deviation of normal RR-intervals (SDNN) over the selected time period might be the simplest time-domain analysis, although its reproducibility in long-term recordings has been found to be questionable (Tarkiainen et al. 2005). This represents the overall variability at all frequencies during the recording. Total variance of HRV increases with the length of analyzed recording, so comparison of SDNN of random ECGs with different durations is not possible, but comparisons of similar length can be done. Another time-domain parameter that is frequently used in HRV analysis is RMSSD, a square root of the mean of the sum of the squares of the differences between adjacent R-to-R peak intervals, which estimates vagally mediated short-term components of HRV (Task Force, 1996).

1.4.2 Frequency domain analysis

Frequency domain analysis breaks down the RRI signal into several frequency components and quantifies these components in terms of their relative intensity, termed "power". Thus, power spectral density (PSD) analysis provides basic information of how power (variance) distributes as a function of frequency. PSD analysis can be performed by parametric or nonparametric methods, and usually these methods yield comparable results.

Most of RRI frequency bands are detected at the respiratory frequency, which is considered to be a range from 0.15 Hz to 0.4 Hz in adults. This means, in practice, oscillations varying between 2.5-7.0 s. During exercise, the range of respiratory frequency could be extended up to 1.0 Hz. In addition to this high frequency (HF) oscillation, other RRI oscillations occur at low frequencies (LF), about 0.04 – 0.15 Hz (7-25 s), at very low (VLF, 0.003-0.04 Hz, 25 s- 5 min) and at ultra-low (ULF, <0.003 Hz) frequencies.

During stationary resting conditions, different maneuvers, such as controlled breathing, muscarinic blockade with atropine or cold face immersion, have shown that the high frequency component of HRV, as well as time-domain parameter RMSSD (in large part), are predominantly the result of fluctuations in vagal-cardiac nerve activity (Akselrod et al. 1981; Berntson et al. 1993; Cacioppo et al. 1994; Pomeranz et al. 1985; Task Force, 1996). Therefore, high frequency power (HFP) may provide an index of vagal activity, but the potential influence of respiration on RSA must be considered, because the rate, and also slightly the depth, of breathing has been found to affect RSA (Akselrod et al. 1981; Task Force, 1996). In addition to resting conditions, both time- (RMSSD) and frequency domain parameters (HFP) of vagal modulation have been proven to indicate the changes in vagal activity during immediate recovery after exercise (Ng et al. 2009).

The evidence for the origin of LF power (LFP) is unclear. LFP, with a center frequency of about 0.1 Hz, appears to reflect a baroreflex resonance frequency (Berntson et al. 1997; Pomeranz et al. 1985). It has been suggested to reflect sympathetic outflow, especially when expressed in normalized units (nu) (Malliani et al. 1994). However, controversy around this conclusion exists, and most

studies have suggested that LFP has both vagal and sympathetic origins (Akselrod et al. 1985; Berntson et al. 1997; Pomeranz et al. 1985), and their distributions vary in different experimental conditions, for example during supine and standing positions (Martinmäki et al. 2006a). Further, the LF/HF ratio has been suggested to offer a measure of sympathovagal balance (Malliani et al. 1994), but this theory has conflicting arguments (Berntson et al. 1997; Eckberg, 1997). A recent study of Rahman et al. (2011) concluded, and replicated previous findings (Moak et al. 2007), that LFP power does not reflect cardiac sympathetic tone but may reflect the ability of baroreflexes to modulate cardiac autonomic outflows. In addition, in the recent study of Ng et al. (2009), β-adrenergic blockade did not affect LFP during immediate recovery after exercise.

The mechanisms and origins of very low frequency rhythms are somewhat unclear, but are apparently affected by the renin-angiotensin regulation system, thermoregulation, circadian rhythms and parasympathetic outflow (Taylor et al. 1998). The mechanisms and origins of ultra-low rhythms remain unclear.

PSD analysis enhances the understanding of modulatory effects of neural mechanisms on the sinus node. The most commonly used frequency domain methods are nonparametric fast Fourier transform (FFT) and parametric autoregressive modeling. The power of frequency components are expressed either as absolute power (milliseconds squared, ms^2), as absolute values with natural logarithm (ln ms^2) or as normalized units (nu), in which the chosen frequency band (LFP or HFP) is divided by the total power from which very low frequency is removed, representing the relative power of the component. The recording of the signal should last at least 10 times the wavelength of the lower frequency bound of the analyzed component (Task Force, 1996).

One characteristic of frequency domain analysis is the requirement of the stationarity of analyzed signal. Only in this case, the power of the chosen spectral component represents true modulation of HRV at that frequency. This feature restricts the use of these analyses during transiently changing autonomic functions such as during immediate recovery after physical exercise when several mechanisms contribute to modulation of cardiac function.

1.4.3 Time-frequency analysis

Both real life and different laboratory tasks, for example an orthostatic task or exercise, produces rapid, transient changes in autonomic modulation. Individual responses to these tasks provide important information about the function of ANS. As traditional frequency domain analysis requires stationarity of the analyzed signal, many daily activities have been ignored from HRV analysis. Lately, different time-frequency methods have been used to analyze RRI signal.

In order localize the spectral information in time, a windowed part of data can be used to present the local spectral contents. Thereafter, this window is moved through the data in order to obtain time-frequency distribution (TFRD) of the data, where for each time instant, a local spectrum is attained. Due to the local nature of the spectrum, nonstationarities of the signal e.g. during changes

in autonomic modulation, do not have a major effect. TFRD can be presented as a three-dimensional graph (Figure 3), in which the x-axis is the time axis, the y-axis is the frequency axis, and the z-axis is the power axis (Oppenheim & Schafer, 1999; Pola et al. 1996; Saalasti, 2003).

One example of TFRD analysis is short-time Fourier transform (STFT), an extension of fast Fourier transform (FFT). STFT calculates consecutive power spectra of short sections of the signal and allows information of changes in the power spectrum as a function of time. This allows second-by-second monitoring of changes in either the power or the frequency of HRV. Another well-known time-frequency distribution analysis is Smoothed Pseudo Wigner transformation (SPWT), in which the summation of the window is used to smooth the estimate over time. The aim of this analysis is to reduce the cross-terms often appearing in the middle of two periodic components, making the interpretation difficult (Saalasti, 2003).

Because time and frequency resolutions of TFRD are inversely related, a high frequency resolution, for example, can be obtained with a short time-window length and vice versa. The chosen frequency components can limit the frequency resolution, as the duration of the time-window should be at least five times the lowest analyzed wavelength (Keselbrener & Akselrod, 1996). Digital filtering can be used to remove frequency components out of interest from the time series, especially useful in analysis of HR signal consisting of two separate frequency components, thus diminishing cross-terms and helping to improve the quality of the signal. The advantage of STFT over SPWT in HR signal analysis is that it is a more robust estimate of the local spectra, SPWT having, on the other hand, an excellent time resolution (Cohen, 1989).

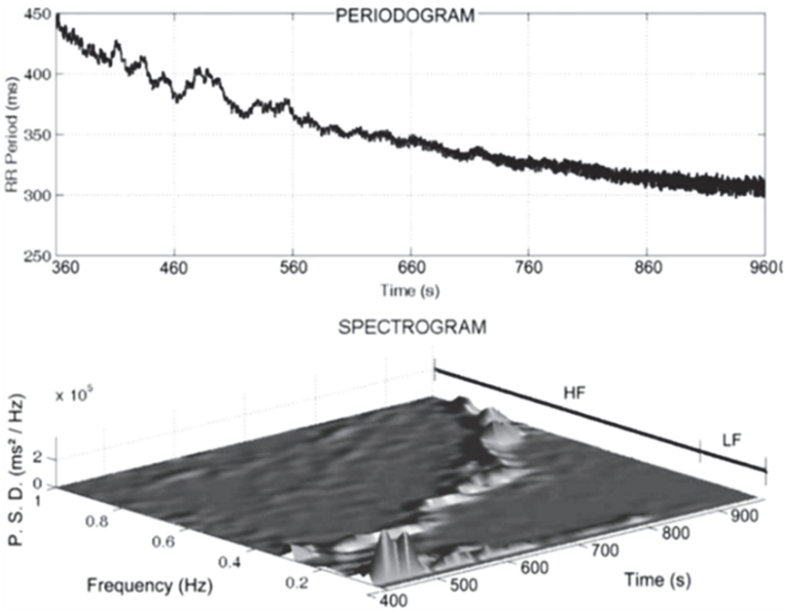

FIGURE 3 An example of RRI periods (top) and the computed spectrogram of one subject during an incremental exercise, using STFT analysis. X-axis = time (s), Y-axis = frequency (Hz), Z-axis = power spectral density (PSD, ms^2/Hz) (Cottin et al. 2006).

1.4.4 Short-term measurements of HRV

An advantage of short-term recordings of HRV is that they can quite easily be performed in very strictly standardized conditions. In short-term recordings, usually from 2 to 15 minutes, two main spectral components, low (LFP, 0.04 – 0.15 Hz) and high (HFP, 0.15 – 0.4 Hz) frequency components, are usually calculated to express variations in HR. The recording time should be determined by the lowest frequency component of interest. For example, at least 1 and 4 minute recordings, respectively, are needed when HFP and LFP components of HRV are investigated (Hartikainen et al. 1998; Task Force, 1996). If time domain methods are used to assess HRV from short-term recordings, at least five minute recordings are recommended to ensure the stability (Hartikainen et al. 1998). In long-term (e.g. 24 h) measurements, time-domain parameters are preferred to frequency-domain parameters, since the measurements no longer meet the demands of being stationary.

The reliability of short-term HRV measurements have been reported in the review of Sandercock et al. (2005b), who concluded that reliability of HRV measurements are heterogenous and depend on several factors. They found better reliability in the HRV measurements of a healthy population when com-

pared to different clinical populations. When the intraindividual relationship between the magnitude of HRV and the level of vagal outflow was studied, the relationship was found to be linear (Martinmäki et al. 2006b). In addition, the time- and frequency domain measures have been found to be highly correlated during recovery after exercise (Ng et al. 2009) and further, the STFT method has been found to measure transient changes in vagal activity during active orthostatic tests (Martinmäki et al. 2006a). The reliability of HRV and HRR measures between exercise sessions may be described as moderate; reliability depends partly on e.g. exercise mode and the chosen parameters and analysis methods (Al Haddad et al. 2011; Bosquet et al. 2008).

2 PURPOSE OF THE STUDY

In order to achieve optimal performance at the right time, training and recovery must be individually balanced. For now, there is no single practical, validated, physiological parameter to estimate physiological TL of single exercise sessions that can easily be used in everyday training. The main purpose of this thesis was to investigate immediate post-exercise dynamics of HRV after different endurance exercise sessions with short-time Fourier transform (STFT) method and find out if HRV recovery can be used as a tool to measure physiological TL of single exercise sessions. Methodological issues have restricted the use of HRV during transiently changing autonomic modulation, e.g. during recovery after exercise. Therefore, the dynamics of HRV immediately after exercise cessation has remained unclear. In addition, the other aim was to find out if the effects of different physical TL of endurance exercise sessions on physiological TL, i.e. disturbance to homeostasis can be detected with immediate post-exercise HRV. ANS functions as a control mechanism for body homeostasis, and therefore it may be speculated that modulations in cardiac autonomic functions after exercise may be an indicator of physiological TL. The specific aims of the study were:

1) To investigate the dynamics of HRV recovery immediately after different endurance exercise sessions using the STFT method. It was hypothesized that the recovery dynamics of HRV may be different to the recovery dynamics of HR, due to possible differences in the control of HRV and HR during recovery. HRV during rest is mainly vagally mediated, but it is not totally clear whether immediate HR recovery reflects mainly vagal tone (Buchheit et al. 2007b), vagal reactivation (Goldberger et al. 2006) or sympathetic withdrawal (Kannankeril et al. 2004). However, it seems that the relative role of sympathetic and vagal divisions during immediate recovery may depend on the physical TL of the exercise (Imai et al. 1994; Martinmäki & Rusko, 2008; Perini et al. 1989).

2) To investigate whether increased exercise intensity can be detected from post-exercise HRV. The main hypothesis was that especially after high-intensity exercises during which the energetic demands are increased, including significantly increased sympathetic activity, vagal activation is diminished (Casties et al. 2006; Goldberger et al. 2006). This could be detected on decreased post-exercise HRV.

3) To investigate whether increased exercise duration can be detected from post-exercise HRV. It was hypothesized that an increase in exercise duration at low intensity may not affect post-exercise HRV (Seiler et al. 2007), but when exercise intensity is at least moderate, a decrease in post-exercise HRV may be detected. EPOC, the "golden standard" in physiological TL estimation, has indicated that also duration of exercise is significant when the intensity is high enough (Gore & Withers, 1990).

4) To compare HRV and traditional TL parameters. It was hypothesized that post-exercise HRV and EPOC are inversely related, due to several findings of decreased post-exercise HRV after high-intensity exercises during which EPOC is significantly increased. In addition, it was hypothesized that HRV may give different results when compared to other traditional, non-physiological, parameters of TL (e.g. TRIMP, session RPE).

5) To compare immediate post-exercise HRV and long-term recovery of HRV after exercises with different physical TL. The main hypothesis was that long-term recovery of HRV may indicate differences between exercises with different physical TL similarly to immediate recovery period, since the recovery of autonomic processes after exhausting exercises have been found to last up to 48 hours (Mourot et al. 2004).

3 METHODS

3.1 Subjects

All the subjects were healthy, non-smoking volunteers (age 20-35 years (I), 20-40 years (II) and 20-45 years (III-IV)), and they were not taking any regular medication(s) that would affect cardiovascular functions. Volunteers with a body mass index (BMI, body mass/height (m^2)) over 30 were excluded from the study. Characteristics of the subjects are presented in the Table 1.

The aim of the study was to include endurance athletes (I) as well as non-trained (II) and recreationally trained subjects (III-IV). Endurance athletes were searched for and recruited from sports clubs. They had endurance training and racing background of at least 5-10 years, and their training hours during the last year were 350-500 h. Non-trained subjects were not completely inactive, but may have performed irregular endurance exercise. None of them had regular endurance training background. Recreationally trained subjects were selected from a larger group of subjects and recruited e.g. from marathon clubs. They had completed 4 (± 1) training sessions per week during the last two months, and all except one had run at least one marathon during the last few years.

Subjects were selected for the study if they were low-risk participants according to the ACSM (American College of Sports Medicine) guidelines (II-IV). The health status of the subjects was screened with a health questionnaire. Any respiratory, cardiovascular or other chronic systemic disease or other contraindication for exercise testing was an exclusion criterion for the present studies, as well as any anomaly in resting ECG (Cardiofax ECG-9320, Japan) (II-IV). In addition, any sudden death in near relatives was considered as an exclusion criterion (III-IV). Subjects gave a written informed consent prior to participation and had the right to withdraw from the study at any time. All exercise sessions were performed in a quiet laboratory, between 8.00 a.m. and 16.00 p.m. Subjects were asked to refrain from intense physical exertion for two days before each exercise day, to avoid alcohol the preceding day and to avoid coffee and other caffeinated drinks on the exercise day. Subjects were also advised to maintain a proper

fluid balance prior to exercise. For each subject, all exercise sessions were carried out approximately at the same time of day (within 4 h). The procedures (I-IV) were approved by the Ethics Committee of the University of Jyväskylä.

TABLE 1 Characteristics of the subjects.

	Study I	Study II	Study III-IV
N	8	13	13
Male	8	-	13
Female	-	13	-
Description	athletes	non-trained	trained
Age (yrs)	26 ± 4	35 ± 3	35 ± 5
Height (cm)	183 ± 3	168 ± 5	179 ± 6
Body mass (kg)	70 ± 6	63 ±10	77 ± 6
BMI (kg/m^2)	21 ± 2	23 ± 3	24 ± 2
Body fat (%)	12 ± 3	27 ± 4	14 ± 3
VO_{2max} (ml/kg/min)	60 ± 5	36 ± 3	54 ± 4
vVO_{2max} (km/h)	15.6 ± 0.9 *	10.9 ± 0.8	15.9 ± 0.8
HR_{max} (bpm)	194 ± 8	191 ± 10	183 ± 9

BMI = body mass index. * slope 5 %

3.2 Graded maximal treadmill test

Maximal aerobic capacity of each subject was assessed by a graded maximal treadmill (Telineyhtymä, Kotka, Finland) test. After becoming accustomed to the study protocol and equipment (II), subjects performed a maximal graded treadmill test with an initial speed of 5 km/h (II) or 8 km/h (I, III-IV) and the slope of 0.5 % (III-IV), 1 % (II) or 5 % (I). An increase in speed of 1 km/h was performed every three minutes until voluntary exhaustion. When the initial speed was 5 km/h, the first two loads of the test were performed by walking, otherwise loads were completed by running. Breath-by-breath respiratory data (oxygen uptake (VO_2), carbon dioxide production VCO_2) (I-II: Vmax 229, Sensor Medics, Palo Alto, USA, III-IV: Oxycon Mobile, Viasys Healthcare GmbH, Hoechberg, Germany) and R-R intervals (I: Polar R-R recorder, Polar Electro, Kempele, Finland, II-IV: Suunto t6 wristop computer, Suunto Oy, Vantaa, Finland) were measured constantly during the test. Fingertip blood samples for blood lactate analysis (Biosen S_line Lab+, EKF-diagnostic GmbH, Barleben, Germany) were taken after each 3 min session. The highest 60 s VO_2 value was considered as VO_{2max}. The respiratory compensation threshold (W_{RCT}) was determined according to Aunola & Rusko (1984).

3.3 Experimental exercise protocols

Subjects performed three (study III-IV), four (study I) or five (study II) controlled endurance exercise sessions on a treadmill (Telineyhtymä, Kotka, Finland). Exercises were performed in a random order once a week (I, II), or separated by at least two days (III-IV). Controlled exercise sessions were carried out on a treadmill at the individually determined running speeds based on the graded maximal treadmill test.

For athletes, the running velocities for the experimental exercises were determined with two different techniques to ensure the same relative intensity for all the subjects: (1) constant percentage of subject's vVO_{2max} (80, 85 and 93 %) and (2) based on work rate at respiratory compensation threshold (W_{RCT}) and W_{max} (maximal work rate) so that a = W_{RCT} + 1/3 (W_{max}–W_{RCT}), b = W_{RCT} +1/2 (W_{max}–W_{RCT}) and c = W_{RCT} + 3/4 (W_{max}–W_{RCT}). The previously described methods gave almost exactly the same intensities for experimental exercises. The controlled exercise modes of the athletes were following: interval exercise of 7 × 3 min with 2 min recovery periods at 85 % of vVO_{2max} (IV_{85}), interval exercise of 7 × 3 min with 2 min recovery periods at 93 % of vVO_{2max} (IV_{93}), continuous exercise of 21 min at 80 % of vVO_{2max} (CO_{80}), continuous exercise of 21 min at 85 % of vVO_{2max} (CO_{85}).

For non-trained subjects, the lowest exercise intensity was determined at 50 % of vVO_{2max}, the highest at W_{RCT}, and one in the middle of 50 % and W_{RCT}. It is known (Casadei et al. 1995; Nakamura et al. 1993; Perini et al. 1990; Tulppo et al. 1998) that when the intensity of exercise exceeds 50-60 % of VO_{2max}, vagal contribution to HR regulation diminishes, and finally disappears. Based on that, the lowest intensity chosen was 50% of vVO_{2max}. The controlled exercise sessions of the non-trained subjects were following: 3.5 km at 50 % of vVO_{2max} (3500_{LI}), 3.5 km at 63 % of vVO_{2max} (3500_{MI}), 3.5 km at 74 % of vVO_{2max} (3500_{HI}), 7.0 km at 50% of vVO_{2max} (7000_{LI}), 7.0 km at 63 % of vVO_{2max} (7000_{MI}).

In studies III and IV, the subjects were recreationally trained, thus the exercise intensities were determined higher when compared to II. Intensities were determined as percentages of vVO_{2max} of the subjects (60 %, 85 % and 105 % of vVO_{2max}). The controlled exercise sessions of the recreationally trained subjects were following: 3.0 km at 60 % of vVO_{2max} (MODE), 3.0 km at 85 % of vVO_{2max} (HI), 14.0 km at 60 % of vVO_{2max} (PRO), interval exercise of 6 × 2 × 250 m with 30 sec recovery periods at 85 % of vVO_{2max} (MO_{250}), interval exercise of 6 × 2 × 250 m with 30 sec recovery periods at 105 % of vVO_{2max} (HI_{250}), interval exercise of 3 × 2 × 500 m with 1 min recovery periods at 85 % of vVO_{2max} (MO_{500}). Experimental exercise protocols (studies I-IV) are presented in the Table 2.

Each exercise session was started with a standardized 5-7 minutes baseline resting measurements. After that, in study I, a 5 min (10 km/h, slope 3°) and in III-IV, a 1 km (60 % vVO_{2max}) warm-up was carried out. Immediately after the exercise cessation, a controlled passive recovery of 15 min (III-IV) or 30 min (I-II) was carried out. In study I, a standardized 5 min cool-down, similar to

warm-up, was performed before recovery. During the baseline and recovery phases, subjects were seated and were prohibited from moving and talking.

In study I, the exercise sessions were designed to represent typical high-intensity exercises of endurance athletes. Therefore all exercises were performed at high intensity and the differences in exercise intensities were quite small between exercise sessions. In further studies, exercise intensities were designed to be at least moderate, with more pronounced increases in exercise duration, in order to achieve changes in cardiac autonomic modulation. The focus in each study was to compare either the effects of a) increased exercise intensity, b) increased exercise duration/distance or c) the effects of exercise mode (interval vs. continuous) on the chosen "control" exercise.

The control exercise sessions of study I (IV_{85}, CO_{80}) were an interval session with seven bouts of 3 min running at 85 % of vVO_{2max} (2 min recovery period between bouts) and a continuous session of 21 min of running at 80 % of vVO_{2max}. The intensity of interval running was increased by 9.4 % (IV_{93}) and the intensity of continuous running with 6.3 % (CO_{85}). The effects of exercise mode were also investigated by comparing these control exercises of equal total work to each other.

In II, the control sessions were low-intensity continuous running of 3.5 km (3500_{LI}) and 7.0 km (7000_{LI}) at 50 % of vVO_{2max}. The exercise intensity of 3.5 km control session was increased with 26 % (3500_{MI}) and 48 % (3500_{HI}), and the 7.0 km control session with 26 % (7000_{MI}). The effect of increased exercise duration (100 %) was investigated by comparing these 3.5 km and 7.0 km control sessions to each other.

In III, the effects of a 41.7 % increase in exercise intensity (HI) and 367 % increase in exercise duration (PRO) of 3 km continuous exercise at 60 % of vVO_{2max} (MODE) were investigated. In IV, the effects of a 29.4 % increase in exercise intensity (HI_{250}) and 100 % increase in the interval duration (MO_{500}) of control interval running with six bouts of 250 m with 30 sec resting intervals (MO_{250}) were investigated. Sessions consisted of two sets of 6 × 250 m intervals, with a 5 min resting period between sets.

TABLE 2 Experimental exercise protocols.

Study	I	II	III	IV
Number of exercises	4	5	3	3
Exercise names and modes	IV_{85} = 7 × 3′ / r 2′ / 85 % vVO_{2max} * IV_{93} = 7 × 3′ / r 2′ / 93 % vVO_{2max} CO_{80} = 21′ / 80 % vVO_{2max}* CO_{85} = 21′ / 85 % vVO_{2max}	3500_{LI} = 3.5 km / 50 % vVO_{2max} * 3500_{MI} = 3.5 km / 63 % vVO_{2max} 3500_{HI} = 3.5 km /74 % vVO_{2max} 7000_{LI} = 7.0 km / 50 % vVO_{2max}* 7000_{MI} = 7.0 km / 63 % vVO_{2max}	MODE = 3 km / 60 % vVO_{2max} * HI = 3 km / 85 % vVO_{2max} PRO = 14 km / 60 % vVO_{2max}	MO_{250} = 2 × 6 × 250 m / r 30′′ / 85 % vVO_{2max} * HI_{250} = 2 × 6 × 250 m / r 30′′ / 105 % vVO_{2max} MO_{500} = 2 × 3 × 500 m / r 1′ / 85 % vVO_{2max}

IV_{85} = interval exercise of 7 × 3 min with 2 minute recovery periods at 85 % of vVO_{2max}, IV_{93} = interval exercise of 7 × 3 min with 2 minute recovery periods at 93 % of vVO_{2max}, CO_{80} = continuous exercise of 21 min at 80 % of vVO_{2max}, CO_{85} = continuous exercise of 21 min at 85 % of vVO_{2max}, 3500_{LI} = 3.5 km at 50 % of vVO_{2max}, 3500_{MI} = 3.5 km at 63 % of vVO_{2max}, 3500_{HI}, 3.5 km at 74 % of vVO_{2max}, 7000_{LI} = 7.0 km at 50% of vVO_{2max}, 7000_{MI} = 7.0 km at 63 % of vVO_{2max}, MODE = 3.0 km at 60 % of vVO_{2max}, HI = 3.0 km at 85 % of vVO_{2max}, PRO = 14.0 km at 60 % of vVO_{2max}, MO_{250} = interval exercise of 6 × 2 × 250 m with 30 sec recovery periods at 85 % of vVO_{2max}, HI_{250} = interval exercise of 6 × 2 × 250 m with 30 sec recovery periods at 105 % of vVO_{2max}, MO_{500} = interval exercise of 3 × 2 × 500 m with 1 min recovery periods at 85 % of vVO_{2max}.* Control exercise, compared with other exercises . ′ = minutes, ′′ = seconds, r = recovery, vVO_{2max} = velocity in maximal treadmill test during which the maximal oxygen uptake was reached.

3.4 Data collection

In each study, R to R peak intervals (RRI) were recorded (I: Polar R-R recorder, Polar Electro, Kempele, Finland, II-IV: Suunto t6 wristop computer, Suunto Oy, Vantaa, Finland) continuously during controlled exercise sessions. Exercise sessions included baseline measurements at rest, and measurements during exercise and controlled passive recovery. In athletes, RRI recordings were carried out constantly for approximately 36 h, from the evening before the exercise session to the morning following the session. RRI data were saved on computers (I: Polar R-R software, Polar Electro Oy; Kempele, Finland, II-IV: Suunto Training Manager, Suunto Oy, Vantaa, Finland).

Breath-by-breath expired VO_2 and VCO_2 (I-II: Vmax 229, Sensor Medics, Palo Alto, USA, III-IV: Oxycon Mobile, Viasys Healthcare GmbH, Hoechberg, Germany) were constantly collected during the exercise sessions, including rest and recovery, except for II, in which respiratory data was collected during rest, the last 3 kilometers of the exercise session, and recovery. Averages of 30 (IV) or 60 s (I-III) of respiratory data at the end of experimental exercises were used in statistical analysis. The highest 30 s (IV) or 60 s (I-III) value of VO_2 during the exercise was considered as VO_{2peak}.

Rating of perceived exertion (RPE, scale 0-10, Borg 1982) and fingertip blood samples for blood lactate analysis (Biosen S_line Lab+, EKF-diacnostic GmbH, Barleben, Germany) were obtained immediately after the exercise cessation (I-IV). Excess post-exercise oxygen consumption (EPOC) was calculated for the entire 15 min recovery (III-IV). Respiratory frequency (RF) was obtained from the breath-by-breath respiratory data during exercise and recovery (II). Also Training Impulse (exercise duration (min) × $(HR_{exercise}\text{-}HR_{rest})/(HR_{max}\text{-}HR_{rest})$ × 0.64 × $e^{[1.92 \times (HRexercise\text{-}HRrest)/(HRmax\text{-}HRrest)]}$, where e = Naperian logarithm having a value of 2.712) (III-IV) and session RPE (exercise duration × RPE) were calculated (III) (Banister, 1991). In addition, individual TRIMP ($TRIMP_i$) according to Manzi et al. (2009) was calculated (IV), where an exponential model is used to calculate individual curve for lactate and fractional HR (% of heart rate reserve).

3.5 HRV analysis

RRI data processing and HRV calculations were performed with MATLAB software (Matlab 7, MathWorks, Inc., Natick, USA). RRIs were checked and artefacts were deleted using a detecting algorithm and subsequently verified by visual inspection.

The original RRI series were then resampled at a rate of 5 Hz by using a linear interpolation to obtain equidistantly sampled time series. In order to remove frequencies below 0.04 Hz and above 1.0 Hz, a polynomial filter and a digital band-pass filter were used (Oppenheim & Schafer, 1999). Since the RRI

time series were not stationary during the recovery after exercises, the traditional spectral analysis could not be used for HRV analysis. Therefore, the short-time Fourier transform (STFT) method, an extension of FFT, was used for HRV time-frequency analysis (Elsenbruch et al. 1999; Keselbrener & Akselrod, 1996).

The STFT method provides time-frequency decompositions of the consecutive RRI time series by calculating consecutive power spectra of short sections of the signal. A section of 201 (I) or 512 samples were multiplied by the Hanning window function and the fast Fourier transform of their product was taken. The window was then shifted one sample forward and the same calculations were performed until the whole RRI time series, baseline, exercise and recovery, was covered. Low frequency power (LFP, 0.04 - 0.15 Hz), high frequency power (HFP, 0.15 - 1.0 Hz) and total power (TP, 0.04 - 1.0 Hz, =LFP+HFP) were calculated as integrals of the respective power spectral density curve. The higher frequency limit of 1.0 Hz was used to include the respiratory frequency during exercise and the immediate recovery to the analysis. The time and frequency resolutions of STFT are inversely related, and therefore the chosen resolutions are result of a compromise. It is recommended that the duration of the time window is five times of the slowest analyzed wavelength (Keselbrener & Akselrod, 1996). In order to obtain a high enough time resolution, a relatively short window was chosen for study I. In order to meet the assumptions of parametric statistical analysis, spectral powers were expressed in natural log transformed values (LFP_{ln}, HFP_{ln}, TP_{ln}) of which abbreviations of LFP, HFP and TP are further used. Very low (VLF) and ultra-low frequencies (ULF) are not of interest in the present study, as their physiological background is unclear.

HRV parameters were calculated as following: In the study I, pre-exercise resting HRV values are calculated as a mean of 2 min between 4.30-6.30 min of the 7 min sitting period. During the immediate 5 min recovery before cool-down, mean of 30 s, and further 60 s, were calculated. After cool-down, 60 s means during the 30 min recovery were calculated, of which further 3 min means (rec_{10} = 8-10 min, rec_{20} = 18-20 min, rec_{30} = 28-30 min) were calculated. Nocturnal HRV was analysed as a three hour mean at 01.00-04.00, providing that at least 30 min of sleep was successfully recorded before 01.00. In the study II, means of 3 min (pre-exercise sitting), 60 s (end of the exercise, immediate 1–5 min recovery) and 4 min (end of the recovery, 27–30 min) were calculated. In the studies III and IV, the first minute of the recovery measurement were excluded and the pre-exercise values were calculated as a 4 min mean between 1-5 min of the 5 min sitting period. Either 30 s (IV) or 60 s (III) means were calculated during the last minute of the exercises. Means of 60 s were calculated during 15 min recovery. In study III, also means for initial 120 s (HFP_{120}, LFP_{120}, TP_{120}) and 180 s (HFP_{180}, LFP_{180}, TP_{180}) of the recovery were calculated.

3.6 Statistical analysis

Statistical analyses were performed with SPSS for Windows software (SPSS, Inc., Chicago, Illinois, USA). The normality of data was tested with Kolmogorov-Smirnov test. The skewness of data was analyzed and histograms were visually inspected. To meet the normality assumptions of parametric statistical analysis, natural logarithm transformation (ln) was used for absolute HFP, LFP and TP values. A standard 1 was added to HRV values before calculating the natural logarithm, so that some of the values would not be negative ($y = \ln(1 + x)$). The distribution of some of the traditional training load parameters (BLa, EPOC, RPE, TRIMP) were skewed, and they were therefore partly analysed both with parametric tests (ANOVA, paired Student's t-test) and nonparametric Wilcoxon Signed Rank test.

In studies I-IV, analysis of variance (ANOVA) for repeated measures was used to compare the changes in HR, HFP, LFP and TP within the exercise sessions as a function of time, separately for each exercise. In addition, the main effects of exercise mode (increased intensity or duration), minute-by-minute dynamics (recovery time) during the immediate 5 min recovery and their interaction were analysed. In studies II-IV, the Bonferroni correction factor for multiple analyses was used in the comparison of three exercises. If the main effect of exercise mode between three exercises was significant, further pairwise analyses with repeated measures ANOVA between the chosen exercises were performed.

A paired, two-tailed Student's t-test was used as the post hoc test to compare the effect of 1) increased intensity of the control exercise session or 2) increased duration of the control exercise session on post-exercise HRV at chosen two exercise sessions, separately on each minute during the immediate (1-5 min) and long-term (10 min – 24 h) recovery as well as in the comparisons of nocturnal HRV between two chosen exercises. In addition, HRV at the end of recovery period was compared to pre-exercise resting levels also with a paired, two-tailed Student's t-test.

The Pearson's correlation coefficient was used to study the relationships between traditional TL parameters and post-exercise HRV during the immediate recovery (1-5 min) (III-IV). Values are expressed as mean ± standard deviation. In all tests, differences were considered significant, when $p < 0.05$. Statistical power calculations could not been made a priori due to lack of information of immediate HRV recovery after exercises, and a posteriori calculations are not recommended (Hoenig & Heisey, 2001) to explain the results.

4 RESULTS

4.1 Physiological training load of experimental exercises

The intensity of controlled exercise sessions of the present study varied from 50 % of vVO_{2max} (non-trained, II) to 105 % of vVO_{2max} (trained, IV) and running distance from 3.0 km (non-trained, II) to 14.0 km (trained, III). HR_{peak} during the exercises varied between 63 % and 98 % of HR_{max}, and BLa_{peak} between 0.8 – 10.6 mMol/l. Highest HR_{peak} (%), RPE and BLa_{peak} were found at athletes during interval exercise of 7 × 3 min at 93 % of $vVO_{2max.}$ Descriptive parameters of training load of the exercises are presented in Table 3.

4.2 HR and HRV at rest and during experimental exercises

There were no differences in resting HR between exercise sessions in athletes, non-trained or trained, except for higher HR ($P < 0.05$) after HI_{250} when compared to control exercise (MO_{250}) in trained subjects (IV). Pre-exercise resting values of HR and HRV of all subjects are presented in the Table 4.

HR and HRV during exercise. In athletes (I), increased intensity of the control exercise resulted in higher ($P < 0.05$) HR during exercise both in interval (IV_{93} compared to IV_{85}) and in continuous (CO_{85} when compared to CO_{80}) exercises. Also the type of exercise (continuous vs. interval) had an effect on HR, which was higher ($P < 0.05$) during CO_{85} as compared to IV_{85} (I) (Table 5).

During the last minute of the exercise, in each group of subjects, HFP, LFP and TP were significantly ($P < 0.001$) lower when compared to pre-exercise resting values. In athletes (I), HRV was rather low during all exercises and therefore increased intensity of the control exercise did not further decrease HFP, LFP or TP during the last minute of exercise, except for LFP ($P < 0.05$) during CO_{85} as compared to CO_{80}.

In non-trained subjects (II), increased exercise intensity from 50 % to 63 % and further to 74 % of vVO_{2max} increased HR ($P < 0.001$, Figure 5 a) and reduced HFP (Figure 5 b) and TP significantly ($P < 0.001$) as compared to the control exercise. Doubled exercise duration did not affect HRV during the exercise.

In recreationally trained subjects (III), increased intensity of continuous control exercise increased HR ($P < 0.001$, Figure 6 a). No effect of increased duration of exercise from 3.0 km to 14.0 km on HR was detected. In interval exercises, HR was higher ($P < 0.001$) both after increased intensity or duration of interval when compared to control exercise.

HRV during the last minute of continuous exercises of recreationally trained subjects were excluded from the statistical analysis because of the noisy RRI data of several subjects. During interval exercises, LFP was lower during HI_{250} and MO_{500} ($P < 0.01$, $P < 0.05$, respectively) and TP during HI_{250} ($P < 0.05$) as compared to MO_{250} (Figure 7).

4.3 HRV during recovery after experimental exercises

4.3.1 HR and HRV dynamics during immediate 5 minute recovery

In athletes (I), a five minute controlled cool-down was performed immediately after exercise and before recovery. A fast decrease ($P < 0.05$) of HR was detected during the first three recovery minute after each exercise, when compared to a previous 30 s (Figure 4 a). Despite the controlled cool-down at low intensity, barely any increase in HFP during the first five minutes of the recovery was found. In 30 s averages, HFP was higher ($P < 0.05$) only at the fifth minute of recovery when compared to the previous 30 s and also to the first 30 s of recovery (Figure 4 b). In TP, a fast increase ($P < 0.05$) was found during the first 90 s of recovery after each exercise when compared to the previous 30 s. In addition, an increase of TP ($P < 0.05$, Figure 4 c) was detected during the fifth minute of the recovery after IV_{85}.

In non-trained subjects (II), a decrease ($P < 0.05$) of HR was found during each minute of the five minute recovery after each exercise when compared to the previous minute, with the exception of 3500_{LI}, after which a HR decrease ($P < 0.05$) was detected mainly during the first two recovery minutes (Figure 5 a). The minute-by-minute increase of HFP in non-trained subjects was found during the first (3500_{LI} and 7000_{LI}, $P < 0.001$, Figure 5 b), fourth (3500_{MI}, $P < 0.05$, Figure 9 b) or fifth (7000_{MI}, $P < 0.05$, Figure 9 b) minute of the recovery when compared to the previous minute. After 3500_{HI}, no recovery of HFP was detected during the immediate five minutes (Figure 5 b). TP increased ($P < 0.05$) during the first minute after each exercise (Figure 5 c).

TABLE 3 Descriptive parameters of the experimental exercises (mean ± sd).

Study	Exercise	HR_{peak} (% of HR_{max})	VO_{2peak} (ml/kg/min)	BLa_{peak} (mMol/l)	RPE (0-10)	EPOC (ml/kg)	TRIMP	Velocity (km/h)
I	IV_{85}	93 ± 3	54 ± 3	4.2 ± 1.2	5 ± 2	-	-	13.3 ± 0.7
	IV_{93}	98 ± 2	57 ± 5	10.6 ± 1.9***	10 ± 1**	-	-	14.5 ± 0.8
	CO_{80}	91 ± 3	53 ± 4	5.1 ± 2.3	6 ± 3	-	-	12.5 ± 0.7
	CO_{85}	97 ± 3	54 ± 5	8.3 ± 0.5***	9 ± 1*	-	-	13.3 ± 0.7
II	3500_{LI}	63 ± 5	16 ± 2	0.8 ± 0.2	2 ± 2	24 ± 6	37 ± 9	5.5 ± 0.4
	3500_{MI}	87 ± 5***	27 ± 3***	2.8 ± 1.4**	6 ± 2***	37 ± 9	88 ± 21	6.9 ± 0.6
	3500_{HI}	92 ± 5***	31 ± 5***	4.3 ± 1.4***	7 ± 2***	40 ± 10	91 ± 17	8.0 ± 0.6
	7000_{LI}	64 ± 6	16 ± 3	1.3 ± 0.8*	3 ± 2*	28 ± 8	79 ± 23	5.5 ± 0.4
	7000_{MI}	88 ± 5***	27 ± 3***	2.8 ± 1.2***	6 ± 3***	37 ± 10	182 ± 39	6.9 ± 0.6
III	MODE	73 ± 4	36 ± 4	1.4 ± 0.6	3 ± 2	29 ± 7	26 ± 5	9.5 ± 0.5
	HI	92 ± 3***	52 ± 4***	7.6 ± 2.9***	6 ± 2 ***	77 ± 25 ***	35 ± 4 ***	13.5 ± 0.7
	PRO	76 ± 3	36 ± 3	2.6 ± 2.0*	4 ± 2**	34 ± 8	117 ± 21***	9.5 ± 0.5
IV	MO_{250}	82 ± 3	44 ± 2	2.7 ± 1.0	4 ± 2	47 ± 7	20 ± 3	13.5 ± 0.7
	HI_{250}	92 ± 4***	51 ± 5***	9.1 ± 3.5 ***	7 ± 3***	80 ± 16***	22 ± 4	16.7 ± 0.9
	MO_{500}	88 ± 4***	49 ± 3***	3.6 ± 1.6**	5 ± 2	49 ± 8	19 ± 3	13.5 ± 0.7

IV_{85} = interval exercise of 7 × 3 min at 85 % of vVO_{2max}, IV_{93} = interval exercise of 7 × 3 min at 93 % of vVO_{2max}, CO_{80} = continuous exercise of 21 min at 80 % of vVO_{2max}, CO_{85} = continuous exercise of 21 min at 85 % of vVO_{2max}, 3500_{LI} = 3.5 km at 50 % of vVO_{2max}, 3500_{MI} = 3.5 km at 63 % of vVO_{2max}, 3500_{HI}, 3.5 km at 74 % of vVO_{2max}, 7000_{LI} = 7.0 km at 50% of vVO_{2max}, 7000_{MI} = 7.0 km at 63 % of vVO_{2max}, MODE = 3.0 km at 60 % of vVO_{2max}, HI = 3.0 km at 85 % of vVO_{2max}, PRO = 14.0 km at 60 % of vVO_{2max}, MO_{250} = interval exercise of 6 × 2 × 250 m at 85 % of vVO_{2max}, HI_{250} = interval exercise of 6 × 2 × 250 m at 105 % of vVO_{2max}, MO_{500} = interval exercise of 3 × 2 × 500 m at 85 % of vVO_{2max}. Different when compared to control exercise (in I: between IV_{85} and IV_{93} or between CO_{80} and CO_{85}) of each study at * $P < 0.05$, ** $P < 0.01$, *** $P < 0.001$.

TABLE 4 Resting HR, LFP, HFP and TP of the subjects during pre-exercise sitting.

Study	Exercise	HR (bpm)	LFP (ln ms²)	HFP (ln ms²)	TP (ln ms²)
I	IV_{85}	63 ± 8	7.3 ± 1.1	7.5 ± 1.4	8.2 ± 1.1
	IV_{93}	62 ± 8	7.2 ± 1.5	7.3 ± 0.8	8.2 ± 0.8
	CO^{80}	62 ± 10	7.4 ± 0.5	7.5 ± 0.9	8.3 ± 0.6
	CO_{85}	65 ± 11	7.3 ± 0.6	7.0 ± 0.8	8.0 ± 0.6
II	3500_{LI}	74 ± 8	6.6 ± 0.7	6.7 ± 1.0	7.5 ± 0.7
	3500_{MI}	75 ± 8	6.9 ± 0.6	6.7 ± 0.6	7.6 ± 0.6
	3500_{HI}	75 ± 10	6.8 ± 0.8	6.7 ± 1.4	7.6 ± 1.0
	7000_{LI}	71 ± 10	6.5 ± 0.9	7.0 ± 0.7	7.5 ± 0.9
	7000_{MI}	74 ± 8	6.8 ± 0.6	6.6 ± 1.1	7.5 ± 0.8
III	MODE	59 ± 12	8.2 ± 1.5	7.8 ± 1.0	8.9 ± 1.2
	HI	57 ± 9	8.2 ± 1.4	7.9 ± 0.9	9.0 ± 1.0
	PRO	56 ± 7	8.2 ± 1.3	7.8 ± 0.9	9.0 ± 1.1
IV	MO_{250}	56 ± 6	8.1 ± 1.4	8.1 ± 1.2	9.0 ± 1.2
	HI_{250}	63 ± 11*	8.0 ± 1.2	7.2 ± 1.2*	8.6 ± 1.1
	MO_{500}	58 ± 8	8.1 ± 1.4	8.0 ± 1.2	9.0 ± 1.2

The explanations of abbreviations of the exercises are presented in table 3. HR = heart rate, LFP = low frequency power, HFP = high frequency power, TP = total power, ln = natural logarithm. *Different when compared to MO_{250} at $P < 0.05$.

In recreationally trained subjects (III, IV), the successive minute-by-minute HR decreased during the 5 min recovery after both continuous and interval sessions ($P < 0.05$), with the exception of the non-significant difference between the third and fourth recovery minute after MODE and between the fourth and fifth minute after HI and PRO (Figure 6 a, 7 a).

After continuous exercises, successive minute-by-minute HFP increased during the first two recovery minutes after MODE ($P < 0.05$), HI ($P < 0.001$) and PRO ($P < 0.05$) (Figure 6 c). Successive minute-by-minute LFP (Figure 6 b) and TP (Figure 6 d) increased during the first two recovery minutes after MODE ($P < 0.01$) and HI ($P < 0.001$), and also during the third minute after PRO ($P < 0.05$). After interval exercises, LFP ($P < 0.001$, Figure 7 b), HFP ($P < 0.01$, Figure 7 c) and TP ($P < 0.01$, Figure 7 d) increased during the first two recovery minutes after each session after which no differences between the successive minutes were detected.

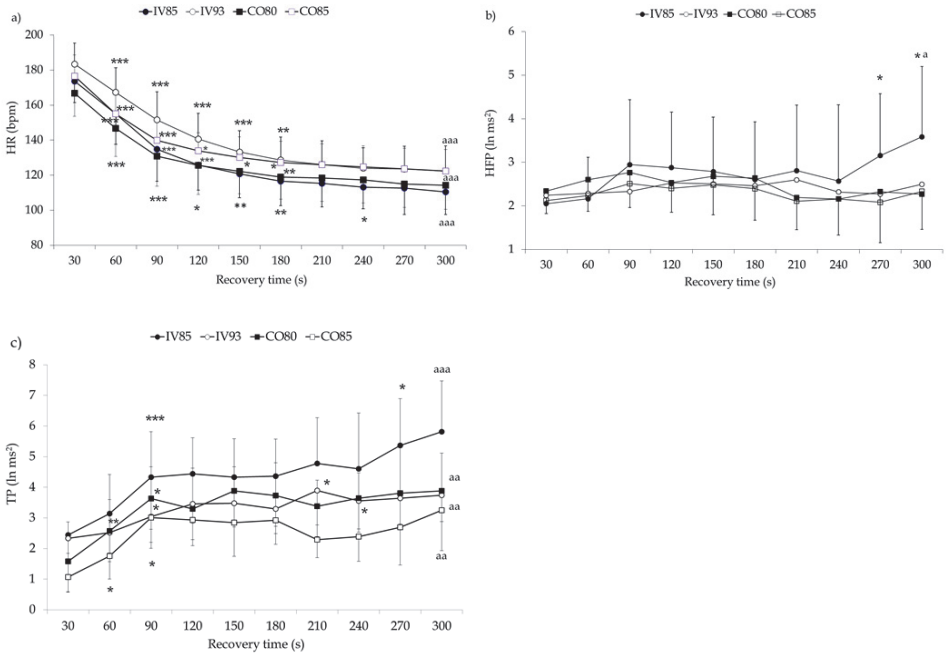

FIGURE 4 Dynamics of HR (a), HFP (b) and TP (c) during a five min recovery after high-intensity exercises of athletes (I). A five-minute controlled cool-down was performed immediately after exercise, before recovery. IV_{85} = interval exercise of 7 × 3 min at 85 % of vVO_{2max}, IV_{93} = interval exercise of 7 × 3 min at 93 % of vVO_{2max}, CO_{80} = continuous exercise of 21 min at 80 % of vVO_{2max}, CO_{85} = continuous exercise of 21 min at 85 % of vVO_{2max}. Different from the previous value at * $P < 0.05$, ** $P < 0.01$, *** $P < 0.001$, different from the first value at 30 s at ^{a} $P < 0.05$, ^{aa} $P < 0.01$, ^{aaa} $P < 0.001$.

4.3.2 The effects of increased exercise intensity on immediate 5 minute post-exercise HRV

In athletes (I), the main effects of recovery time ($P < 0.001$) and the interaction of recovery time x exercise intensity ($P < 0.05$) for HR were significant in interval exercises. In continuous exercises, the main effects of recovery time ($P < 0.001$) and the main effects of exercise intensity ($P < 0.05$) for HR were significant, but not their interaction. The main effects of recovery time ($P < 0.01$) and the interaction of recovery time x exercise intensity for HFP were significant in interval exercises but not in continuous exercises. The main effects of recovery time and exercise intensity, as well as their interaction for LFP ($P < 0.001$, $P < 0.05$, $P < 0.05$, respectively) and TP ($P < 0.001$, $P = 0.064$, $P < 0.01$) were also significant in interval exercises. In continuous exercises, the main effect of recovery time was significant for both LFP and TP ($P < 0.001$). LFP was lower ($P < 0.05$) during the first five minutes after IV_{93} and when minute-by-minute comparisons to the control exercise at lower intensity were made. In addition, TP was lower after IV_{93} at the fifth minute of the recovery, when compared to IV_{85} (Table 5).

In the non-trained subjects, the main effect of exercise intensity ($P < 0.001$) and the main effect of recovery time ($P < 0.001$) for HR, HFP and TP were significant between the three exercises at different intensities. In addition, the interaction (exercise intensity x recovery time) was significant in HFP ($P < 0.05$). Increased exercise intensity from 50 % to 63 % and further to 74 % of vVO_{2max} resulted in higher HR ($P < 0.01$, Figure 5 a) and lower HFP ($P < 0.05$, Figure 5 b) and TP ($P < 0.05$, Figure 5 c) during each minute of the immediate 5 min recovery.

Similar findings were found also in the recreationally trained subjects when all three continuous exercises (III) were compared. The main effects of exercise (increased intensity or duration), recovery time and their interaction for HR, HFP, and TP were all significant ($P < 0.05$) between these three exercises. In LFP, the main effect of exercise ($P < 0.001$) and recovery time ($P < 0.001$) were significant between these three exercises, but not their interaction. After increased exercise intensity of continuous exercises from 60 % to 85 % of vVO_{2max}, HR was higher ($P < 0.001$, Figure 6 a) and LFP ($P < 0.01$, Figure 6 b) HFP ($P < 0.05$, Figure 6 c) and TP ($P < 0.01$, Figure 6 d) lower during each minute of the immediate 5 min recovery.

In interval exercises (IV), the main effects of exercise ($P < 0.001$) and recovery time ($P < 0.001$), as well as their interaction ($P < 0.05$) were significant for HR, LFP, HFP and TP. Increased intensity from 85 % to 105 % of vVO_{2max} resulted in higher HR ($P < 0.001$, Figure 7 a) and lower LFP ($P < 0.001$, Figure 7 b), HFP ($P < 0.01$, Figure 7 c) and TP ($P < 0.001$, Figure 7 d) during each minute of the immediate 5 min recovery.

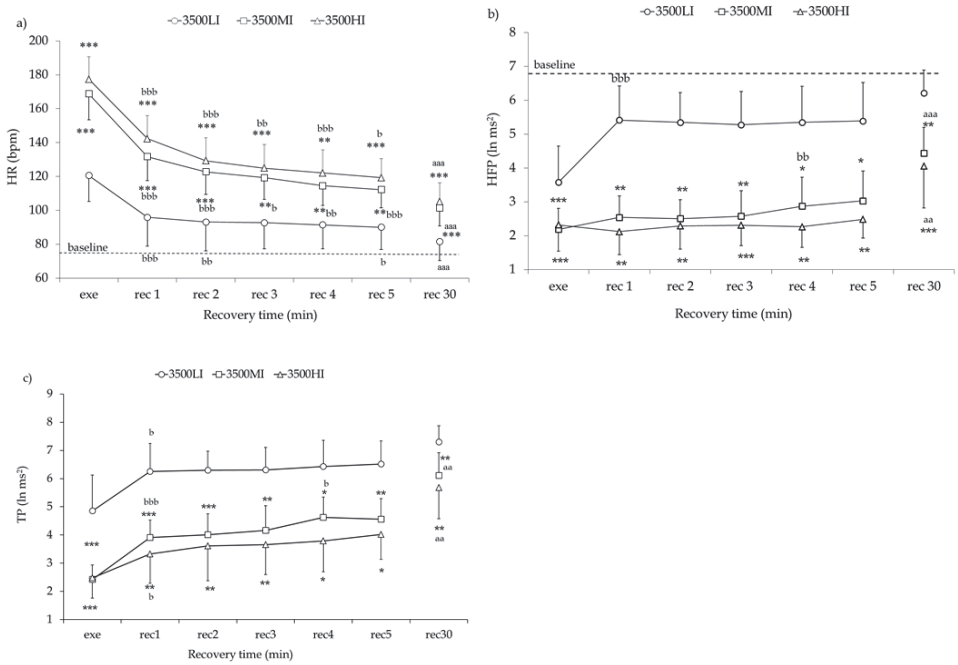

FIGURE 5 The effects of increased exercise intensity on immediate HR (a), HFP (b) and TP (c) recovery of non-trained subjects (II). 3500_{LI} = 3500 m at 50% vVO_{2max}, 3500_{MI} = 3500 m at 63% vVO_{2max}, 3500_{HI} = 3500 m at 74% vVO_{2max}, 7000_{LI} = 7000 m at 50 % vVO_{2max}, 7000_{MI} = 7000 m at 63% vVO_{2max}. Different when compared to 3500_{LI} at *P < 0.05, **P < 0.01, ***P < 0.001, different when compared to the resting value at ^{aa} P < 0.01, ^{aaa} P < 0.001, different when compared to a previous minute at ^{b} P < 0.05, ^{bb} P < 0.01, ^{bbb} P < 0.001.

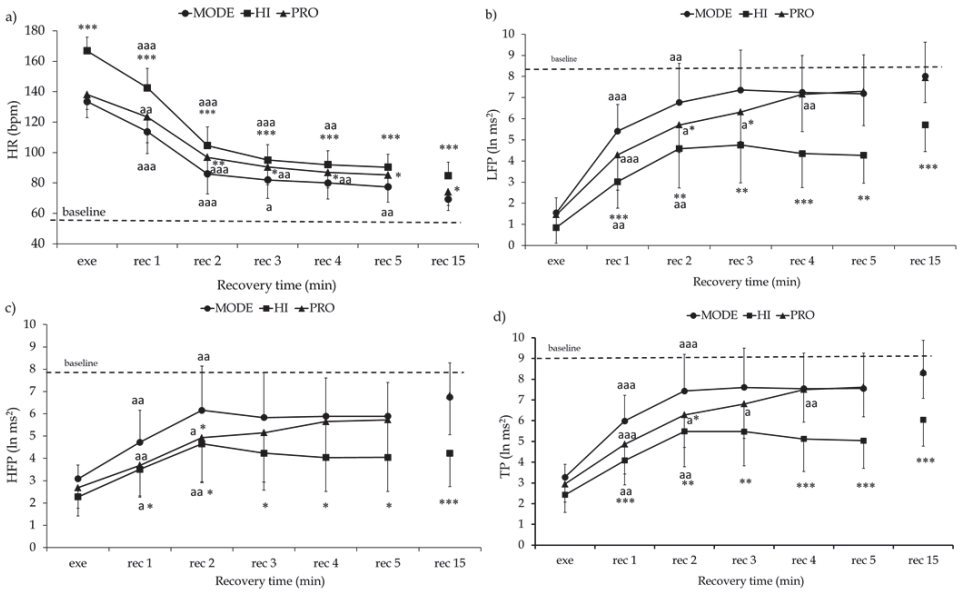

FIGURE 6 The effects of increased exercise intensity or duration on post-exercise HR (a), LFP (b), HFP (c) and TP (d) of recreationally trained subjects (III). MODE (moderate intensity) = 3 km at 60% vVO_{2max}, HI (high intensity) = 3 km at 85% vVO_{2max}, PRO (prolonged) = 14 km at 60% vVO_{2max}. Different when compared to MODE at *P < 0.05, **P < 0.01, ***P < 0.001, different when compared to a previous minute at ^{a} P < 0.05, ^{aa} P < 0.01, ^{aaa} P < 0.001.

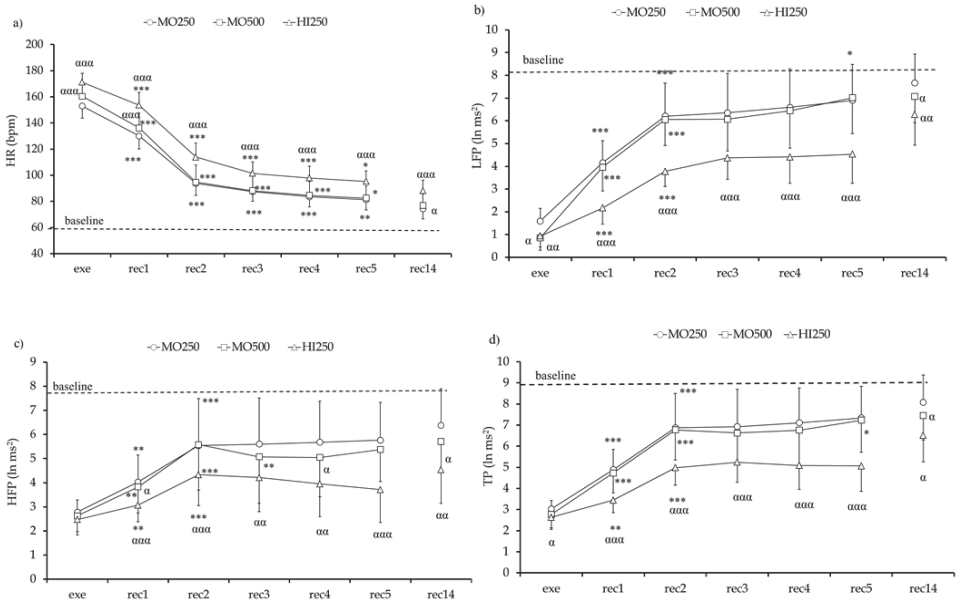

FIGURE 7 The effects of increased interval intensity (HI_{250}) or duration (MO_{500}) on immediate HR (a), LFP (b), HFP (c) and TP (d) recovery of recreationally trained subjects (IV). MO_{250} = moderate intensity exercise of 2 × 6 × 250 m at 85% vVO_{2max}, MO_{500} = moderate-intensity exercise of 2 × 3 × 500 m at 85% vVO_{2max}, HI_{250} high-intensity exercise of 2 × 6 × 250 m at 105% vVO_{2max}. Different when compared to MO_{250} at $^{\alpha}$ $P < 0.05$, $^{\alpha\alpha}$ $P < 0.01$, $^{\alpha\alpha\alpha}$ $P < 0.001$. Different when compared to a previous minute at * $P < 0.05$, ** $P < 0.01$, *** $P < 0.001$.

HFP of the first recovery minute varied from 36 ± 9 to 83 ± 20 % of the pre-exercise baseline values, when the results of athletes, non-trained and trained subjects were included. Increased exercise intensity to high-intensity control exercise did not change the difference in HFP between pre-exercise rest and the first recovery minute (I), but when control exercise was of low or moderate intensity (II and III), resulted the increased exercise intensity in greater difference in HFP during the first recovery minute when compared to pre-exercise rest. The difference between resting HFP and 1st minute recovery HFP (%) of all groups are presented in Figure 8.

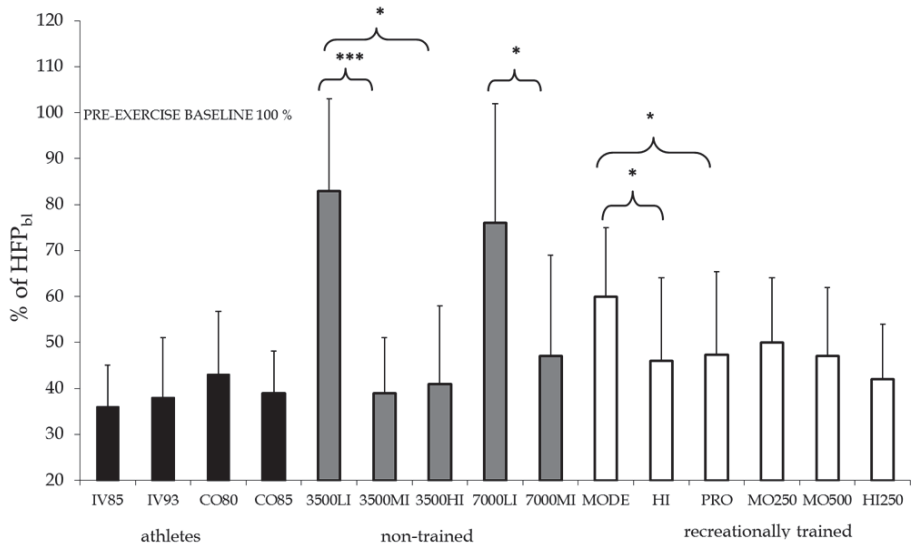

FIGURE 8 The effects of increased exercise intensity or duration on HFP at the first minute of the recovery (% of baseline (HFP_{bl}) after controlled exercises of athletes (I), non-trained (II) and recreationally trained (III-IV). The abbreviations of the exercises are explained in the table 3. Different when compared to the control exercise of each group (IV_{85}, 3500_{LI} or 7000_{LI}, MODE, MO_{250}) at * $P < 0.05$, *** $P < 0.001$.

4.3.3 The effects of increased exercise duration on immediate 5 minute post-exercise HRV

In the non-trained (II), doubled exercise duration from 3.5 km to 7.0 km at 50 % (Figure 9 a, 9 b) and 63 % (Figure 9 c, 9 d) of vVO_{2max} did not affect immediate 5 min post-exercise HR or HRV recovery, either in HFP, LFP or TP. The main effect of exercise duration on HR, HFP, TP was not significant between3500_{LI} and 7000_{LI} or between 3500_{MI} and 7000_{MI}.

In the recreationally trained subjects (III, IV), the main effects of exercise, recovery time and their interaction on HR, HFP, and TP were all significant ($P < 0.05$) between three exercises. When the duration of 3.0 km control exercise was increased to 14.0 km at 60 of % vVO_{2max}, only a few differences were noticed in minute-by minute HRV during the first five minutes (Figure 6). When two- (Figure 10 a) or three- (Figure 10 b) minute averages were used, the differences between exercises became visible, as HFP, LFP and TP were lower ($P < 0.05$) after prolonged exercise. Similarly, HFP at the first minute of the recovery (% of baseline) after prolonged exercise was lower when compared to control exercise (Figure 8). Thus, in the present study, the duration of exercise became significant when the distance of the exercise was over fourfold when compared to the control exercise (III).

In interval exercises (IV), HFP was lower at the first and fourth minute of the recovery after the interval was doubled in duration (MO_{500}) when compared to MO_{250} (Figure 7 c), but no other differences were found.

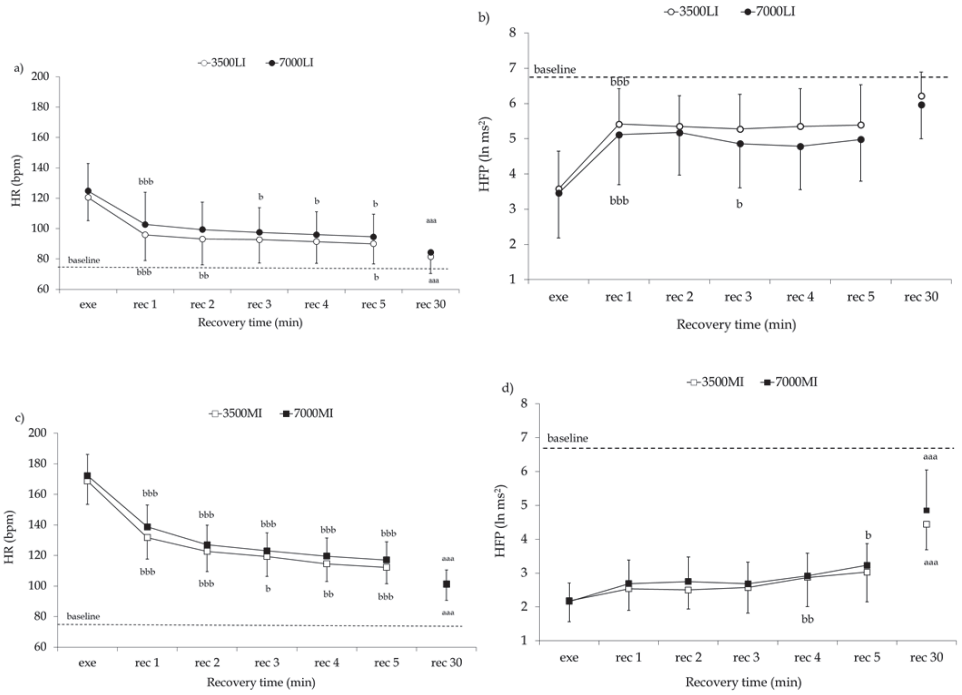

FIGURE 9 The effects of increased exercise duration at the intensity of 50 % of vVO_{2max} (a, b) and at the intensity of 63 % of vVO_{2max} (c, d) on immediate HR (a, c) and HFP (b, d) recovery of non-trained subjects (II). Significantly different from the resting value at ^{aa} $P < 0.01$, ^{aaa} $P < 0.001$, Significantly different when compared to a previous minute at ^{b} $P < 0.05$, ^{bb} $P < 0.01$, ^{bbb} $P < 0.001$. The explanations of abbreviations of exercises are presented in the Figure 5.

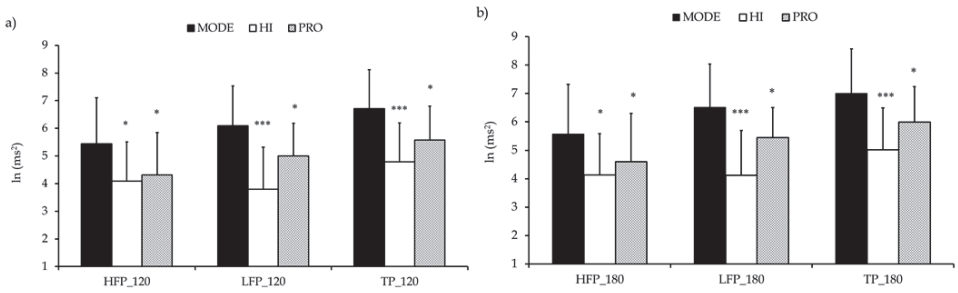

FIGURE 10 Differences in HFP, LFP and TP between the continuous exercises of recreationally trained subjects (III) during the immediate recovery as two- (a) and three (b) minute means. MODE (moderate intensity) = 3 km at 60% vVO_{2max}, HI (high intensity) = 3 km at 85% vVO_{2max}, PRO (prolonged) = 14 km at 60% vVO_{2max}. Significantly different from MODE at *P < 0.05, **P < 0.01, ***P < 0.001.

4.3.4 The effects of exercise intensity and duration on short-term 15-30 min recovery of HRV

Controlled passive recovery was carried out for 15 min (III-IV) or 30 min (I-II). In the athletes, HR remained higher and HRV lower (P < 0.05) at the end of 30 min recovery when compared to the pre-exercise resting values after each controlled exercise session, with the exception of LFP and TP after IV_{85}, which were recovered to pre-exercise level. When compared to the end of exercise, no increases were observed in HFP during the 30 min recovery after $CO_{85.}$ When compared to a control exercise, either interval or continuous, increased exercise intensity delayed the recovery of HFP and TP (Table 5).

In the non-trained subjects (II), HR was higher (P < 0.001) than pre-exercise resting level after each exercise at the end of 30 min recovery. HFP (Figure 9 b) and TP had increased to the resting level during the 30 min recovery only after 3500_{LI} and 7000_{LI}. Similarly to the immediate recovery, HFP, LFP and TP were still lower (P < 0.01) after 3500_{MI} and 3500_{HI} when compared to $3500_{LI.}$

In the recreationally trained subjects (III), HR was still higher (*P* < 0.001) while HFP and TP were lower (*P* < 0.05) at the end of the 15 min recovery after each continuous exercise when compared with the pre-exercise baseline. LFP was lower than baseline after HI (P < 0.05), but did not differ from the baseline after MODE and PRO. The differences between HI and MODE were still visible at the end of the recovery in HR, HFP, LFP and TP (P < 0.001). After interval exercises (IV), all HRV parameters, except for LFP after MO_{250}, were lower (P < 0.05) at the end of the 15 min of recovery when compared to pre-exercise baseline. Both increased intensity (HI_{250}) and increased duration of interval (MO_{500}) resulted in lower HFP, LFP and TP at the end of the 15 min recovery.

TABLE 5 Differences in HR, HFP and TP between experimental exercises during rest, exercise and recovery in the athletes (I) (mean ±sd).

	IV_{85}	IV_{93}	CO_{80}	CO_{85}
HR (bpm)				
$rest_{sitting}$	63 ± 8	62 ± 8	62 ± 10	65 ± 11
exe_{end}	177 ± 10	187 ± 12*	174 ± 14	185 ± 12^{*a}
rec_5	112 ± 14	123 ± 14	115 ± 13	123 ± 12^{a}
rec_{10}	89 ± 12	102 ± 13*	90 ± 13	100 ± 14^{*a}
rec_{20}	84 ± 11	95 ± 11**	83 ± 12	94 ± 12$^{* a}$
rec_{30}	82 ±11	93 ± 12**	81 ± 10	92 ± 12^{**a}
HFP (ln ms^2)				
$rest_{sitting}$	7.4 ± 1.4	7.3 ± 0.8	7.4 ± 0.9	7.0 ± 0.8
exe_{end}	2.1 ± 0.5	2.3 ± 0.7	2.2 ± 0.6	2.0 ± 0.6
rec_5	3.4 ± 1.5	2.4 ± 0.8	2.3 ± 1.0	2.2 ± 0.7
rec_{10}	5.1 ± 1.3	3.0 ± 0.9**	3.8 ± 1.2	2.4 ± 1.1^{**aa}
rec_{20}	5.4 ± 1.1	3.5 ± 0.9***	4.9 ± 0.9	3.2 ± 1.2^{**a}
rec_{30}	5.5 ±1.0	3.7 ± 0.8***	5.1 ± 1.1	3.2 ± 1.1^{**aaa}
TP (ln ms^2)				
$rest_{sitting}$	8.1 ± 1.1	8.1 ± 0.8	8.1 ± 0.6	7.8 ± 0.6
exe_{end}	2.3 ± 0.5	2.3 ± 0.6	2.3 ± 0.7	2.1 ± 0.5
rec_5	5.6 ± 1.6	3.7 ± 0.9*	3.8 ± 1.4	3.0 ± 1.3^{a}
rec_{10}	6.9 ± 1.3	5.1 ± 0.8**	5.5 ± 1.2	4.2 ± 1.3$^{* aa}$
rec_{20}	7.3 ± 1.2	5.7 ± 0.8***	6.6 ± 0.8	5.5 ± 1.1
rec_{30}	7.2 ± 0.9	6.0 ± 0.7***	6.7 ± 0.7	5.5 ± 1.1^{**aa}

The explanations of abbreviations of exercises are presented in table 3. HR = heart rate, HFP = high frequency power, TP = total power, ln = natural logarithm, exe_{end} = last minute of the exercise, rec_5 = fifth minute after exercise cessation (1 min mean), rec_{10} = recovery during 8-10 minutes after cool-down cessation, rec_{20} = recovery during 18-20 minutes after cool-down cessation, rec_{30} = recovery during 28-30 minutes after cool-down cessation. Statistically significant differences between IV_{93} vs. IV_{85} and between CO_{80} vs. CO_{85} at * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$. Statistically significant differences between IV_{85} and CO_{85}, at ^{a} $P < 0.05$, ^{aa} $P < 0.01$, ^{aaa} $P < 0.001$.

4.3.5 The effects of exercise intensity and duration on long-term recovery of HRV

During the night following (night 2) experimental exercises of athletes, HR and HRV was similar to the preceding night (night 1) after each exercise. The results of nocturnal HRV are presented in the Table 6.

TABLE 6 HR and HRV of athletes during the night preceding (night 1) and following (night 2) the controlled exercise interventions.

Night 1	IV_{85}	IV_{93}	CO_{80}	CO_{85}
RR (ms)	1274 ± 100	1358 ± 143	1323 ± 130	1331 ± 125
HR (bpm)	48 ± 4	45 ± 5	46 ± 5	46 ± 4
RMSSD (ln ms^2)	8.4 ± 0.9	8.7 ± 1.1	8.4 ± 0.7	8.4 ± 1.3
HF (ln ms^2)	7.4 ± 1.3	7.9 ± 1.4	7.4 ± 1.1	7.4 ± 1.8
TP (ln ms^2)	8.2 ± 1.2	8.5 ± 1.3	8.2 ± 1.0	8.1 ± 1.5
Night 2				
RR (ms)	1283 ± 147	1257 ± 108	1309 ± 120	1314 ± 58
HR (bpm)	48 ± 5	48 ± 4	47 ± 4	46 ± 2
RMSSD (ln ms^2)	8.4 ± 1.0	8.2 ± 1.4	8.1 ± 1.0	8.9 ± 0.7
HF (ln ms^2)	7.4 ± 1.4	7.4 ± 1.9	7.0 ± 1.5	8.2 ± 0.9
TP (ln ms^2)	8.2 ± 1.3	8.2 ± 1.6	7.8 ± 1.3	8.9 ± 0.7

The explanations of abbreviations of exercises are presented in table 3. HR = heart rate, RMSSD = square root of the mean of the sum of the squares of the differences between adjacent R-to-R peak intervals, LFP = low frequency power, HFP = high frequency power, TP = total power, ln = natural logarithm.

When comparing HR or HRV of the morning after the experimental exercises to the pre-exercise resting levels, there were no differences in interval exercises. After continuous exercises, differences were still visible. HFP (Figure 11) and TP were significantly ($p < 0.05$) lower the morning after CO_{80} than before exercise. In addition, HR was significantly ($p < 0.01$) higher and HFP and TP ($p < 0.01$) lower the next morning after CO_{85} than before exercise. The results of HR and HRV after 24 h recovery are presented in the Table 7.

TABLE 7 HR and HRV of athletes at pre-exercise baseline and following morning after controlled experimental exercises (mean ± SD).

Pre-exercise	IV_{85}	IV_{93}	CO_{80}	CO_{85}
RR (ms)	966 ± 116	988 ± 136	989 ± 142	939 ± 146
HR (bpm)	63 ± 8	62 ± 8	62 ± 10	65 ± 11
HFP (ln ms^2)	7.4 ± 1.4	7.3 ± 0.8	7.4 ± 0.9	7.0 ± 0.8
TP (ln ms^2)	8.1 ± 1.1	8.1 ± 0.8	8.1 ± 0.6	7.8 ± 0.6
24 h recovery				
RR (ms)	995 ± 170	968 ± 205	958 ± 165	880 ± 112 **
HR (bpm)	62 ± 10	65 ± 13	65 ± 13	69 ± 10 **
HFP (ln ms^2)	7.3 ± 0.9	7.0 ± 1.2	6.9 ± 0.7 *	6.1 ± 0.8 **
TP (ln ms^2)	8.0 ± 1.1	8.0 ± 1.0	7.5 ± 0.7 *	7.2 ± 0.7 **

The explanations of abbreviations of exercises are presented in table 3. HR = heart rate, HFP = high frequency power, TP = total power, ln = natural logarithm. Significantly different from the pre-exercise baseline at * $P < 0.05$, ** $P < 0.01$.

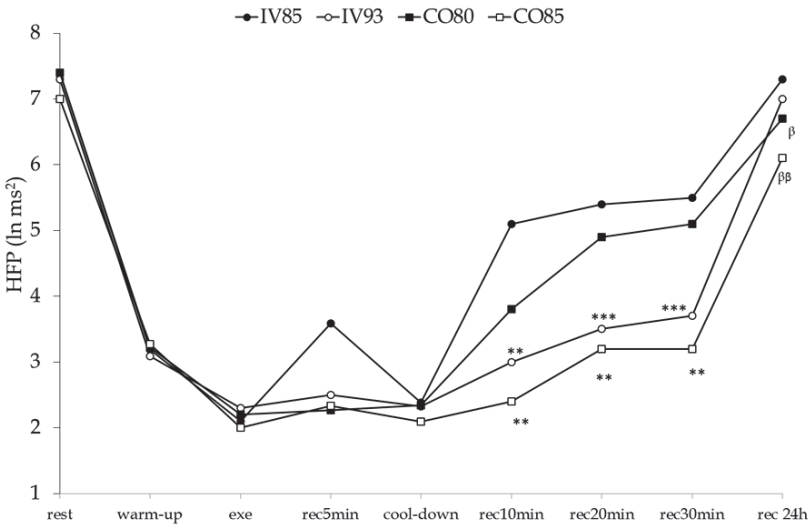

FIGURE 11 HFP of athletes before, during, and after high-intensity exercises. IV_{85} = interval exercise for 7 × 3 min at 85% vVO_{2max} with 2-min recovery intervals, IV_{93} = interval exercise for 7 × 3 min at 93% vVO_{2max} with 2-min recovery intervals, CO_{80} = continuous exercise for 21 min at 80% vVO_{2max}, CO_{85} = continuous exercise for 21 min at 85% vVO_{2max}. vVO_{2max} = velocity at VO_{2max}; VO_{2max} = maximal oxygen uptake. Difference between IV_{85} and IV_{93} or between CO_{80} and CO_{85} ** $P < 0.01$, *** $P < 0.001$. Different from pre-exercise baseline $^{\beta}$ $P < 0.05$, $^{\beta\beta}$ $P < 0.01$.

4.4 Relationship between traditional training load indicators and post-exercise HRV

In the recreationally trained subjects, post-exercise HRV and traditional TL parameters correlated negatively both in continuous (III) and in interval (IV) exercise sessions. When all three continuous exercise sessions were included, RPE ($P < 0.001$) and BLa ($P < 0.05$) were negatively associated with post-exercise HRV. To be more specific, associations between RPE and HFP in each continuous exercise session (MODE, HI, PRO) are presented separately in Figure 12.

Similarly, when all three interval exercise sessions were included, RPE ($P < 0.001$) and BLa ($P < 0.05$) were negatively associated with post-exercise HRV. In addition, in interval exercise sessions there was also a negative correlation also between EPOC and HRV ($P < 0.05$). Findings were parallel when 60 s, 120 s or 180 s averages of HRV were used. The associations between traditional TL parameters and HRV are presented in Table 8 a and 8 b.

TABLE 8a Correlations between traditional TL parameters and post-exercise HRV in continuous exercise sessions of recreationally trained subjects (III).

	RPE (0-10)	BLa (mmol/l)	EPOC (ml /kg)	TRIMP	RPEs
HFP_{120}	-.604***	-.401*	ns.	ns.	-.414*
LFP_{120}	-.634***	-.601***	-.399*	ns.	ns.
TP_{120}	-.691***	-.569**	ns.	ns.	ns.
HFP_{180}	-.586***	-.409*	ns.	ns.	-.367*
LFP_{180}	-.638***	-.622***	-.406*	ns.	ns.
TP_{180}	-.674***	-.580**	ns.	ns.	ns.

BLa = blood lactate at the end of the exercise, RPEs (session RPE) = exercise duration × RPE, TRIMP = Training Impulse, EPOC = excess post-exercise oxygen consumption during the 15-minute recovery. HFP_{60} = mean HFP during the first recovery minute, HFP_{120} = mean HFP during the first two recovery minutes, HFP_{180} = mean HFP during the first three recovery minutes, TP_{60} = mean TP during the first recovery minute, TP_{120} = mean TP during the first two recovery minutes, TP_{180} = mean TP during the first three recovery minutes. * P < 0.05, ** P < 0.01, *** P < 0.001. * P < 0.05, ** P < 0.01, *** P < 0.001. All three exercises (MODE, HI, PRO) are included.

TABLE 8b Correlations between traditional TL parameters and post-exercise HRV in interval exercises of recreationally trained subjects (IV).

	RPE (0-10)	BLa (mmol/l)	EPOC (ml /kg)	TRIMP	$TRIMP_i$
HFP_{60}	-.778***	-.447**	-.447**	-.539**	-.368*
HFP_{120}	-.727***	-.397*	-.419*	-.503**	-.302*
HFP_{180}	-.700***	-.366*	-.389*	-.484**	-.338*
TP_{60}	-.731***	-.653***	-.633***	-.371*	-.329*
TP_{120}	-.749***	-.627***	-.613***	-.417**	-.338*
TP_{180}	-.738***	-.570***	-.544***	-.417**	-.326*

BLa = blood lactate at the end of the exercise, EPOC = excess post-exercise oxygen consumption during the 15-minute recovery, TRIMP = Training Impulse, $TRIMP_i$ = individualized TRIMP. HFP_{60} = mean HFP during the first recovery minute, HFP_{120} = mean HFP during the first two recovery minutes, HFP_{180} = mean HFP during the first three recovery minutes, TP_{60} = mean TP during the first recovery minute, TP_{120} = mean TP during the first two recovery minutes, TP_{180} = mean TP during the first three recovery minutes. * P < 0.05, ** P < 0.01, *** P < 0.001.

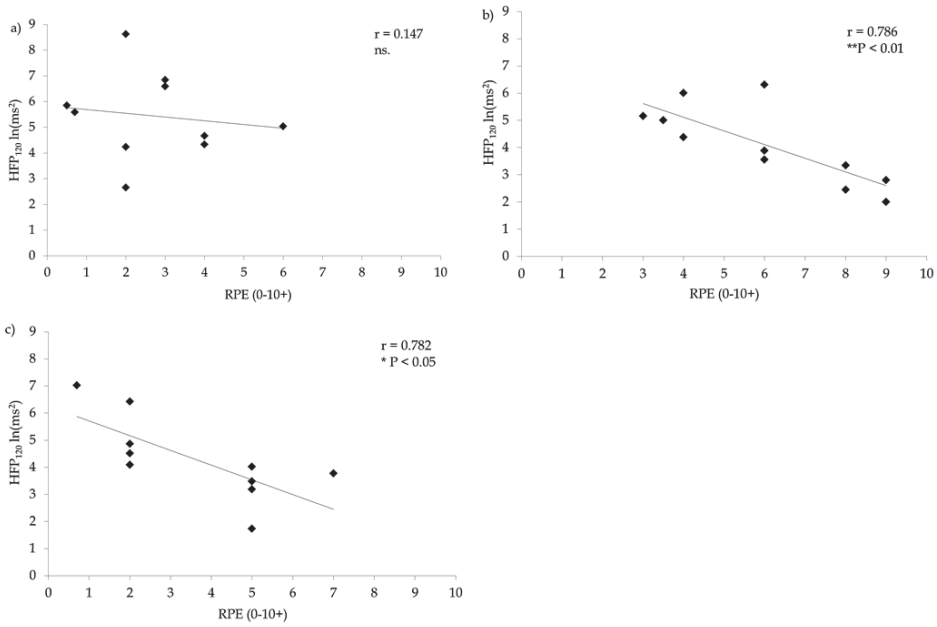

FIGURE 12 Correlation between immediate HFP [ln(ms^2)], 2 minute mean) and RPE in MODE (a), HI (b) and PRO (c) of recreationally trained subjects (III). Explanations of abbreviations of exercises are presented in figure 9. HFP = high frequency power [ln(ms^2)].

5 DISCUSSION

The main aim of this thesis was to investigate the recovery dynamics of HRV immediately after different endurance exercise sessions in subjects with different training backgrounds, using a time-frequency analysis method. Methodological issues have restricted the studies of HRV during transiently changing autonomic modulation, for example during immediate recovery after exercise and therefore the immediate recovery dynamics of HRV have remained unclear. The results of the present study indicated that recovery of HRV may be visible already during the first recovery minute after endurance exercise sessions, when compared to the end of the exercise, and the recovery pattern of HRV is different from HR. Minute-by-minute recovery dynamics of HRV seemed to be slightly different depending on the exercise session and training background of the subjects. Another aim of the study was to compare endurance exercises with different physical TL, that is, different intensity and/or duration, and find out if changes in physical TL could be detected with post-exercise HRV indices. The novel finding was that the differences between exercises could be detected in HRV already during the first recovery minutes. The main factor determining post-exercise HRV seemed to be exercise intensity, but increases in exercise duration could be detected in post-exercise HRV as well, if the exercise intensity was at least moderate.

5.1 The dynamics of HRV immediately after exercise

A great deal of knowledge exists about the recovery dynamics of HR after different experimental conditions. Delayed recovery of HR after physical exercise has been found as a risk factor for different cardiac complications (Cole et al. 1999; Nissinen et al. 2003) and is also a predictor of the training status of athletes (Daanen et al. 2012). Increased vagal activity has been suggested to be responsible for the fast decrease of HR during the first minute after exercise cessation (Imai et al. 1994; Perini et al. 1989). Whether there is any clinical relevance

for the recovery of HRV remains unclear. In many studies, immediate HRV recovery during the first minutes after exercise cessation has been excluded from the analysis due to methodological issues, as commonly used traditional frequency domain analyses require a stationary signal. Therefore, the recovery dynamics of HRV during the immediate recovery phase has remained unclear. During the past few years, a new approach to the analysis of HRV, a time-frequency method, has been used during different, frequently changing conditions and physiological tasks, e.g. orthostatic test, exercise and recovery. It has been found to detect transient changes in vagal activity (Keselbrener & Akselrod, 1996; Martinmäki et al. 2006a), which has given an opportunity to investigate the pattern of rapid HRV changes immediately after exercise cessation.

During exercise, HRV is significantly decreased, as sympathetic nervous activity is augmented and vagal activity attenuated. Immediately after exercise cessation changes occur in baroreceptor function, central command and cardiac pre-load and contractility, which affect cardiac functions together with increased vagal activity (Miles et al. 1984; Oida et al. 1997; O'Leary, 1993; Plotnick et al. 1986). In the present study, the recovery of HR and HRV were followed continuously with subjects in a sitting position, for either 15 or 30 minutes, but the main focus was on the immediate 5 min recovery. A significant increase of HRV, when compared to the end of exercise, was detected between the first and the fifth minute of recovery in most of the exercise sessions of the present study. The increase of HRV towards resting levels continued thereafter from minutes to hours. There were some differences in the recovery dynamics of HRV after exercises with different physical TL, i.e. intensities and durations of the exercises.

Findings of the recovery dynamics of HRV in the present study were similar to the few studies that had investigated the immediate recovery of HRV after endurance exercise sessions. The lowest exercise intensity in the present study was 50 % of the VO_{2max} of untrained women, based on knowledge of the approximate intensity at which vagal activity diminishes (Casadei et al. 1995; Perini et al. 1990; Tulppo et al. 1998). After 3.5 km or 7.0 km of exercise at such intensity, a fast increase of HFP and TP almost to the pre-exercise levels was detected already during the first recovery minute after exercise cessation. Similar results were found by Martinmäki & Rusko (2008), who found fast recovery of HRV during the first recovery minute after low-intensity (30% VO_{2max}) exercise using the same time-frequency HRV method as in the present study.

The recovery dynamics of HRV were slightly slower after moderate intensity exercises (60 - 63 %) when compared to low-intensity exercise. A minute-by-minute increase of HFP after a moderate-intensity exercise session of trained and untrained subjects was observed during the second and fourth recovery minutes, respectively. Martinmäki & Rusko (2008) also found a quite similar pattern of HFP recovery after moderate (61 % of maximal power) endurance exercise. One explanation for the slower vagal reactivation after exercise at a higher intensity may be the higher proportion of sympathetic modulation and accumulating anaerobic metabolites during higher-intensity exercise.

The highest exercise intensities in continuous exercises were 85 % of vVO_{2max} in athletes (~ 5.5 km) and recreationally trained men (3.0 km). After the continuous exercises of the athletes, HFP remained suppressed for the entire immediate 5 min recovery. In contrast to athletes, HFP increased significantly during the first two minutes after high-intensity exercise of recreationally trained subjects. A similar pattern of post-exercise HRV recovery after high-intensity exercise has been introduced by Goldberger et al. (2006), although using different HRV parameters and analysis methods. In the study of Goldberger et al. (2006), RMS and MSSD, indices of vagal activity, increased significantly during the first 90 seconds after exercise cessation, after which recovery was slower. To explain differences between the results of athletes and recreationally trained subjects of the present study, it may be speculated that a longer duration of exercise session and maybe the shorter training background of the young athletes may affect the results, i.e. the physiological TL of the exercises in athletes were higher than in recreationally trained subjects.

When high-intensity continuous and interval exercises of recreationally trained subjects are compared, the recovery dynamics of HRV seemed to be similar. Regardless of the remarkable increase of intensity to 105 % of vVO_{2max}, no difference in the minute-by-minute recovery dynamics were noticed, even if the level of HRV was lower after exercise at the highest intensity. After the high-intensity interval exercise (at 85 % of vVO_{2max}) of the athletes, remained HFP suppressed for four minutes, after which it started to increase slowly. A slight difference in HFP dynamics between the interval and continuous exercises of the athletes could be detected during the fifth minute of the immediate recovery period, since no recovery of HFP was visible after the continuous exercises.

Differences in the dynamics of immediate HRV recovery between exercise sessions at different intensities may originate from activation and inhibition of sympathetic and vagal systems. In the low-intensity exercises, vagally mediated fast recovery of HRV was detected during the first minute after which pre-exercise levels were almost achieved. In low-intensity exercises, the role of sympathetic activation was presumably small, in contrast to moderate and high-intensity exercises, during which sympathetic activity is more pronounced (Carter et al. 2003).

In the present study, some differences in the recovery dynamics of HRV between different frequencies (LFP, HFP) were detected, although one previous study suggested that both HFP and LFP are of vagal origin during recovery after exercise (Ng et al. 2009). For example, in athletes (I), HFP recovery after high-intensity exercise was not observed during the immediate 5 min recovery despite increased LFP and TP were detected. One possible explanation for the LFP increase after the exercises may be due to alterations in baroreflex sensitivity. Baroreflex sensitivity is known to be inhibited during high-intensity exercise and to recover after the cessation of exercise (Berntson et al. 1997; Casadei et al. 1995). The similarity of LFP and TP recovery patterns during immediate recovery also suggests that during recovery from high-intensity exercise, in contrast

to rest, TP may consist mostly of low frequency variation and therefore, indicates overall changes in autonomic modulation rather than changes in vagal modulation. Although it is assumed that low frequency variation of HRV consists of both vagal and sympathetic proportions, the actual contribution of sympathetic modulation may be only speculated, since no measurements of sympathetic nervous activity were made.

As found also in previous studies (Buchheit et al. 2007b; Kannankeril et al. 2004), the recovery dynamics of HR and HRV after different exercises were not similar in the present study. Differences may be explained by the possible variation in the physiological control of these parameters; HRV is believed to describe mainly the reactivation of vagal modulation, whereas the recovery of HR is probably affected by both vagal reactivation and a decrease in sympathetic activation. According to the results of the present study, it seems that after low-intensity exercises, the fast recovery of HR as well as HRV occurs during the first minute of the recovery. After the first minute, recovery is slower and the resting level may be reached already during the first 15 minutes. It may be speculated that after low-intensity exercises, the fast recovery of both HR and HRV during the first minute of the recovery may be originated from the reactivation of vagal modulation, since no major sympathetic contribution is presumably present during these exercises, but instead the increased heart rate and decreased HRV during the exercise may be a result of decreased vagal activity. In the present study, the dynamics of HR and HRV after high-intensity exercises were different, since a significant decrease of HR was noticed during the first five minutes of the high-intensity exercises whereas barely any recovery of HRV could be detected. It is possible that after high-intensity exercises, during which a substantial proportion of sympathetic activity contribute to the increase of HR, the immediate recovery of HR is mainly due to declined sympathetic activity, rather than increased vagal activity. In contrast to HR, the recovery of HRV, especially of HFP, is due to increased vagal activation, which seemed to be delayed after high-intensity exercises of the present study.

5.2 The effects of exercise intensity on post-exercise HRV

In previous studies, HRV *during* exercise has been found to differentiate exercises that were performed below and above (the second) ventilatory threshold (Cottin et al. 2004). Vagal outflow is known to decrease at the onset of exercise, causing an increase in heart rate and a decrease in HRV. During moderate- or high-intensity exercise, sympathetic activity is vigorously increased and sympathetic metabolites, norepinephrine and epinephrine, increase in the blood. This is accompanied by further decreases in vagal activity that may be detected as markedly decreased HFP.

In addition, increased exercise intensity has been found to have an effect on HRV *after* exercise, both during early (Casties et al. 2006; Goldberger et al. 2006; Martinmäki & Rusko, 2008) and later recovery (Buchheit et al. 2007a;

Gladwell et al. 2010; Parekh & Lee, 2005; Saboul et al. 2015; Seiler et al. 2007; Terziotti et al. 2001).

According to the results of the present study, exercise intensity seems to be the main factor in determining immediate HRV recovery after exercise. After exercises with increased intensity when compared to a control exercise, HRV was decreased during the immediate 5-minute recovery. The lowest exercise intensity, 50 % of vVO_{2max}, of the present study could be determined as low intensity, without accentuated sympathetic activity, which has been proposed to have an impact on HRV recovery. When this low-intensity exercise of untrained women was used as a control exercise session, a substantial decrease in post-exercise HRV was detected when exercise intensity was increased to 63 % and further 74 % of vVO_{2max}.

Similar results were found by Gladwell et al. (2010) and Martinmäki & Rusko (2008), who studied the effects of 20 min moderate, hard, and vigorous (60 %, 76 % and 87 % of W_{max}, respectively) and 10 min of low (29 %) and high (61%) intensity exercise in sedentary subjects. Increased exercise intensity resulted in decreased HRV during the first 15 recovery minutes in both studies. The time needed for HRV to recover to the pre-exercise level seemed to differ between studies with different experimental exercises. Similar to the non-trained subjects of the present study, decreased HRV was found in trained men when HRV after the control exercise at 60 % of vVO_{2max} was compared to a higher exercise intensity of 85 % of vVO_{2max}. It should be remembered that the non-trained subjects of the present study were women, in contrary to other two groups of subjects of the present study. It has been suggested that a lower sympathetic and higher vagal control of the heart may exist in women (Carter et al. 2003), which may also affect the results of the present study, although the knowledge of the effects of gender on HRV dynamics after exercise is lacking. Although we did not compare the results of untrained and trained subjects, it seems that the recovery of HRV between groups is quite similar. It is presumable that the slightly slower recovery of HRV of non-trained women is due to training background rather than the gender.

In contrast to the results of non-trained and recreationally trained subjects, increased exercise intensity from 80 % to 85 % of vVO_{2max} in the athletes did not affect immediate 5 min recovery of HRV. The control exercise of 80 % of vVO_{2max} was strenuous enough to diminish HRV almost completely and, therefore, a further decrease after exercise at 85 % of vVO_{2max} could not be detected. In addition, the increase in intensity was smaller when compared to non-trained and recreationally trained subjects. Although immediate recovery did not indicate differences between these high-intensity exercises, increased intensity resulted in significantly lower HRV during the short-term 10-30 minute recovery. It is possible that the differences between two high-intensity exercises become visible only during later recovery, when the recovery processes of autonomic functions begin to recover.

In the present study, both the athletes and recreationally trained subjects participated in interval exercises at 85 % (both groups) and 93 % (athletes) or

105 % (trained) of vVO_{2max}. Similarly to continuous exercises, a significant effect of increased exercise intensity on post-exercise HRV was also found after interval exercises. In both groups, increased exercise intensity resulted in lower HRV during both immediate and short-term recovery when compared to a lower-intensity control exercise. To our knowledge, no previous studies on immediate HRV recovery after different interval exercises exist. As the results of the present study suggest, the reactivation of cardiac vagal modulation after interval exercises probably occurs quite similarly to continuous exercise sessions. Although the dynamics of HRV were quite similar after interval and continuous exercises, the level of HRV after continuous exercises were lower, even if the total distance and intensity of the interval and continuous exercises were identical. Thus, it seems that the resting periods between intervals decrease the physiological training load of the exercises which can be detected as a higher HRV during immediate recovery when compared to a continuous exercise of identical workload.

Contrary to our finding of decreased HRV after continuous exercise when compared to interval exercise of similar intensity and total work, Mourot et al. (2004) found a more pronounced decrease in HRV after interval exercise when compared to continuous exercise of equal total work (kJ/kg). A possible explanation for the differences in the results may be the differences in exercise protocols, as Mourot et al. (2004) did not include actual resting periods between the intervals, only periods of exercise at two different intensities. In addition, interval exercise in their study was performed at a higher intensity than the continuous exercise.

The effects of increased exercise intensity on HRV during the immediate 5 min recovery and the short-term 15-30 min recovery were mainly similar in the present study. However, in the high-intensity exercises in which HRV remained blunted during the immediate 5 min recovery, some of the differences between exercises became visible only during the short-term recovery. It seemed that the reactivation of vagal modulation may not occur during the first few minutes after the exercise if the exercise has been carried out nearly to exhaustion, which was the case at least in the highest-intensity exercises of the athletes. In order to find out differences between high-intensity exercises with different physical TL, a recording of HRV longer than 5 min may be needed.

The effects of increased exercise intensity on long-term recovery of HRV were studied in the athletes of the present study. It has been found in previous studies (Hautala et al. 2001; Hynynen et al. 2010; Mourot et al. 2004) that the recovery of vagal modulation may be delayed even 24-48 h after a prolonged high-intensity exercise. In the present study, no differences were found in the nocturnal HR or HRV between the nights before and after the experimental exercises. However, decreased vagal modulation after the continuous exercises could be detected in the following morning, when resting measurements were carried out. These results are similar to the previous study of Hynynen et al. (2006), who found impaired cardiac modulation after awakening in overtrained subjects, despite similar nocturnal HRV when compared to a control group. It

may be speculated that similar alterations in the vagal modulation of healthy subjects may also be possible after a strenuous exercise, after which some, but not total, recovery of vagal modulation has occurred.

5.3 The effects of exercise duration on post-exercise HRV

In the present study, the effects of increased exercise duration were investigated with different exercise intensities and durations both in trained and untrained subjects. While it was hypothesized that increased exercise intensity delays post-exercise HRV recovery, the effects of increased exercise duration were unclear. The well-known phenomenon of augmented sympathetic activation and accumulation of metabolites during and after high-intensity exercise sessions known to affect recovery of autonomic function may not occur during prolonged exercise sessions at low or moderate intensity. Still, prolonged exercise duration may cause increased physiological TL and impair recovery of autonomic functions.

In the present study, exercise durations were either doubled or increased over fourfold at low and moderate exercise intensities. Doubled exercise duration from 3.5 km to 7.0 km did not delay immediate HRV recovery in non-trained subjects when exercise intensity was either 50 % or 63 % of vVO_{2max} (II). In the recreationally trained subjects (III), the 14-km exercise averaging duration of 90 minutes delayed HRV recovery when compared to the 3-km exercise, which averaged duration of 19 minutes. Minute-by-minute comparison of HRV during immediate 5 min recovery did not reveal differences between exercise sessions. However, when the immediate recovery of high frequency component of HRV was either compared to pre-exercise baseline or calculated for longer two- and three-minute periods to increase the stability of the results, the delayed recovery of the prolonged exercise was observable. Despite the statistical significance, the differences between these exercises were minor and the impact of this kind of differences between exercises on an actual training period remains unclear.

The effects of increased exercise duration on immediate post-exercise HRV recovery have been, to our knowledge, previously investigated only by Seiler et al. (2007). They studied the effects of increased intensity or duration of low-intensity (mean 61 % of VO_{2max}) exercise in highly trained athletes. The results of the present study are quite similar to their findings, as they found no effect of doubled exercise duration from 60 min to 120 min, when exercises were performed below the first ventilatory threshold. The intensity of 60 % of vVO_{2max} in recreationally trained subjects in the present study was comparable to 61% of VO_{2max} in their study. It may be speculated that the slightly delayed recovery in HRV after the prolonged exercise of the present study may be due to greater increase in exercise duration as well as different training backgrounds of the subjects. Although the relative intensities were similar (% VO_{2max}), physiological TL of exercise may have been greater for subjects with lower aerobic capaci-

ty. Seiler et al. (2007) speculated that exercise should probably be performed above the first ventilatory/lactate threshold to observe an autonomic stress reaction, which could be detected on post-exercise HRV.

The effects of increased interval duration of interval exercises were also investigated in the present study in recreationally trained subjects (IV). The duration of interval was doubled -from 250 m to 500 m at the exercise intensity of 85 % of vVO_{2max} (2 × 6 × 250 m /recovery 30 s vs. 2 × 3 × 500 m /recovery 60 s), keeping the total distance of the exercises equal. When the minute-by minute recovery of HFP during the immediate 5 min period was investigated, only a modest decrease after prolonged intervals at the first and fourth minute was detected, and no differences in LFP or TP were noticed. At the end of the 15 min recovery, the increased duration of interval could be detected, similarly to increased intensity, as lower HRV when compared to a control exercise. So, it may be possible to observe a decrease, at least minor, in post-exercise HRV after interval exercise with similar total work but with longer interval duration.

Although post-exercise HRV after interval exercises with different interval durations have not been investigated, changes in physiological strain, i.e. physiological TL of exercises have been detected by Vuorimaa et al. (2000). The exercises in the study of Vuorimaa et al. (2000) were performed at the maximal exercise intensity (100% of VO_{2max}) and, therefore, the duration of exercise had greater effect on physiological TL compared to lower intensity exercises (85% of V_{max}) as in the recreationally trained subjects of the present study.

In previous studies, it has been shown that the effects of a high-intensity prolonged exercise on post-exercise HRV may be observed until the night following exercise or even longer (Hautala et al. 2001; Hynynen et al. 2010; Mourot et al. 2004), either when compared to resting day (Hautala et al. 2001) or some kind of control exercise (Hynynen et al. 2010; Myllymäki et al. 2012). In the study of Hynynen et al. (2010), the intensity of the baseline exercise was increased, so the exact effects of the increased duration of exercise cannot be analyzed, but the results of the study indicated a long-lasting decrement of vagal activation after high-intensity endurance exercise. We did not investigate the effects of increased exercise duration on nocturnal HRV, but based on the results during short-term recovery, probably no differences would have been noticed, since differences between exercises were quite small at the end of the 15 min controlled recovery. In contrary, in a recent study by Myllymäki et al. (2012), exercise duration was increased from 30 minutes to 90 minutes at the intensity of 60 % vVO_{2max} in moderately physically active male subjects, which is quite similar to exercise protocols of the recreationally trained subjects of the present study. They found increased nocturnal HR after 90 min exercise when compared to 30 or 60 min exercises, and decreased HRV after 90 min exercise when compared to a control day without exercise. The effects of different exercises on nocturnal HRV may be affected by the time of day when exercise has been performed. In the present study, the aim was to perform exercises at the same time of day, but some variation existed in the exercise schedules. This, in

addition to the differences in physiological TL of exercises, may explain the differences in the results of these studies.

5.4 HRV and traditional training load parameters

Exercise-induced physiological TL has traditionally been estimated with heart rate, blood lactate, rating of perceived exertion (RPE), excess post-exercise oxygen consumption (EPOC) and different indices, for example training impulse (TRIMP) and session RPE. These traditional TL parameters measure mainly exercise intensity, but to some extent, also take exercise duration into account. Whether post-exercise HRV could be used in addition to, or instead of, these parameters was evaluated in this study. Until now, knowledge regarding the associations between traditional TL parameters and HRV is lacking, and only one study (Buchheit et al. 2009) has reported the relationships between immediate post-exercise HRV recovery and TL parameters, to the best of my knowledge.

A negative relationship between traditional TL parameters during exercise sessions and immediate post-exercise HRV was found in recreationally trained subjects. This finding could partly be expected, as traditional TL parameters are mainly affected by exercise intensity, and exercise intensity determines also, by large part, HRV during the recovery. The relationship seemed to be strongest between RPE at the end of the exercise and HRV, supporting the findings of previous studies where RPE has been found to correlate with cardiorespiratory and metabolic demands of exercise (Noble, 1982; Skinner et al. 1973). In the present study, it seemed that the association between RPE and HRV was not so visible in the lowest-intensity exercise of 60 % of vVO_{2max} of the recreationally trained subjects. It could be speculated that the large individual variation in vagal modulation during rest may affect the results of MODE exercise. Vagal activation decreases significantly around 50 - 60 % of VO_{2max} (Hautala et al. 2003; Tulppo et al. 1998), so the recovery after the exercise with the lowest physical TL, without significant contribution of anaerobic processes, might be near the individual resting level. When physical TL of exercise is increased, either by increased intensity or duration, HRV might better describe the actual physiological TL instead of the individual level of resting vagal modulation.

In addition to RPE, EPOC, blood lactate concentration and TRIMP were associated occasionally with HRV, but the relationships were weaker. The associations between HRV and blood lactate concentration of the present study were comparable to previous findings (Buchheit et al. 2009). EPOC might be the best "golden standard" parameter to measure physiological TL, being purely of physiological background. However, measuring EPOC demands laboratory circumstances and equipment, and should be measured for several hours. Therefore, EPOC cannot be used in daily training in field conditions. The present results indicate that the effects of different exercise sessions on HRV recovery are somewhat similar to the nature of EPOC, meaning that exercise intensity

is the main determinant of post-exercise HRV recovery when the intensity of the exercise is low, approximately at or below 50 % of VO_{2max}. However, even low intensity exercise seems to affect HRV recovery when the duration is prolonged quite clearly. The effect of duration was found remarkable when the exercise intensity is at least moderate, at least 60 % of VO_{2max}. Individual training status may have a great impact on the effects of different physical TL on HRV recovery. Therefore the results of the present study cannot be generalized to a great extent.

One finding in the present study was that even if the results of traditional training load parameters like HR, BLa, VO_2 and RPE and post-exercise HRV correlated, i.e. they differentiated exercises quite similarly, the exercise which seemed to be most strenuous according to traditional training load parameter, was not the same that delayed the recovery of HRV until the next morning. This is reasonable, as traditional training load parameters describe mainly the intensity of exercise, which may be increased only for few minutes in order to reach near maximal values. This result confirms the assumption that HRV may be more appropriate in estimating the physiological training load of exercise when compared to traditional training load parameters.

Recently, Saboul et al. (2015) published a pilot study of HRV in quantification of training load. They calculated an index of training load with pre- and post-exercise RMSSD and compared the results with TRIMP and session RPE. The experimental training sessions were of different duration and intensity, and so not matched for training volume. The results of Saboul et al. (2015) indicate that all three methods estimated training load quite similarly. In addition, other mechanisms than intensity seemed to affect HRV recovery, as HRV after the exercise of highest intensity (6 min, 100 % of maximal aerobic speed) was higher than after a lower intensity exercise (3x10 min, 3 min recovery period at 85 % of maximal aerobic speed).

One previous effort (Al Haddad et al. 2009) has been made to clarify the association between post-exercise nocturnal HRV and blood lactate concentration during exercise. They investigated nocturnal HRV in the second night after a supramaximal intermittent exercise (15 s running periods at 120% of vVO_{2peak}) and found correlations between vagal-related indices and blood lactate level after exercises. The results of Al Haddad et al. (2009) suggest that HRV may be used as indicator of physiological TL of endurance exercises, but further studies are needed to clarify the associations between HRV and BLa, especially during different, intermittent, anaerobic performances. In order to confirm the effects of the TL of single exercises on cardiac vagal function found on the present study, more studies are needed in subjects with different training backgrounds.

The association between HR/HRV recovery and physical TL during training periods of different durations has been studied for some extent (Borresen & Lambert, 2007; Buchheit & Gindre, 2006; Buchheit et al. 2004; Manzi et al. 2009). Usually, subjects with at least moderate training background have improved cardiac vagal activity when compared to sedentary subjects. Buchheit & Gindre (2006) hypothesized that the positive effect of TL on HRV found in many stud-

ies may actually be more attributed to improved cardiorespiratory fitness than increased TL itself. According to the results of their study, HR recovery may be more related to physical TL, whereas HRV may better describe the cardiorespiratory fitness. In the recent study of Duarte et al. (2015), a 12-wk training period improved vagal reactivation of healthy young men even in the absence of resting vagal modulation improvement. In the group with higher pre-training resting vagal control, only an improvement of vagal reactivation was detected whereas the group with lower vagal control, both resting vagal control and post-exercise vagal reactivation were improved. They concluded that the improvements in post-exercise vagal reactivation may not necessarily be related to an increase in resting vagal control (Duarte et al. 2015).

Studies have also found that some kind of non-linear dose-response may exist between physical TL and HRV. Manzi et al. (2009) found dose-related adaptations in ANS function during a 6-month training period of recreational marathon runners; a training period with highest $TRIMP_i$ yielded in lowest high-frequency HRV. In addition, Buchheit et al. (2004) found increased nocturnal vagal indices in subjects with moderate TL when compared to sedentary counterparts, but high TL did not produce further improvements in cardiac vagal function when compared to moderate TL (Buchheit et al. 2004).

To sum, according to model of supercompensation, an individual threshold probably exists, beyond which a greater TL is not always better than less, but may result in decreased physical performance, noticeable with declined cardiac vagal activity and decreased HRV.

5.5 Limitations

There are some limitations in the present study that must be taken into account. First, each study was performed with a small number of subjects because of practical limitations. Therefore, the results of each individual play an important role, and each great deviation from the mean may change the results significantly. However, no deviation of the results was found, which would have been abnormally different as compared to others. In addition, the study protocol was well reproducible, as the results of athletes, trained, and non-trained subjects were parallel.

A statistical power calculation was not made a priori because at that time, there were no earlier studies of immediate recovery of HRV after exercise. A posteriori calculations are not recommended to explain the results, but can be made in order to plan further studies (Hoenig & Heisey, 2001). The lack of power calculations does not interrupt the interpretation of the significant differences between exercises, because the significant difference between two exercises indicate sufficient power. Only it leaves an open question whether the non-significant differences between some exercises are related to lack of statistical power or not. To speculate, in the present study, the differences between such

exercises that did not reach statistical significance seemed minor, and were subjectively evaluated with no actual clinical relevance.

During rest, the changes in cardiac autonomic modulation, especially in vagal activation, can be described reliably with HRV. Validation studies using autonomic blockades have revealed a linear relationship between HRV and vagal effects on the heart (Akselrod et al. 1981; Berntson et al. 1993; Cacioppo et al. 1994; Martinmäki et al. 2006b). Although autonomic pharmacological blockade is usually used as the "golden standard" validation method for estimating autonomic modulation of the heart, it might be speculated that the true autonomic modulation may be altered via blockade, in addition to possible biases caused by incomplete blockades or nonselective actions of the blocking drugs (Berntson et al 1994).

In addition to rest, post-exercise HRV has been proven to detect changes in autonomic modulation (Ng et al. 2009). However, the exact relationship between cardiac autonomic functions and HRV during recovery after exercise is unclear. Although there are studies that have found linear relationships between vagal functions and HRV during an orthostatic task (Martinmäki et al. 2006b), during which fast changes, similar to recovery, occurs in autonomic modulation, may physical exercise change the response of autonomic nervous system. In addition, despite the linearity between vagal functions and HRV, the differences between individuals in the relation between vagal activity and HRV may be quite large (Martinmäki et al. 2006a).

Al Haddad et al (2011) investigated the reliability and repeatability of resting HRV, post-exercise HR recovery and different HRV indices on four different occasions. They found a lower coefficient of variation (CV) for HFP_{ln} during rest (13 %) when compared to immediate 5 minute recovery after supramaximal bouts of exercise (26 %). Acknowledging the possible sources of measurement biases, these were avoided in the present study by preparing the laboratory such that noise, temperature, lighting, and all test procedures would be similar between the different exercise sessions.

Each study was carried out with spontaneous breathing during rest, an exercise session, and a controlled recovery session. It is known that both tidal volume and breathing frequency may affect high frequency component of HRV at rest. Increased tidal volume has been found to increase HRV, whereas increased breathing frequency decreases it (Grossman et al. 1991; Keselbrener & Akselrod 1996). However, Bartels et al. (2004) suggested that HFP during exercise presents true cardiovascular vagal modulation instead of being affected by changes in ventilation. In some of the present investigations, breathing frequency was measured. We decided not to control breathing, since true autonomic modulation during recovery could have been disturbed by changes to natural breathing and second, the aim of the study was to find an applied method to use of HRV in evaluation of physiological TL, not only to interpret the modulation of HRV during recovery. In addition, as the dynamics of breathing frequency and HFP during immediate recovery were not similar, it is likely that the recovery of HFP after exercise was not affected by changes in breathing.

One limitation of the present study is related to the measurement of EPOC. EPOC was measured in the present study from 15 minutes of passive sitting recovery, using a 5 minute pre-exercise resting period as a baseline. Both the baseline and the length of the post-exercise measurement could have affected results of EPOC. The differences between exercises could have been different if EPOC was measured for longer period of time.

The chosen exercise protocols undoubtedly have a major effect on the results. The exercise sessions of the athletes, for example, were quite similar when the intensities and durations are compared. The high intensities were intentionally chosen to reflect typical exercise sessions for endurance athletes, but caused similar results as they all clearly disturbed body homeostasis and blunted exercise HRV to almost a minimum. More diverse exercise protocols could have given better insight regarding autonomic modulation after different exercise sessions. In addition, as the effects of exercise duration on post-exercise HRV were unclear, we did not know that increases in exercise duration, especially at low intensities, needed to be remarkable before the effects could be detected in HRV.

6 MAIN FINDINGS AND CONCLUSIONS

The main finding of this thesis was that differences in physical TL, either by increased exercise intensity or increased exercise duration could be detected by post-exercise heart rate variability already during the first few minutes after cessation of exercise. However, rather small increases in exercise intensity, especially if already at moderate or high level during which HRV is significantly decreased or even disappeared, do not necessarily induce further changes in autonomic modulation of the heart and therefore differences are not visible in post-exercise HRV. Similarly, the increase of exercise duration must be rather large to affect post-exercise HRV, especially, if the intensity of the exercise is low. In addition, it is possible that differences between exercises with high physical TL become visible only after longer recovery, since reactivation of cardiac autonomic modulation after high-intensity exercises may take longer than few minutes. The findings of immediate post-exercise HRV are somewhat comparable with the basic principles of "golden standard" method of TL measurement, post-exercise oxygen consumption. First, exercise intensity seemed to be the main factor determining post-exercise HRV, and second, a high enough intensity is needed to produce changes in HRV with increased exercise duration. Post-exercise dynamics of HRV depended on physical TL of the exercises. In general, fast recovery of HRV was detected already during the first two recovery minutes if the exercise intensity was low or moderate. In high-intensity exercises, the recovery of HRV was delayed, occurring either during the first five minutes or not until during the 30 minute period of recovery.

The conclusions of the present thesis are the following:

1) The immediate recovery of HRV after different endurance exercise sessions can be detected with time-frequency analysis method already during the first few minutes after exercise cessation. The recovery dynamics of HRV during the first recovery minutes is different from the typical dynamics of heart rate.

2) Increased exercise intensity can be detected as decreased HRV during immediate recovery. However, small increases in exercise intensity, especially if already at high intensity, do not necessarily cause further changes in post-exercise HRV.

3) An increase in exercise duration can be detected from immediate post-exercise HRV. However, at low exercise intensities, increased exercise duration does not necessarily cause changes in post-exercise HRV unless the increase in duration is remarkable.

4) Post-exercise HRV was inversely related to traditional training load parameters, and especially to RPE.

5) The present endurance exercises did not affect nocturnal HRV, but the effects of high-intensity exercises were visible on the following morning after awakening.

YHTEENVETO

Sykevariaatio sydämen autonomisen säätelyn kuvaajana yksittäisten harjoitusten aiheuttaman harjoituskuormituksen ja palautumisen arvioinnissa

Tahdosta riippumaton autonominen hermosto säätelee sydämen ja verenkiertoelimistön toimintaa sekä levossa että fyysisen rasituksen aikana. Autonomisen hermoston sympaattinen osa toimii useimpia elimistön toimintoja kiihdyttävänä ”moottorina”, ja sympaattisen hermoston lisääntynyt aktiivisuus mm. nostaa verenpainetta ja kiihdyttää sydämen sykettä sekä parantaa sydämen supistusvoimakkuutta. Sympaattinen hermoston on aktiivinen mm. fyysisen rasituksen aikana, jolloin elimistöltä vaaditaan lepotilannetta suurempaa kapasiteettia rasituksen vaatimuksiin sopeutumiseen. Parasympaattisen hermoston aktivaatio puolestaan aiheuttaa useimmissa elimissä päinvastaisia reaktioita kuin sympaattisen, ja se on pääasiallinen säätelymekanismi levon aikana. Parasympaattisen hermoston aktiivinen toiminta on yhteydessä hyvään sydämen ja verenkiertoelimistön kuntoon, ja fyysisen tai psyykkisen stressin sekä erilaisten sairauksien on todettu vähentävän parasympaattisen hermoston aktiivisuutta sekä sen herkkyyttä reagoida elimistöön kohdistuviin sisäisiin ja ulkoisiin stressitekijöihin.

Autonomisen hermoston toiminnan muutoksia seuraamalla on pyritty arvioimaan myös akuutin liikuntasuorituksen aiheuttamaa kuormitusta elimistölle. Autonomisen hermoston toimintaa ei voida mitata suoraan, mutta sydämen sykevälivaihtelun eli sykevariaation on osoitettu kuvaavan muutoksia autonomisen hermoston toiminnassa sekä levossa että erilaisten toimintakokeiden aikana. Aikaisemmissa tutkimuksissa on sykevariaation avulla havaittu, että autonomisen hermoston toiminnan palautumiseen liikuntasuorituksen jälkeen voi kulua tunneista jopa useampiin päiviin, riippuen liikuntasuorituksen kestosta ja tehosta. Sykevariaation välitöntä palautumisdynamiikkaa erilaisten harjoitusten jälkeen ei ole juurikaan tutkittu, koska aikaisemmin liikunta-alan tutkimuksissa on ollut käytössä analyysimenetelmiä jotka edellyttävät autonomisen säätelyn pysyvän muuttumattomana mittausjakson ajan. Näitä menetelmiä ei ole voitu käyttää välittömän palautumisen aikana, koska autonomisen hermoston toiminnassa tapahtuu tällöin nopeita muutoksia. Uudenlainen aikataajuusanalyysi mahdollistaa mittaukset myös niissä tilanteissa, joissa tapahtuu nopeita muutoksia autonomisessa säätelyssä.

Urheilijan harjoittelun tavoitteena on parhaan mahdollisen suorituskyvyn saavuttaminen oikealla hetkellä. Suorituskyvyn kehittyminen vaatii optimaalista kuormittumisen ja palautumisen suhdetta, minkä löytäminen ei käytännön harjoittelussa ole aina helppoa. Osatakseen optimoida harjoittelunsa, urheilijalla täytyy olla riittävästi tietoa yksittäisen harjoitusten aiheuttamasta vasteesta elimistössä ja harjoituskuormituksen vaatimasta palautumisajasta, mutta yksittäisen harjoituksen aiheuttamaa kuormitusta ei ole tähän mennessä voitu luotettavasti mitata jokapäiväisessä harjoittelussa minkään yksittäisen fysiologisen mittarin avulla.

Tämän tutkimuksen tarkoituksena oli selvittää, minkälainen on sykevariaation välitön palautumisdynamiikka erilaisten kestävyysharjoitusten jälkeen. Toisena tavoitteena oli selvittää voidaanko muutoksia harjoituskuormituksessa, eli harjoituksen tehossa tai kestossa, havaita sykevariaation avulla ensimmäisten palautumisminuuttien aikana.

Erimittaisten ja –tehoisten tasavauhtisten ja intervallijuoksuharjoitusten vaikutusta sykevariaation palautumiseen seurattiin kontrolloidusti sekä harjoitelleilla että harjoittelemattomilla henkilöillä. Harjoitukset olivat teholtaan 50 % - 105 % verrattuna tutkittavien maksimaalisessa testissä saavuttamasta työmäärästä, ja harjoitukset olivat pituudeltaan 3 km – 14 km. Tulokset osoittivat että sykevariaation palautumisdynamiikka oli erilainen kuin sykkeen, ja että sykevariaation palautumisdynamiikassa tapahtui muutoksia kun harjoituskuormitusta muutettiin joko harjoituksen kestoa tai tehoa lisäämällä. Harjoituksen teho oli tulosten perusteella merkittävin sykevariaation palautumiseen vaikuttava tekijä, mutta jos tehoa lisättiin jo ennestään kovatehoiseen harjoitukseen, ei eroja harjoitusten välillä havaittu. Myös harjoituksen keston lisäys havaittiin hidastuneena sykevariaation palautumisena jos keston lisäys oli merkittävä, ja harjoituksen teho vähintään kohtalainen. Tulosten perusteella voitiin myös havaita, että sykevariaation palautuminen oli käänteisesti verrannollinen muihin harjoituskuormitusta kuvaaviin muuttujiin, kuten subjektiivisesti koettuun rasituksen tunteeseen, veren laktaattipitoisuuteen tai harjoituksen jälkeiseen ylimääräiseen hapenkulutukseen.

Tutkimuksen tulosten perusteella voidaan todeta, että erot kestävyysharjoitusten harjoituskuormituksessa voitiin havaita heti ensimmäisten palautumisminuuttien aikana aikataajuusmenetelmällä analysoidun sykevariaation avulla. Sykevariaation avulla on mahdollista saada entistä tarkempaa tietoa yksittäisen harjoituksen aiheuttamasta fysiologisesta harjoituskuormituksesta ja sen aiheuttamista muutoksista sydämen autonomisessa säätelyssä. Tämä voi osaltaan auttaa harjoittelun suunnittelussa ja toteutuksessa ja sitä kautta edistää suorituskyvyn kehittymistä sekä urheilijoilla että muilla tavoitteellisesti liikuntaa harrastavilla. Menetelmän avulla tieto on mahdollista saada jo muutaman minuutin seurannalla harjoituksen jälkeen ilman monimutkaisia ja kalliita laboratoriomittauksia.

REFERENCES

Achten, J. & Jeukendrup, A. 2003. Heart rate monitoring. Applications and limitations. Sports Medicine 33, 517-538.

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

Akselrod, S., Gordon, D., Madwed, J., Snidman, N., Shannon, D. & Cohen, R. 1985. Hemodynamic regulation: investigation by spectral analysis. American Journal of Physiology: Heart and Circulatory Physiology 249, H867-875.

Al Haddad, H., Laursen, P., Ahmaidi, S. & Buchheit, M. 2009. Nocturnal heart rate variability following supramaximal intermittent exercise. International Journal of Sports Physiology and Performance 4, 435–447.

Al Haddad, H., Laursen, P., Chollet, D., Ahmaidi, S. & Buchheit, M. 2011. Reliability of resting and postexercise heart rate measures. International Journal of Sports Medicine 32, 598-605.

Arai, Y., Saul, J., Albrecht, P., Hartley, L., Lilly, L., Cohen, R. & Colucci, W. 1989. Modulation of cardiac autonomic activity during and immediately after exercise. American Journal of Physiology: Heart and Circulatory Physiology 256, H132–H141.

Arts, F. & Kuipers, H. 1994. The relation between power output, oxygen uptake and heart rate in male athletes. International Journal of Sports Medicine 15, 228-231.

Aunola, S. & Rusko, H. 1984. Reproducibility of aerobic and anaerobic thresholds on 20-50 year old men. European Journal of Applied Physiology 53, 260-266.

Åstrand, P-O. & Rodahl, K. 1986. Textbook of work physiology. Physiological basis of exercise. New York: McGraw-Hill Book Company.

Banister, E. 1991. Modelling elite athletic performance. In MacDougall J.D., Wenger H.A. and Green H.J. (editors) Physiological Testing of the High-Performance Athlete 2nd edition. Champaign: Human Kinetics Publishers Ltd p. 403-424.

Bartels, M., Jelic, S., Ngai, P., Gates, G., Newandee, D., Reisman, S., Basner, R. & De Meersman, R. 2004. The effect of ventilation on spectral analysis of heart rate variability during exercise. Respiratory Physiology & Neurobiology 144, 91–98.

Baumert, M., Brechtel, L., Lock, J., Hermsdorf, M., Wolff, R., Baier, V. & Voss, A. 2006. Heart rate variability, blood pressure variability and baroreflex sensitivity in overtrained athletes. Clinical Journal of Sports Medicine 16, 412-417.

Bernardi, L., Salvucci, F., Suardi, R., Solda, P., Calciati, A., Perlini, S., Falcone, C. & Ricciardi, L. 1990. Evidence for an intrinsic mechanism regulating heart rate variability in the transplanted and the intact heart during submaximal dynamic exercise? Cardiovascular Research 24, 969–981.

Berntson, G., Cacioppo, J. & Quigley, K. 1993. Respiratory sinus arrhythmia: autonomic origins, physiological mechanisms, and psychophysiological implications. Psychophysiology 30, 183–196.

Berntson, G., Cacioppo, J. & Quigley, K. 1994. Autonomic cardiac control. I. Estimation and validation from pharmacological blockades. Psychophysiology 31, 572-585.

Berntson, G., Bigger, J., Eckberg, D., Grossman, P., Kaufmann, P., Malik, M., Nagaraja, H., Porges, S., Saul, J., Stone, P. & Van Der Molen, M. 1997. Heart rate variability: Origins, methods and interpretive caveats. Psychophysiology 34, 623-648.

Borg, G. 1982. Psychophysical bases of perceived exertion. Medicine and Science in Sports and Exercise 14, 377-381.

Borresen, J. & Lambert, M. 2007. Changes in heart rate recovery in response to acute changes in training load. European Journal of Applied Physiology 101, 503-511.

Borresen, J. & Lambert, M. 2008a. Autonomic control of heart rate during and after exercise. Sports Medicine 38, 633-646.

Borresen, J. & Lambert, M. 2008b. Quantifying training load: a comparison of subjective and objective methods. International Journal of Sports Physiology and Performance 3, 16-30.

Borresen, J. & Lambert, M. 2009. The quantification of training load, the training response and the effect on performance. Sports Medicine 39, 779-795.

Børsheim, E. & Bahr, R. 2003. Effect of exercise intensity, duration and mode on post-exercise oxygen consumption. Sports Medicine 33, 1037-1060.

Bosquet, L., Gamelin, F.-X. & Berthoin, S. 2008. Reliability of post-exercise heart rate recovery. International Journal of Sports Medicine 29, 238-243.

Bouchard, C. & Rankinen, T. 2001. Individual differences in response to regular physical activity. Medicine and Science in Sports and Exercise 33, S446-S451.

Buchheit, M., Simon, C., Piquard, F., Ehrhart, J. & Brandenberger, G. 2004. Effects of increased training load on vagal-related indexes of heart rate variability: a novel sleep approach. American Journal of Physiology: Heart and Circulatory Physiology 287, H2813-2818.

Buchheit, M. & Gindre, C. 2006. Cardiac parasympathetic regulation: respective associations with cardiorespiratory fitness and training load. American Journal of Physiology: Heart and Circulatory Physiology 291, H451-H458.

Buchheit, M., Laursen, P. & Ahmaidi, S. 2007a. Parasympathetic reactivation after repeated sprint exercise. American Journal of Physiology: Heart and Circulatory Physiology 293, H133-H141.

Buchheit, M., Papelier, Y., Laursen, P. & Ahmaidi, S. 2007b. Noninvasive assessment of cardiac parasympathetic function: postexercise heart rate recovery or heart rate variability? American Journal of Physiology: Heart and Circulatory Physiology 293, H8-H10.

Buchheit, M., Al Haddad, H., Laursen, B. & Ahmaidi, S. 2009. Effect of body posture on postexercise parasympathetic reactivation in men. Experimental Physiology 94, 795-804.

Buchheit, M., Chivot, A., Parouty, J., Mercier, D., Al Haddad, H., Laursen, P. & Ahmaidi, S. 2010. Monitoring endurance running performance using cardiac parasympathetic function. European Journal of Applied Physiology 108, 1153-1167.

Byrne, E., Fleg, J., Vaitkevicius, P., Wright, J. & Porges, S. 1996. Role of aerobic capacity and body mass index in the age-associated decline in heart rate variability. Journal of Applied Physiology 81, 743-750.

Cacioppo, J., Berntson, G., Binkley, P., Quigley, K., Uchino, B. & Fieldstone, A. 1994. Autonomic cardiac control. II. Noninvasive indices and basal response as revealed by autonomic blockades. Psychophysiology 31, 586 – 598.

Cannon, W. 1914. The emergency function of the adrenal medulla in pain and the major emotions. American Journal of Physiology 33, 356-372.

Carter, J., Banister, E. & Blaber, A. 2003. Effect of endurance exercise on autonomic control of heart rate. Sports Medicine 33, 33-46.

Casadei, B., Cochrane, S., Johnston, J., Conway, J. & Sleight, P. 1995. Pitfalls in the interpretation of spectral analysis of the heart rate variability during exercise in humans. Acta Physiology Scandica 153, 125–131.

Casadei, B., Moon, J., Johnston, J., Caiazza, A. & Sleight, P. 1996. Is respiratory sinus arrhythmia a good index of vagal tone in exercise? Journal of Applied Physiology 81, 556–564.

Casties, J.-F., Mottet, D. & Le Gallais, D. 2006. Non-linear analysis of heart rate variability during heavy exercise and recovery in cyclists. International Journal of Sports Medicine 27, 780-785.

Cohen, J. & Segal, K. 1985. Left ventricular hypertrophy in athletes: an exercise-echocardiographic study. Medicine and Science in Sports and Exercise 17, 695-700.

Cohen, L. 1989. Time-frequency distributions - a review. Proceedings of the IEEE. 77, 941–981.

Cole, C., Blackstone, E., Pashkow, F., Snader, C. & Lauer, M. 1999. Heart-rate recovery immediately after exercise as a predictor of mortality. New England Journal of Medicine 341, 1351-1357.

Cottin, F., Médigue, C., Leprêtre, P., Papelier, Y., Koralsztein, P. & Billat, V. 2004. Heart rate variability during exercise performed below and above ventilatory threshold. Medicine and Science in Sports and Exercise 36, 594-600.

Cottin, F., Leprêtre, P., Lopes, P., Papelier, Y., Médigue, C. & Billat, V. 2006. Assessment of ventilator thresholds from heart rate variability in well-trained subjects during cycling. International Journal of Sports Medicine 27, 959-967.

Coyle, E. & González-Alonso, J. 2001. Cardiovascular drift during prolonged exercise: new perspectives. Exercise and Sports Science Reviews 29, 88-92.

Daanen, H., Lamberts, R., Kallen, V., Jin, A. & Van Meeteren, N. 2012. A systematic review on heart rate recovery to monitor changes in training status in athletes. International Journal of Sports Physiology and Performance 7, 251-260.

Duarte, A., Soares, P., Pescatello, L. & Farinatti, P. 2015. Aerobic training improves vagal reactivation regardless of resting vagal control. Medicine and Science in Sports and Exercise. Published ahead of print. DOI: 10.1249/MSS.000000000000 0532.

Eckberg, D. 1976. Temporal response patterns of the human sinus node to brief carotid baroreceptor stimuli. Journal of Physiology 258, 769-782.

Eckberg, D. 1980. Phase relationship between normal human respiration and baroreflex responsiveness. Journal of Physiology 304, 489-502.

Eckberg, D. 1997. Sympathovagal balance: A critical appraisal. Circulation 96, 3224-3232.

Elsenbruch, S., Wang, Z., Orr, W. & Chen, J. 1999. Time–frequency analysis of heart rate variability using short-time Fourier analysis. Physiological Measurement 21, 229–240.

Foster, C. 1998. Monitoring training in athletes with reference to overtraining syndrome. Medicine and Science in Sports and Exercise 30, 1164-1168.

Fukusaki, C., Kawakubo, K. & Yamamoto, Y. 2000. Assessment of the primary effect of aging on heart rate variability in humans. Clinical Autonomic Research 10, 123-130.

Gladwell, V., Sandercock, G. & Birch S. 2010. Cardiac vagal activity following three intensities of exercise in humans. Clinical Physiology and Functional Imaging 30, 17-22.

Goldberger, J., Le, F., Lahiri, M., Kannankeril, P., Ng, J. & Kadish, A. 2006. Assesment of parasympathetic reactivation after exercise. American Journal of Physiology: Heart and Circulatory Physiology 290, H2446-H2452.

Gore, C. & Withers, R. 1990. The effect of exercise intensity and duration on the oxygen deficit and excess post-exercise oxygen consumption. European Journal of Applied Physiology 60, 169-174.

Grossman, P., Karemaker, J., Wieling, W. 1991. Prediction of tonic parasympathetic cardiac control using respiratory sinus arrhythmia: the need for respiratory control. Psychophysiology 28, 201-216.

Guerra, Z., Peçanha, T., Moreira, D., Silva, L., Laterza, M., Nakamura, F. & Lima, J. 2014. Effects of load and type of physical training on resting and postexercise cardiac autonomic control. Clinical Physiology and Functional Imaging 34, 114-120.

Hainsworth, R. 1998. Physiology of the cardiac autonomic system. In: Malik, M (ed.) Clinical guide to cardiac autonomic tests. The Netherlands: Kluwer Academic Publishers, 3-29.

Hartikainen, J., Tahvanainen, K. & Kuusela, T. 1998. Short-term measurement of heart rate variability. In: Clinical guide to cardiac autonomic tests. Malik, M. The Netherlands: Kluwer Academic Publishers, 149-176.

Hautala, A., Tulppo, M., Mäkikallio, T., Laukkanen, R., Nissilä, S. & Huikuri, H. 2001. Changes in cardiac autonomic regulation after prolonged maximal exercise. Clinical Physiology 21, 238-245.

Hautala, A., Mäkikallio, T., Kiviniemi, A., Laukkanen, R., Nissilä, S., Huikuri, H. & Tulppo, M. 2003. Cardiovascular autonomic function correlates with the response to aerobic training in healthy sedentary subjects. American Journal of Physiology: Heart and Circulatory Physiology 285, H1747-H1752.

Hayano, J., Sakakibara, Y., Yamada, A., Yamada, M., Mukai, S., Fujinami, T., Yokoyama, K., Watanabe, Y. & Takata, K. 1991. Accuracy of assessment of cardiac vagal tone by heart rate variability in normal subjects. American Journal of Cardiology 67, 199-204.

Hoenig, J. & Heisey, D. 2001. The abuse of power: the pervasive fallacy of power calculations for data analysis. The American Statistician 55, 1-6.

Hopkins, W. 1991. Quantification of training in competitive sports: methods and applications. Sports Medicine 12, 161-183.

Huikuri, H., Pikkujämsä, S., Airaksinen, J., Ikäheimo, M., Rantala, A., Kauma, H., Lilja, M. & Kesäniemi, A. 1996. Sex-related differences in autonomic modulation of heart rate in middle-aged subjects. Circulation 94, 122-125.

Hynynen, E., Uusitalo, A., Konttinen, N. & Rusko, H. 2006. Heart rate variability during night sleep and after awakening in overtrained athletes. Medicine and Science in Sports and Exercise 38, 313-317.

Hynynen, E., Nummela A., Rusko, H., Hämäläinen, I. & Jylhä, R. 2007. Effects of training on cardiac autonomic modulation during night sleep in cross country skiers. In : Linnamo, V., Komi, P., Müller, E. (Eds) Science in nordic skiing, Vol. UK: Meyer & Meyer Sport Ltd., 90-98.

Hynynen, E., Vesterinen, V., Rusko, H. & Nummela, A. 2010. Effects of moderate and heavy endurance exercise on nocturnal HRV. International Journal of Sports Medicine 31, 428-432.

Iellamo, F. 2001. Neural mechanisms of cardiovascular regulation during exercise. Autonomic Neuroscience: Basic and Clinical 90, 66-75.

Imai, K., Sato, H., Hori, M., Kusuoka, H., Ozaki, H., Yokoyama, H., Takeda, H., Inoue, M. & Kamada, T. 1994. Vagally mediated heart rate recovery after exercise is accelerated in athletes but blunted in patients with chronic heart failure. Journal of American College of Cardiology 24, 1529–1535.

Javorka, M., Zila, I., Balharek, T. & Javorka, K. 2002. Heart rate recovery after exercise: relations to heart rate variability and complexity. Brazilian Journal of Medical and Biological Research 35, 991-1000.

Kamath, M., Fallen, E. & McKelvie, R. 1991. Effects of steady state exercise on the power spectrum of heart rate variability. Medicine and Science in Sports and Exercise 23, 428-434.

Kannankeril, P., Le, K., Kadish, A. & Goldberger, J. 2004. Parasympathetic effects on heart rate recovery after exercise. Journal of Investigative Medicine 52, 394–401.

Karvonen, M., Kentala, E. & Mustala, O. 1957. The effects of training on heart rate: a longitudinal study. Annales Medicinae Experimentalis Et Biologiae Fenniae 35, 307-315.

Katona, P. & Jih, F. 1975. Respiratory sinus arrhythmia: noninvasive measure of parasympathetic cardiac control. Journal of Applied Physiology 39, 801-805.

Kellmann, M. 2010. Preventing overtraining in athletes in high-intensity sports and stress/recovery monitoring. Scandinavian Journal of Medicine and Science in Sports 20, 95-112.

Keselbrener, L. & Akselrod, S. 1996. Selective discrete Fourier Transform algorithm for time-frequency analysis: method and application on simulated and cardiovascular signals. IEEE Transactions on Biomedical Engineering 43, 789-802.

Kupper, N., Willemsen, G., van den Berg, M., de Boer, D., Posthuma, D., Boomsma, D. & de Geus, E. 2004. Heritability of ambulatory heart rate variability. Circulation 110, 2792-2796.

Lambert, M. & Borresen, J. 2010. Measuring training load in sports. International Journal of Sports Physiology and Performance 5, 406-411.

Lamberts, R. & Lambert, M. 2009. Day-to-day variation in heart rate at different levels of submaximal exertion: implications for monitoring training. Journal of Strength and Conditioning Research 23, 1005-1010.

Lamberts, R., Swart, J., Noakes, T. & Lambert, M. 2009a. Changes in heart rate recovery after high-intensity training in well-trained cyclists. European Journal of Applied Physiology 105, 705-713.

Lamberts, R., Swart, J., Capostagno, B., Noakes, T. & Lambert, M. 2009b. Heart rate recovery as a guide to monitor fatique and predict changes in performance parameters. Scandinavian Journal of Medicine and Science in Sports 20, 449-457.

La Rovere, M., Bigger, J., Marcus, F., Mortara, A. & Schwartz, P. 1998. Baroreflex sensitivity and heart rate variability in prediction of total cardiac mortality after myocardial infarction. The Lancet 351, 478-484.

La Rovere, M., Pinna, G. &Mortara, A. 1998. Assessment of baroreflex sensitivity. In: Clinical guide to cardiac autonomic tests. Malik, M. The Netherlands: Kluwer Academic Publishers, p.257-281.

Macor, F., Fagard, R. & Amery, A. 1996. Power spectral analysis of RR interval and blood pressure short-term variability at rest and dynamic exercise: comparison between cyclists and controls. International Journal of Sports Medicine 17, 175-181.

Malliani, A., Pagani, M. & Lombardi, F. 1994. Physiology and clinical implications of variability of cardiovascular parameters with focus on heart rate and blood pressure. American Journal of Cardiology 73, 3C-9C.

Manzi, V., Castagna, C., Padua, E., Lombardo, M., D'Ottavio, S., Massaro, M., Volterrani, M. & Iellamo, F. 2009. Dose-response relationship of autonomic nervous system responses to individualized training impulse in marathon

runners. American Journal of Physiology: Heart and Circulatory Physiology 296, H1733-40.

Martinmäki, K., Rusko, H., Saalasti, S. & Kettunen, J. 2006a. Ability of short-time Fourier Transform method to detect transient changes in vagal effects on hearts: a pharmacological blocking study. American Journal of Physiology: Heart and Circulatory Physiology 290, H2582-H2589.

Martinmäki, K., Rusko, H., Kooistra, L., Kettunen, J. & Saalasti, S. 2006b. Intraindividual validation of heart rate variability indices to measure vagal effects on hearts. American Journal of Physiology: Heart and Circulatory Physiology 290, H640-H647.

Martinmäki, K. & Rusko, H. 2008. Time-frequency analysis of heart rate variability during immediate recovery from low and high intensity exercise. European Journal of Applied Physiology 102, 353-360.

Mazzeo, R. 1991. Catecholamine responses to acute and chronic exercise. Medicine and Science in Sports and Exercise 23, 839-845.

McGarvey, W., Jones, R. & Petersen, S. 2005. Excess post-exercise oxygen consumption following continuous and interval cycling exercise. International Journal of Sport Nutrition and Exercise Metabolism 14, 28-37.

Meeusen, R., Duclos, M., Gleeson, M., Rietjens, G., Steinacker, J. & Urhausen, A. 2012. Prevention, diagnosis and treatment of the overtraining syndrome. Joint consensus statement of the European college of sport science and the American college of sports medicine. Medicine and Science in Sports and Exercise 45, 186-205.

Miles, D., Sawka, M., Hanpeter, D., Foster, J., Doerr, B. & Basset Frey, M. 1984. Central hemodynamics during progressive upper and lower body exercise and recovery. Journal ofApplied Physiology 57, 366-370.

Mitchell, J., Kaufman, M. & Iwamoto, G. 1983. The exercise pressor reflex: Its cardiovascular effects, afferent mechanisms and central pathways. Annual Review of Physiology 45, 229.

Moak, J., Goldstein, D., Eldadah, B., Saleem, A., Holmes, C., Pechnik, S. & Sharabi, Y. 2007. Supine low-frequency power of heart rate variability reflects baroreflex function, not sympathetic innervation. Heart Rhythm 4, 1523-1529.

Moodithaya, S. & Avadhany, S. 2012. Gender differences in age-related changes in cardiac autonomic nervous function. Journal of Aging Research doi: 10.1155 / 2012 / 679345.

Mølgaard, H., Sørensen, K. & Bjerregaard, P. 1991. Attenuated 24-h heart rate variability in apparently healthy subjects, subsequently suffering sudden cardiac death. Clinical Autonomic Research 1, 233-237.

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 continuous and interval training exercises. European Journal of Applied Physiology 92, 508–517.

Myllymäki, T., Rusko, H., Syväoja, H., Juuti, T., Kinnunen, M.-L. & Kyröläinen, H. 2012. Effects of exercise intensity and duration on nocturnal heart rate variability and sleep quality. European Journal of Applied Physiology 112, 801-809.

Nakamura, Y., Yamamoto, Y. & Muraoka, I. 1993. Autonomic control of heart rate during physical exercise and fractal dimension of heart rate variability. Journal of Applied Physiology 74, 875-881.

Ng, J., Sundaram, S., Kadish, A. & Goldberger, J. 2009. Autonomic effects on the spectral analysis of heart rate variability after exercise. American Journal of Physiology: Heart and Circulatory Physiology 297, H1421-H1428.

Nissinen, S., Mäkikallio, T., Seppänen, T., Tapanainen, J., Salo, M., Tulppo, M. & Huikuri, H. 2003. Heart rate recovery after exercise as a predictor of mortality among survivors of acute myocardial infarction. American Journal of Cardiology 91, 711-714.

Noble, B. 1982. Clinical applications of perceived exertion. Medicine and Science in Sports and Exercise 14, 406–411.

Nummela, A., Hynynen, E., Kaikkonen, P. & Rusko, H. 2010. Endurance performance and nocturnal HRV indices. International Journal of Sports Medicine 31, 154-159.

Oida, E., Moritani, T. & Yamori, Y. 1997. Tone-entropy analysis on cardiac recovery after dynamic exercise. Journal of Applied Physiology 82, 1794-1801.

O'Leary, D. 1993. Autonomic mechanisms of muscle metaboreflex control of heart rate. Journal of Applied Physiology 74, 1748-1754.

Oliveira, T., Mattos, R., Silva, R., Rezende, R. & Lima, J. 2013. Absence of parasympathetic reactivation after maximal exercise. Clinical Physiology and Functional Imaging 33, 143-149.

Oppenheim, A. & Schafer, R. 1999. Discrete-time signal processing. Prentice Hall, Upper Saddle River, NJ.

Parekh, A. & Lee, M. 2005. Heart rate variability after isocaloric exercise bouts of different intensities. Medicine and Science in Sports and Exercise 37, 599–605.

Perini, R., Orizio, C., Comandè, A., Castellano, M., Beschi, M. & Veicsteinas, A. 1989. Plasma norepinephrine and heart rate dynamics during recovery from submaximal exercise in man. European Journal of Applied Physiology 58, 879–883.

Perini, R., Orizio, C., Baselli, G., Cerutti, S. & Veicsteinas, A. 1990. The influence of exercise intensity on the power spectrum of heart rate variability. European Journal of Applied Physiology 61, 143–148.

Pichon, A., De Bisschop, C., Roulaud, M., Denjean, A. & Papelier, Y. 2004. Spectral analysis of heart rate variability during exercise in trained subjects. Medicine and Science in Sports and Exercise 36, 1702–1708.

Pichot, V., Roche, F., Gaspoz, J., Enjolras, F., Antoniadis, A., Minini, P., Costes, F., Busso, T., Lacour, J. & Barthelemy, J. 2000. Relation between heart rate

variability and training load in middle-distance runners. Medicine and Science in Sports and Exercise 32, 1729-1736.

Plotnick, G., Becker, L., Fisher, M., Gerstenblith, G., Renlund, D., Fleg, J., Weisfeldt, M. & Lakatta, E. 1986. Use of the Frank-Starling mechanism during submaximal versus maximal upright exercise. American Journal of Physiology: Heart and Circulatory Physiology 251, H1101-H1105.

Pola, S., Macerata, A., Emdin, M. & Marchesi, C. 1996. Estimation of the power spectral density in nonstationary cardiovascular time series: assessing the role of the time-frequency representations (TFR). IEEE Transactions on Biomedical Engineering 43, 46-59.

Pomeranz, B., Macaylay, R., Caudill, M., Kutz, I., Adam, D., Gordon, D., Kilborn, K., Barger, A., Shannon, D. & Cohen, R. 1985. Assessment of autonomic function in humans by heart rate spectral analysis. American Journal of Physiology: Heart and Circulatory Physiology 248, H151-153.

Porges, S. 1992. Vagal tone: a physiological marker of stress vulnerability. Pediatrics 90, 498-504.

Porges, S. 1995. Cardiac vagal tone: A physiological index of stress. Neuroscience and biobehavioral reviews 19, 225-233.

Rahman, F., Pechnik, S., Gross, D., Sewell, L. & Goldstein, D. 2011. Low frequency power of heart rate variability reflects baroreflex function, not cardiac sympathetic innervation. Clinical Autonomic Research 21, 133-141.

Rowell, L. & O'Leary, D. 1990. Reflex control of the circulation during exercise: chemoreflexes and mechanoreflexes. Journal of Applied Physiology 69, 407-418.

Saalasti, S. 2003. Neural networks for heart rate time series analysis. Ph.D. Dissertation. Department of Mathematical Information Technology, University of Jyväskylä, Finland. Jyväskylä Studies in Computing 33, University of Jyväskylä, Jyväskylä.

Saboul, D., Balducci, P., Millet, G., Pialoux, V. & Hautier, C. 2015. A pilot study on quantification of training load: The use of HRV in training practice. European Journal of Sport Sciences. http://dx.doi.org/10.1080/17461391.-2015.1004373.

Sandercock, G., Bromley, P. & Brodie, D. 2005a. Effects of exercise on heart rate variability: inferences from meta-analysis. Medicine and Science in Sports and Exercise 37, 433-439.

Sandercock, G., Bromley, P. & Brodie, D. 2005b. The reliability of short-term measurements of heart rate variability. International Journal of Cardiology 103, 238-247.

Savin, W., Davidson, D. & Haskell, W. 1982. Autonomic contribution to heart rate recovery from exercise in humans. Journal of Applied Physiology 53, 1572-1575.

Scheuer, J. & Tipton, C. 1977. Cardiovascular adaptations to physical training. Annual Review of Physiology 39, 221-251.

Seiler, S., Haugen, O. & Kuffel, E. 2007. Autonomic recovery after exercise in trained athletes: intensity and duration effects. Medicine and Science in Sports and Exercise 39, 1366-1373.

Semrád, B., Fišer, B. & Honzíková, N. 1998. Ageing and cardiac autonomic status. In: Malik, M (ed.) Clinical guide to cardiac autonomic tests. The Netherlands: Kluwer Academic Publishers, 285-300.

Singh, J., Larson, M., O'Donnell, C., Tsuji, H., Evans, J. & Levy, D. 1999. Heritability of heart rate recovery. Circulation 99, 2251-2254.

Skinner, J., Hustler, R., Bergsteinova, V. & Buskirk, E. 1973. The validity and reliability of a rating scale of perceived exertion. Medicine and Science in Sports 5, 97–103.

Stein, R., Medeiros, C., Rosito, G., Zimerman, L. & Ribeiro, J. 2002. Intrinsic sinus and atrioventricular node electrophysiological adaptations in endurance athletes. Journal of American College of Cardiology 39, 1033-1038.

Strange, S., Secher, N., Pawelczyk, J., Karpakka, J., Christensen, N., Mitchell, J. & Saltin, B. 1993. Neural control of cardiovascular responses and of ventilation during dynamic exercise in man. Journal of Physiology 470, 693-704.

Takahashi, T., Okada, A., Saitoh, T., Hayano, J. & Miyamoto, Y. 2000. Difference in human cardiovascular response between upright and supine recovery from upright cycle exercise. European Journal of Applied Physiology 81, 233–239.

Tarkiainen, T., Timonen, K., Tiittanen, P., Hartikainen, J., Pekkanen, J., Hoek, G., Ibald-Mulli, A. & Vanninen, E. 2005. Stability over time of short-term heart rate variability. Clinical Autonomic Research 15, 394-399.

Task Force of the European Society of Cardiology the North American Society of Pacing Electrophysiology. 1996. Circulation 93, 1043-1065.

Taylor, J., Carr, D., Myers, C. & Eckberg, D. 1998. Mechanisms underlying very-low-frequency RR-interval oscillations in humans. Circulation 98, 547-555.

Terziotti, P., Schena, F., Gulli, G. & Cevese, A. 2001. Post-exercise recovery of autonomic cardiovascular control: a study by spectrum and cross-spectrum analysis in humans. European Journal of Applied Physiology 84, 187–194.

Tulppo, M., Mäkikallio, T., Seppänen, T., Laukkanen, R. & Huikuri, H. 1998. Vagal modulation of heart rate during exercise: effects of age and physical fitness. American Journal of Physiology: Heart and Circulatory Physiology 274, H424–H429.

Tulppo, M., Kiviniemi, A., Hautala, A., Kallio, M., Seppänen, T., Tiinanen, S., Mäkikallio, T. & Huikuri, H. 2011. Sympatho-vagal interaction in the recovery phase of exercise. Clinical Physiology and Functional Imaging 31, 272-281.

Ulrich-Lai, Y. & Herman, P. 2009. Neural regulation of endocrine and autonomic stress responses. Nature Reviews Neuroscience 10, 397-409.

Urhausen, A. & Kindermann, W. 1999. Sports-specific adaptations and differentiation of the athlete's heart. Sports Medicine 28, 237-244.

Uusitalo, AL., Tahvanainen, K., Uusitalo, AJ. & Rusko, H. 1996. Non-invasive evaluation of sympathovagal balance in athletes by time and frequency-domain analyses of heart rate and blood pressure variability. Clinical Physiology 16, 575-588.

Uusitalo, AL. 2001. Overtraining: making a difficult diagnosis and implementing targeted treatment. Physician and Sportsmedicine 29, 35-50.

Uusitalo, AL., Vanninen, E., Levälahti, E., Battié, M., Videman, T. & Kaprio, J. 2007. Role of genetic and environmental influences on heart rate variability in middle-aged men. American Journal of Physiology: Heart and Circulatory Physiology 293, H1013-1022.

Vandeput, S., Verheyden, B., Aubert, A. & Van Huffel, S. 2012. Nonlinear heart rate dynamics: circadian profile and influence of age and gender. Medical Engineering & Physics 34, 108-117.

Vuorimaa, T., Vasankari, T. & Rusko, H. 2000. Comparison of physiological strain and muscular performance of athletes during two intermittent running exercises at the velocity associated with VO_{2max}. International Journal of Sports Medicine 21, 96–101.

Yamamoto, Y., Hughson, R. & Peterson, J. 1991. Autonomic control of heart rate during exercise studied by heart rate variability spectral analysis. Journal of Applied Physiology 71, 1136–1142.

ORIGINAL PAPERS

I

POST-EXERCISE HEART RATE VARIABILITY OF ENDURANCE ATHLETES AFTER HIGH-INTENSITY EXERCISE INTERVENTIONS

by

Piia Kaikkonen, Heikki Rusko & Kaisu Martinmäki 2008

Scandinavian Journal of Medicine and Science in Sports 18, 511–519

http://onlinelibrary.wiley.com/doi/10.1111/j.1600-0838.2007.00728.x/abstract

II

HEART RATE VARIABILITY DYNAMICS DURING EARLY RECOVERY AFTER DIFFERENT ENDURANCE EXERCISES

by

Piia Kaikkonen, Ari Nummela & Heikki Rusko 2007

European Journal of Applied Physiology 102, 79–86

http://link.springer.com/article/10.1007/s00421-007-0559-8

III

CAN HRV BE USED TO EVALUATE TRAINING LOAD IN CONSTANT LOAD EXERCISES?

by

Piia Kaikkonen, Esa Hynynen, Theresa Mann,
Heikki Rusko & Ari Nummela 2010

European Journal of Applied Physiology 108, 435–442

http://link.springer.com/article/10.1007%2Fs00421-009-1240-1

IV

HEART RATE VARIABILITY IS RELATED TO TRAINING LOAD VARIABLES IN INTERVAL RUNNING EXERCISES

by

Piia Kaikkonen, Esa Hynynen, Theresa Mann,
Heikki Rusko & Ari Nummela 2012

European Journal of Applied Physiology 112, 829–838

http://link.springer.com/article/10.1007/s00421-011-2031-z