STUDIES IN SPORT, PHYSICAL EDUCATION AND HEALTH
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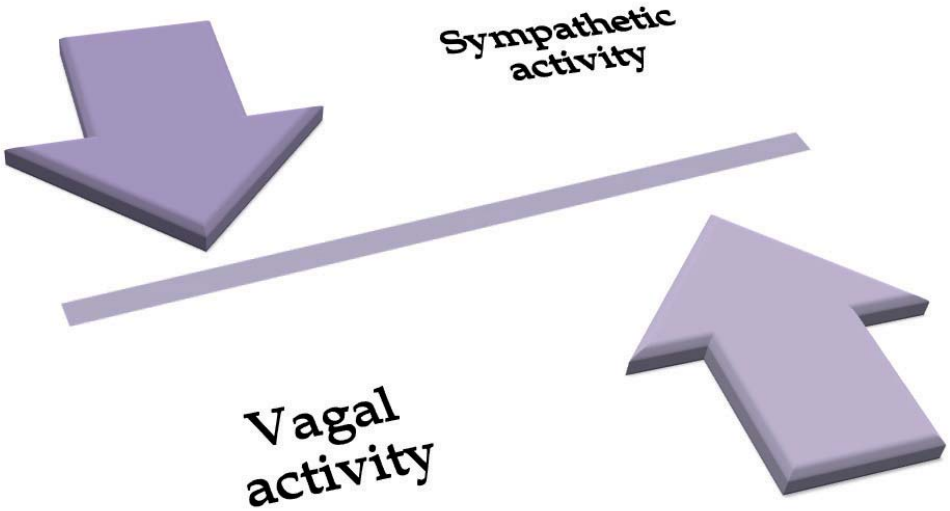
Esa Hynynen

Heart Rate Variability in Chronic and Acute Stress

With Special Reference to Nocturnal Sleep and Acute Challenges after Awakening



JYVÄSKYLÄN YLIOPISTO

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ABSTRACT

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Finnish summary
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The present study was designed to investigate the association between chronic and acute forms of physical and psychological stress and cardiac autonomic modulation in real-life situations. The results showed that chronic physical stress (overtraining) did not affect nocturnal autonomic modulation measured either by stress hormone secretion or heart rate (HR) and/or heart rate variability (HRV) indices. HRV measured immediately after awakening in an orthostatic test was diminished in the presence of chronic physical stress. Furthermore, overtrained athletes showed lower cognitive performance and HRV responses to a cognitive task and relaxation after the task than control athletes. In contrast to chronic physical stress, acute physical stress produced through endurance exercises was found to be related to a decrease in nocturnal HRV. In addition, nocturnal HRV responses to the endurance exercises had a dose-response relationship. Chronic psychological stress was found to be associated with lower HRV after awakening in an orthostatic test, but no difference was found in nocturnal HR, HRV and stress hormone secretions between low and high stress groups. Acute psychological stress was related to high HR response during a parachute jump and further, HR was found to be higher in novices than in experienced jumpers both in absolute units and in relation to age-predicted maximum HR. Despite of the high sympathetic activation during the acute stress, no association was found to any HR or HRV indices during night sleep and orthostatic tests after awakening. Slightly elevated adrenaline secretion was found during the night preceding the jump, but it may be explained by seasonal variation, and was therefore not related to the psychological stress. These results indicate that both chronic physical and chronic psychological stress can diminish HRV, especially during the awakening period in the morning. This phenomenon may be used to estimate the stress-induced withdrawal of parasympathetic autonomic modulation which may have negative effects on human health. Furthermore, nocturnal HRV may be used to estimate changes in autonomic modulation during the recovery phase from acute endurance exercises. These methods can be utilized in individual follow-ups of stress management or athletic training programs.

Keywords: cardiovascular autonomic modulation, stress, overtraining, awakening response, endurance training, stress hormones

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In Jyväskylä, December 21st, 2010
Esa Hynynen

ABBREVIATIONS

BMI	body mass index
CA	control athletes
CVRRI	coefficient of variation of RR intervals
ECG	electrocardiography
EXP	experienced parachute jumpers
HFP	high frequency power
HIGH	high stress group
HPLC	high-performance liquid chromatography
HR	heart rate
HR_{ave}	average heart rate
HR_{max}	maximal heart rate
HRV	heart rate variability
LFP	low frequency power
LF/HF	ratio of low frequency power to high frequency power
LOW	low stress group
NOV	novice parachute jumpers
OA	overtrained athletes
OTS	overtraining syndrome
PSS	perceived stress scale
RIA	radioimmunoassay
RMSSD	square root of the mean squared differences between successive RR intervals
RRI	RR interval
SDNN	standard deviation of RR intervals
STRESS	two-category stress level
Stroop	Stroop Color Word Test
TP	total power
VLFP	very low frequency power
VO_{2max}	maximal oxygen uptake

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

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

III Hynynen E, Konttinen N, and Rusko H (2009). Heart rate variability and stress hormones in novice and experienced parachutists anticipating a jump. Aviat Space Environ Med 80: 976-980.

IV Hynynen E, Vesterinen V, Rusko H, and Nummela A (2010). Effects of moderate and heavy endurance exercise on nocturnal HRV. Int J Sports Med 31: 428-432.

V Hynynen E, Konttinen N, Kinnunen U, Kyröläinen H, and Rusko H (2010). The incidence of stress symptoms and heart rate variability during sleep and orthostatic test. Eur J Appl Phys (in press).

CONTENTS

1 INTRODUCTION

Stress and its effects on humans have been studied intensively. For example, at the time of preparing this manuscript, PubMed gave over 194.000 hits on the keywords "*psychological stress*" or "*physiological stress*", starting from March 1914 (Karsner and Denis 1914). The word stress may have a negative connotation, but it may also be considered as a stimulus or challenge. Why has this area of research been so popular? Why scientists are so interested in the phenomenon called stress? Stansfield and Marmot (2002) gave a three-folded answer to these questions of which the two first reasons provide a foundation for this study: "Firstly, to understand how the psyche and soma interact to cause human disease. Secondly, an overall aim has been the disease prevention and health promotion. The third reason is the treatment." The word "disease" is used to describe the negative effects of stress on human health, as well as physiological and psychological stability.

French scientist Claude Bernard is acknowledged as the first author to recognize that humans, by maintaining internal stability (*milieu intérieur*), are able to withstand the challenges of the external environment (Bernard 1865). The work of Bernard was refined by Cannon in the early 1920's, as he described the actions of cells responding to perturbing stimuli in terms of dynamic equilibria, or "homeostasis" (Cannon 1914). Forty years later, Selye introduced "the general adaptation syndrome" (Selye 1956), where he investigated several internal and external stressors that may disturb the homeostatic balance. Among these stressors was, for example, strenuous exercise. The general adaptation syndrome is made up of three stages: 1) Alarm reaction; the reaction of an organism when it is suddenly exposed to a stressor to which it is not adapted. If the organism can survive, this initial reaction is followed by the adaptation, or the 'stage of resistance'. 2) Stage of resistance; the organism is fully adapted to the stressor and the symptoms disappear. However, sustained or frequent alarm reaction may deplete the adaptation or resistance resources of the organism. 3) State of exhaustion; occurs when the organisms adaptation resourses are depleted (Selye 1956). Recently, more interest has risen on the sustained or prolonged physiological activation leading to increased duration of the stress re-

sponse (Brosschot et al. 2005). This prolonged stress response may manifest itself in three forms: anticipatory responses to potential stressors, slow recovery from stressors, and/or recurrent activity related to past stressors (Brosschot et al. 2005).

In athletic training, which can be considered as a mainly physical stress model, the overload principle is widely used. The overload training principle is intended to disturb the homeostasis of the body through the physical stress of a training session (alarm reaction). During the subsequent recovery phase, the physical capacity is recovered, possibly to a higher level than before the training session (resistance or adaptation phase). This process needs coordination of various systems that are appropriate to counteract the threats to homeostasis. The autonomic nervous system, including both the parasympathetic and sympathetic pathways, is highly responsible for this regulation of homeostasis. The role of parasympathetic nervous system has been highly implicated in the mediation of homeostasis, and it has been suggested to be an index of homeostasis (Porges 1992). If the adaptability of an athlete is finite, and/or the recovery is inadequate, he or she can enter into an overtraining state. Overtraining has been suggested to exist in two main categories stemming from the autonomic nervous system: parasympathetic and sympathetic overtraining (Israel 1976). These two types of overtraining may represent different stages of the general adaptation syndrome (Kuipers and Keizer 1988). Parasympathetic overtraining is assumed to be typical among endurance and older athletes, while sympathetic overtraining is more typical among sprinters, power athletes and young athletes (Israel 1976). Recently, new models of overtraining have been developed and the main interest has been the relationship between stress and rest (for a recent review, see e.g. Kellmann 2010).

As a response to a challenge of a stressor, the rate of release of certain hormones is altered. In most cases one hormone will cause a release of another, which then causes the release of a third one. This sequence of events is referred as a hormonal axis. In stress research, two hormonal axes are often referred: sympathetic-adrenal-medullary axis and hypothalamic-pituitary-adrenocortical axis. Both these axis involve adrenal glands, the medulla in former and cortex in latter. The medulla is activated by the sympathetic branch of the autonomic nervous system and its products are adrenaline and noradrenaline, common catecholamines. The product of the cortex is a group of hormones known as the corticosteroids, and perhaps the most important of these is cortisol. In the beginning of stress research, investigations concentrated on responses of the endocrine system to different stressors, but the studies on usage of autonomic cardiac indices as an indicator of autonomic modulation have increased during late 20th century. The variability of heart rate is not a new finding, even though it has become a very popular method as recently as the last 20-30 years. Already in 1733, Hales (1733) reported increase in heart rate (HR) during inspiration and decrease during expiration. This phenomenon of beat-to-beat fluctuation of HR has been termed respiratory sinus arrhythmia. During recent decades, a variety of heart rate variability (HRV) methods have been developed and their ability

to evaluate the cardiac autonomic modulation has been proven in multiple situations, as well as under influence of different stressors (Task Force 1996).

Despite the merits of large previous literature of stress research, people still face overwhelming stress frequently. In competitive athletes, the incidence of overtraining has been reported to be even as high as 65 % (Morgan et al. 1987). Among working people, work-related stress disorders were found to be the second most common (after back pain) work-related health problem in the European Union affecting 22 % of workers from EU (27 nations) in 2005 (Milczarek et al. 2009). As the autonomic modulation is responsible for the stress reactions in the body, non-invasive measurements of autonomic modulation may be beneficial for both working people and competitive athletes. In the words of Selye (1983): *"…knowledge of stress can benefit everyone regardless of educational background or profession."* Cardiac parasympathetic modulation has actually been previously proposed as a method to assess both the stress response and the vulnerability to stress on an individual basis (Porges 1992, 1995). While the models of overtraining question the relationship of stress and recovery, similar effort-recovery modeling has been done in the work context. In the case of insufficient recovery, cumulative effects of stress may occur (Mejman & Mulder 1998). Based on their own experience and previous literature (Porges 1995, Meijman and Mulder 1998), Rusko et al. (2007) proposed a model where the effects of stress and recovery influence the resources of the autonomic nervous system. The model shows, what kind of autonomic resources are activated when responding to a random, moderate challenge. Under optimal conditions, the autonomic resources are fully recovered and mainly vagal resources are needed. Typically however, the resources are not fully recovered, but there is no risk of problems in case of disposition to stress. If the resources are low, the risk of problems in autonomic modulation increases, as the sympathetic activation is increased already during rest. In the case of chronic exhaustion the resources are very small, and mainly sympathetic. The purpose of this thesis is based on the theories of Porges (1992, 1995) and Meijman-Mulder (1998) as modified in this model by Rusko et al, which emphasizes the importance of the recovery for well-being (Figure 1). This thesis intends to investigate the usability of HRV measurements as a tool to estimate the effects of chronic and acute physical and psychological stress on cardiac autonomic modulation, in real life situations.

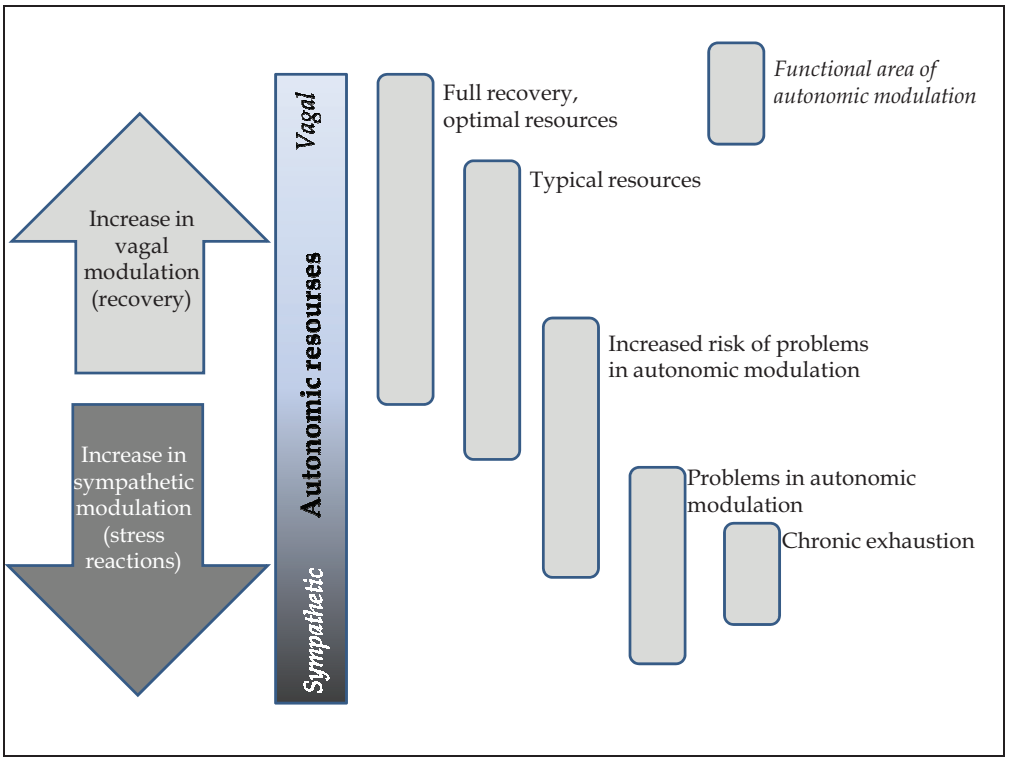

FIGURE 1 Model of the autonomic resources. Modified from Rusko et al. (2007).

2 REVIEW OF THE LITERATURE

The basic means by which the work of the heart is regulated are intrinsic cardiac regulation and control of the heart by autonomic nervous system. The intrinsic ability of the heart to adapt to changing volumes of inflowing blood is called Frank-Starling mechanism. This mechanism means that the greater the heart muscle is stretched during filling (venous return), the greater will be the force of contraction and the quantity of blood pumped into the aorta. In addition, the stretch of the right atrial wall directly increases the HR by 10 to 20 % (Guyton and Hall 1996). As this thesis concentrates on the autonomic modulation of heart, it is discussed in more detailed manner in the following section.

2.1 Autonomic modulation of the heart

The portion of the nervous system that regulates the bodily functions is called the autonomic nervous system. This portion generally functions without consciousness or volition. The autonomic nervous system consists of sympathetic nerves (thoraco-lumbar outflow) and parasympathetic nerves (cranial and sacral outflows). In general the effects of the two divisions are complementary, with activity of sympathetic nerves exciting the heart and parasympathetic nerves eliciting the opposite response. The autonomic nervous system ensures optimal function of the cardiovascular system during various activities, and on the other hand, mediates several of the manifestations of cardiac diseases (Hainsworth 1998). This section provides the background information on autonomic modulation of heart to help understanding the following sections of HRV.

2.1.1 Vagal modulation of the heart

The tenth cranial or vagus nerve is responsible for the vagal (parasympathetic) modulation of heart. The cell bodies lie in the dorsal motor nucleus of the vagus and in the nucleus ambiguous of the brain stem (Hainsworth 1998). The vagus nerves provide rich innervations to the sino-atrial node, atrio-ventricular con-

ducting pathways, the atrial myocardium (Hainsworth 1998) and possibly the ventricular myocardium (Johnson et al. 2004). Activity in the vagal nerves slows the HR by slowing the rate of spontaneous depolarizing of pacemaker cells. At rest, heart rate decreases from the intrinsic value of 110 – 120 bpm to 60 – 80 bpm by the predominance of vagal activity over sympathetic activity. The balance between vagal and sympathetic activity is responsible for adjusting the heart rate. HR values lower than the intrinsic values indicate vagal predominance while HR values over intrinsic values reflect sympathetic predominance (Hainsworth 1998). In most cases in this thesis, HR was lower than 100 bpm, and therefore, HR and HRV are considered to reflect cardiac autonomic modulation, even though vagal autonomic modulation might be more precise (see section 2.2 for more information on HRV analysis).

When vagal activity is high, cardiac slowing is profound and even a complete atrial asystole may occur. The chronotropic effect of vagal stimulation has a very short latency and vagal responses can influence heart rate on a beat-to-beat basis (Levy et al. 1970). These effects are graded according to the frequency of vagal nerve impulses, but the relationship between stimulus frequency and change in heart rate is not linear. As the effect of vagal impulses is to delay the subsequent pacemaker depolarization, the interval between depolarizations, R to R interval (RRI), has been found to be linearly related to vagal activity (Parker et al. 1984).

2.1.2 Sympathetic modulation of the heart

The sympathetic supply to the heart originates in the cells of the intermediolateral column of the spinal cord. All regions of the heart are innervated: pacemaker and conducting tissue, and atrial and ventricular myocardium. Increase in sympathetic activity increases heart rate by increasing the rate of depolarization of pacemaker cells. Whereas vagal activity can delay the very next heart beat, sympathetic responses are much slower. Maximal responses may not occur for as long as 20-30 seconds. Similarly to the vagal effects, RRI is more closely related to the frequency of sympathetic stimulus. High sympathetic drive is responsible for the high heart rates seen during maximal exercise, also increasing the force of contraction and shortening the duration of systole (Hainsworth 1998).

2.2 HRV as an indicator of cardiac autonomic modulation

The first documented observation of respiratory pattern from blood pressure and pulse was measured in a horse and was published almost three centuries ago by Hales (1733). Approximately 120 years later, Ludwig was able to observe the acceleration of pulse rates with inspiration and deceleration with exhalation in a dog. Several references to respiratory sinus arrhythmia have been made during the early 20th century, but clinical research interests in respiratory sinus

arrhythmia were rekindled during 1950´s and 1960´s by Hon and Wolf (Berntson et al. 1997). Hon treated HRV as a global index of fetal distress (Hon 1958, Hon and Lee 1963), and Wolf focused on the contribution of central nervous system factors to sudden cardiac death (Wolf 1967). As HRV reflects autonomic modulation of heart and the mechanisms underlying this modulation, interest on HRV has been increased during the last decades. One of the major research areas has been the association between HRV and cardiac health, because it has been found that high HRV may reflect better survival after myocardial infarction (Task Force 1996) and longevity (Zulfiqar et al. 2010). This section focuses on the physiological basis of HRV, describes typical HRV approaches, and effects of age and gender to HRV. Even though it is the variability of RRIs rather than variability of heart rate per se, that is studied, the term HRV has gained wide acceptance, and it is therefore used in this document.

2.2.1 Methods for analyzing HRV

HRV may be analyzed by a number of methods, but the most commonly used and highly validated methods are time domain and conventional frequency domain methods. The **time domain analysis** can be easily calculated with simple statistical methods. The simplest index is the standard deviation of the RRIs (or normal-to-normal intervals of the electrocardiogram) over the selected period (SDNN). This index includes RRI fluctuations at all frequencies and represents the overall variability of RRIs. The most commonly used index derived from the differences of RRIs is the square root of the mean squared differences of successive RRIs (RMSSD). RMSSD estimates high frequency variation in heart rate and is considered to be mainly vagally mediated (Task Force 1996, Hartikainen et al. 1998).

Frequency domain analysis, also named as power spectral density, decomposes the RRI data into its frequency components and quantifies them in their relative intensity, termed power. It provides information how overall HRV is distributed as a function of frequency. The most commonly used frequency domain methods are nonparametric Fast Fourier Transformation and parametric autoregressive modeling. The advantages of the nonparametric methods are the simplicity of the algorithm and high processing speed, while the advantages of parametric methods are smoother spectral components, easy post-processing with an automatic calculation of different components and an accurate estimation of power spectral density, even on small number of samples. In most instances, these two methods provide comparable results but also share the same major problem: assumption of a stationary signal. This means that steady state conditions can be studied and compared, but no transient changes in autonomic modulation are allowed during the analyzed period (Task Force 1996, Hartikainen et al. 1998).

In frequency domain methods, the overall variability of RRIs is represented by total power (TP). Three spectral components can be distinguished: high frequency (HF, 0.15-0.40 Hz), low frequency (LF, 0.04-0.15 Hz) and very low frequency (VLF, 0-0.03 Hz) components (Task Force 1996, Harti-

kainen et al. 1998). However, the physiological explanation of VLF component has not been well defined, even though it seems to vanish during vagal blockade, as discussed earlier (Uusitalo et al. 1996, Taylor et al. 1998, Martinmäki et al. 2006a). In addition, VLF represents very low frequency oscillations, and to assess these oscillations reliably, long recordings of approximately one hour are needed. To assess the HF and LF oscillations, recordings of 1 and 2 minutes are the shortest to provide reliable results, respectively. The magnitude of each frequency component is usually expressed in absolute power (integral of power spectral density curve within the corresponding frequency bounds). LF and HF can also be expressed in normalized units, which represent the relative value of each power component in proportion to TP minus VLF. Also, the ratio of LF to HF is often provided. However, normalized units and LF/HF should be quoted together with the absolute values, since they are not accurate measures; they only represent the relative power of the frequency components (Task Force 1996, Hartikainen et al. 1998). For example, in a recent pharmacological blockade study, it was shown that normalized units and LF/HF are not valid quantitative indices of autonomic modulation or sympathovagal balance (Martinmäki et al. 2006a).

2.2.2 Physiological basis of HRV

Respiratory sinus arrhythmia is strongly related to vagal modulation, since it is diminished during vagal blockade (Katona and Jih 1975, Akselrod et al. 1981, Uusitalo et al. 1996, Martinmäki et al. 2006a). Increases in the frequency of respiration and decreases in tidal volume are associated with decreases in respiratory sinus arrhythmia and vice versa, but not to the tonic level of vagal modulation (Hirsch and Bishop 1981, Eckberg 1983, Grossman and Kollai 1993). Intracardiac reflex and/or mechanical stretch of the sinoatrial node due to respiration may explain the small amplification of the respiratory sinus arrhythmia found in the absence of autonomic modulation (Bernardi et al. 1989, Saul et al. 1991, Taylor et al. 2001).

Spectral analysis of HRV has enhanced understanding of the autonomic modulation of heart. HRV at higher frequencies reflects vagal modulation, but some controversy exists in the interpretation of HRV at lower frequencies (Task Force 1996). Some investigators have suggested that HRV at lower frequencies, especially when expressed in normalized units, is of sympathetic origin (Pagani et al. 1986, Malliani et al. 1991), while most of the investigators have suggested that the parameter includes both sympathetic and vagal modulation (Akselrod et al. 1981, Saul et al. 1990). However, many recent studies have shown that regardless of the frequency of HRV, it is diminished after vagal blockade suggesting that lower frequencies also reflect mainly vagal modulation of the heart (Uusitalo et al. 1996, Taylor et al. 1998, Martinmäki et al. 2006a). It should be noted, that HRV has a circadian pattern with the highest values occurring during night sleep followed by a decrease after awakening (Furlan et al. 1990, Huikuri et al. 1990, Huikuri et al. 1992, Guo and Stein 2003). HRV has been mostly studied during daytime in rest or during different challenges, but it has also

been suggested, that nocturnal HRV may have some benefits: It can be free of external disturbance, and it can be recorded in real-life situation (Pichot et al. 2000). Furthermore, a recent study showed that nocturnal HR, but not daytime HR, was an independent predictor of survival after acute myocardial infarction (Carney et al. 2008).

2.2.3 Effects of age, gender and physical fitness on HRV

In healthy subjects, HRV decreases during adult life are associated with normal aging (De Meersman 1993, Umetani et al. 1998, Antelmi et al. 2004, Zhang 2007, Barantke et al. 2008, Zulfiqar et al. 2010). This decrement is related to decline in autonomic modulation in general, but also to a decrement in aerobic capacity (De Meersman 1993, Byrne et al. 1996, De Meersman and Stein 2007). Gender has been found to affect HRV, but gender differences in HRV may depend on age and particular indices. To conclude these findings: at age of younger than 40 years, women seem to have lower overall HRV than men (Ramaekers et al. 1998, Umetani et al. 1998, Barantke et al. 2008), but may have similar or higher HF than men (Ramaekers et al. 1998, Antelmi et al. 2004, Barantke et al. 2008). In addition to associations with age, gender and physical fitness, these studies show great inter-individual variation in HRV indices. It should also be noted that heritable factors may explain a substantial proportion of variation in heart rate (HR) and HRV (Singh et al. 1999).

2.3 Changes in HRV in relation to physical stress

According to the overload principle the physical stress of a training session has to be hard enough to disturb the homeostasis of the bodily functions and the recovery time has to be long enough to allow an increase in the determinants of performance, resulting in the training effect. If the disturbance is too small, no training effect will occur (Rusko 2003). This disturbance of homeostasis can be seen in autonomic modulation even hours after an exercise session has been stopped (Furlan et al. 1993, Hautala et al. 2001). Cumulative training effects on nocturnal HRV have been studied in endurance athletes and sedentary people during different training periods (Pichot et al. 2000, Iellamo et al. 2002, Pichot et al. 2002b, Baumert et al. 2006, Hynynen et al. 2007). Pichot et al. (2000) found progressive decreases of up to 40 % in nocturnal HRV during the hard training period, followed by rebound during the relative resting week in middle-distance runners during their usual training cycle. A similar trend was found in track and field as well as triathlon athletes during intensified physical training of two weeks followed by a few days rest (Baumert et al. 2006). Decrement in nocturnal HRV in international level cross-country skiers after an overreaching period has also been reported (Hynynen et al. 2007). Pichot et al. (2002b) also found decreased nocturnal HRV after intensified training period in sedentary people, even though the overload training period preceding the intensified

training period had increased the nocturnal HRV. If the physical stress is increased by increasing the amount of exercise while keeping the intensity low, reduced HR with no change in HRV have been reported (Hedelin et al. 2000). Not only physical exercise, but also occupational work can be physically stressful; A progressive decrease in nocturnal HRV was detected during a physically demanding 3-week work period (Pichot et al. 2002a). During the following rest week the nocturnal HRV indices of the workers recovered to their initial level. Recently, the physical work environment was also found to have effects on nocturnal HRV in office workers (Thayer et al. 2010).

If the recovery is not good enough however, overload training period can change into overreaching or overtraining. This means that after exceedingly demanding physical training, competitive performance is diminished for a period of time. The main difference between overreaching and overtraining is the time needed for recovery; after overreaching the restoration of performance capacity may take several days or several weeks but after overtraining, several months or even years may be needed (Fry et al. 1991, Uusitalo 2001, Meeusen et al. 2006). Findings of decreased nocturnal HRV after overreaching periods (Pichot et al. 2000, Pichot et al. 2002b, Baumert et al. 2006, Hynynen et al. 2007) suggest that it could be used as a method for detecting / avoiding the development of overtraining, but Bosquet et al. (2003) found no significant changes in nocturnal HR and HRV in overtrained endurance athletes. However, decrement of HRV in overtrained athletes has been reported in both supine and upright posture in tilt table tests (Mourot et al. 2004). Moreover, the changes in HRV indices by the change of posture have been found to be smaller in overtrained athletes when compared to control athletes (Mourot et al. 2004). In a longitudinal study of Uusitalo et al. (2000), it was found that the responses to overtraining were individual in overtrained female endurance athletes, but the HR changes in head-up tilt decreased as a result of heavy training.

2.4 Changes in HRV in relation to psychological stress

Stressful conditions and prolonged exposure to stress can manifest themselves into a number of emotional, cognitive, physiological and somatic symptoms (Melamed et al. 2006). There are a large number of investigations dealing with HRV during exposure to standardized psychological stressors such as mental arithmetics (Hohnloser and Klingenheben 1998), but whether autonomic modulation is also affected during real-life situations and under influence of real-life stressors, may be more significant to human health. After all, data from laboratory experiments are often limited by their intrinsic artificiality. Dishman et al. (2000) suggested that vagal modulation of heart appears to be sensitive to recent experiences of persistent emotional stress regardless of age, gender, respiration rate or cardiorespiratory fitness (Dishman et al. 2000). Also real-life stress of academic examination has been found to affect HRV during orthostatic test performed 30 to 60 min before the examination (Lucini et al. 2002). These two

studies however, used the normalized units of LFP and HFP, which are not the best variables to describe autonomic modulation accurately (Hartikainen et al. 1998, Martinmäki et al. 2006a).

In a recent study of teachers, no differences were found in HRV between work day and weekend day, but there was a tendency of increasing HRV during the holiday season (Ritvanen et al. 2004). In another study of low and high stress periods in school, it was found that younger teachers had decreased HR during low stress period when compared to the high stress period. The difference was found in both home and work situations, but not in the group of older teachers (Ritvanen et al. 2006). Elevated HR has been reported at work during stressful periods, but social support may have a buffering effect on this (Steptoe 2000). In a laboratory study of Hall et al. (2004) a standard speech task paradigm was used to elicit acute stress in the immediate pre-sleep period. The subjects in the stress group were told that they had to give an oral speech on awakening in the morning. During the following night's sleep, HRV was found to decrease under influence of an acute and/or anticipated psychological stressor (Hall et al. 2004). In addition, chronic work stress (high effort – low reward) has been associated to low HRV during work, leisure and sleep both during work days and weekends (Vrijkotte et al. 2000).

Furthermore, it has been suggested that the risk for cardiovascular disease increases if the stress-related cardiovascular activation is prolonged (Vrijkotte et al. 2000, Pieper and Brosschot 2005). Psychosocial stress symptoms during workday may not be harmful for the health, but if prolonged, they may lead to cardiovascular disease (Pieper and Brosschot 2005, Kivimäki et al. 2006). Actually, the negative association of stress with cardiovascular health received a dramatic confirmation in the INTERHEART study, where data was collected from 52 countries (Rosengren et al. 2004). In a relatively large sample of male workers, it was found that 24-hour HRV was reduced as perception of work stressors increased (Clays et al. 2010). The authors concluded this to support the idea that the autonomic nervous system may play a role in the link between work stress and cardiovascular disease (Clays et al. 2010). In addition, previous literature on HRV and cardiovascular health have shown that increased HR (Carney et al. 2008) and reduced levels of HRV have been found to be related to increased risk for cardiac events and increased mortality rate in patients with a previous myocardial infarction (Bigger et al. 1992, Tsuji et al. 1996, Huikuri et al. 1998).

2.5 Stress hormone responses to physical and psychological stress

Emergency function theory of the adrenal medulla, proposed by Cannon (1914) almost a century ago, suggested that the release of adrenaline increases the animal's struggling efficiency. This was further developed in mid 50's, when Selye

(1956) introduced the General Adaptation Syndrome, a general physiological reaction to a wide range of stressors. This reaction can be regarded as a defense reaction activating the hypothalamic-pituitary-adrenal axis. Later, enhanced cortisol secretion has been introduced to prevent the defense reactions from overshooting rather than enhancing defense reactions (Munck et al. 1984). Basically, it seems that the stress hormone response to a large variety of stressful stimuli is fundamentally the same. Probably the stress response is not caused by the stressor per se, but by the ability of the individual to deal with the stressor (Biondi and Picardi 1999).

Overtraining may be considered as a chronic, mainly physical stress. It has been proposed that two types of overtraining might exist: parasympathetic and sympathetic (Israel 1976). Physical stress induced by athletic training has been related to changes in stress hormone secretions, but the findings have been somewhat controversial. This may be due to many factors, as aerobic (Lehmann et al. 1991, Lehmann et al. 1992, Uusitalo et al. 1998) and resistance training (Häkkinen et al. 1988, Fry et al. 1998) stimulate different responses. Both hormone concentrations at rest (chronic adaptation) and during exercise (acute response) respond differentially to extensive aerobic or resistance training (Armstrong and VanHeest 2002). Nocturnal stress hormone concentrations (markers of basal autonomic tone) have been used to analyze the development of the overtraining state. In the final phase of this development, or in the parasympathetic type of overtraining, the basal urinary catecholamine excretion can be reduced by 50–70 % (Lehmann et al. 1991, Lehmann et al. 1992). The findings concerning nocturnal urine catecholamines have been however, controversial and the differences in stress hormone concentrations between a well-trained and an overtrained state are not clear (Lehmann et al. 1991, Lehmann et al. 1992, Uusitalo et al. 1998).

There are a large number of studies dealing with stress hormone responses to standardized psychological stressors in a laboratory setting. The conclusion of these studies are that catecholamines and cortisol levels rise due to variety of stress stimuli, like mental arithmetics, speech tasks and Stroop tests (Biondi and Picardi 1999). Also, different real-life stressors have been studied. The stress of academic examination, bereavement and parachute jumping for example, are related to significant changes in stress hormone levels (Biondi and Picardi 1999). Previous studies have suggested that exposure to psychological stress is associated with increased stress hormone response to awakening (Dodt et al. 1997, Pruessner et al. 1999, Wust et al. 2000, Lundberg and Hellström 2002, Maina et al. 2009, Vreeburg et al. 2010), while prolonged stress may decrease them (Pruessner et al. 1999, O'Connor et al. 2009).

3 AIMS OF THE STUDY

This study was designed to investigate the association of chronic and acute, as well as physical and psychological stress with autonomic modulation. The main interest was to examine the effects of chronic exposure to physical and psychological stress on autonomic modulation. As the severity of stress effects on human health is expected to grow if stress is prolonged, the autonomic modulation may be disturbed most in the stage of exhaustion (Figure 1), the final phase of the general adaptation syndrome (Selye 1956). If the adaptation energy is finite, it may be seen in basal autonomic modulation during night, but it has also been suggested that responses to a challenge may be the most sensitive to stress (Keselbrener and Akselrod 1998). Therefore, both resting measurements as well as physical and psychological challenges were used to provoke the autonomic stress reactions. HR, HRV and stress hormone secretions were used as indicators of the autonomic modulation.

The specific aims of the study were:

1) To study the association of chronic physical stress (overtraining) with HR, HRV and stress hormones at rest as well as responses of HR and HRV to light physical (an orthostatic test) and psychological (a cognitive test and relaxation) challenges. The main hypothesis was that the overtrained athletes would exhibit lower HRV and nocturnal urinary stress hormone concentrations, as well as lower HR and HRV responses to light physical and psychological challenges, when compared to the control athletes. (Original papers I and II)
2) To study the association of an acute moderate and acute heavy physical challenge with HR and HRV at rest. It was hypothesized that heavy endurance exercise would be related to a decrease of nocturnal HRV and an increase of HR in relation to a rest situation or a moderate endurance exercise. In addition, the observed changes in HRV would be related to the resting HRV. Moderate endurance exercise was not expected to be in association with changes in nocturnal HR and HRV. (Original paper IV)

3) To investigate the association of chronic psychological stress with HR, HRV and stress hormones at rest as well as responses of HR and HRV to light physical challenge after awakening. The main hypothesis was that workers with higher incidence of stress symptoms would exhibit lower HRV and nocturnal urinary stress hormone concentrations, as well as lower HR and HRV responses to light physical stress, when compared to the workers with low incidence of stress symptoms. (Original paper V)
4) To investigate the association of high acute psychological stress of a forthcoming parachute jump with HR, HRV and stress hormones during the preceding night and responses of HR and HRV to light physical challenge after awakening. The main hypothesis was that the forthcoming parachute jump would attenuate HRV and accentuate HR and stress hormone secretion, and these responses would be greater in novice than experienced jumpers. (Original paper III)

4 METHODS

4.1 Subjects

All the subjects (Table 1) were healthy volunteers free of hypertension or other systemic diseases. They were not taking any medication or drugs that would alter the activity of autonomic nervous system. The procedures were conducted according to the declaration of Helsinki and were approved by the Ethics Committee of the Central Finland Health Care District (I-III, V) or by the Ethics Committee of the University of Jyväskylä (IV). The subjects gave their written informed consent prior to participation, and they had a right to withdraw from the experiments at any time.

TABLE 1 Characteristics of the subjects.

	I and II		III		IV	V	
	OA	CA	NOV	EXP		LOW	HIGH
	n = 12	n = 12	n = 13	n = 7	n = 10	n = 40	n = 59
Gender	6 ♂, 6 ♀	6 ♂, 6 ♀	9 ♂, 4 ♀	6 ♂, 1 ♀	10 ♂	28, 12 ♀	31 ♂, 28 ♀
Age (yrs)	25±7	24±5	26±4	37±4[b]	37±5	37±9	43±10[c]
Height (cm)	174±10	172±7	176±10	179±8	177±7	175±11	170±7[c]
Body mass (kg)	71±13	64±8	73±13	81±10	76±6	78±13	74±14
BMI ($kg \cdot m^{-2}$)	23±2	22±2	23±3	25±2	24±2	25±3	25±4
PSS (scale 0-56)	25±11	15±6[a]	16±6	15±5		15±5	27±8[d]
VO_{2max} ($mL \cdot kg^{-1} \cdot min^{-1}$)	49±9	60±9[a]			52±5		

The values are mean±sd. ♂, males; ♀, females; OA, overtrained athletes; CA, control athletes; NOV, novice parachutists; EXP, experienced parachutists; LOW, low stress group; HIGH, high stress group; BMI, body mass index; PSS, perceived stress scale; VO_{2max}, maximal oxygen uptake. Significant difference at [a] $p < 0.05$ compared to OA, at [b] $p < 0.001$ compared to NOV, at [c] $p < 0.01$ and at [d] $p < 0.001$ compared to LOW.

The general health status of the subjects was assessed with a questionnaire (I-V), a standard medical examination including an interview (I-II) and a resting ECG (IV). The questionnaire prescreened for inherited propensity to cardiovascular diseases, autonomic nervous system abnormalities and contraindications to maximal exercise test (I-II and IV). The subjects were asked to refrain from alcohol ingestion and avoid caffeinated beverages during the day of the measurements and during the day before them. If they performed regularly physical activity, they were allowed a light exercise, but not a prolonged or strenuous one (except in study IV, where the measurements where done after endurance exercises). The incidence of stress symptoms during last month were screened with Perceived Stress Scale (Cohen et al. 1983) (I-III, V). Table 1 shows characteristics of the subjects.

4.1.1 Chronic physical stress (I, II)

Athletes with overtraining syndrome (OTS) served as chronic physical stress subjects. OTS was diagnosed by a medical doctor using the following criteria, an athlete 1) had suffered from an unexplained decrement in physical performance and fatigue even after a recovery time of at least 3 weeks, which was verified by carefully interviewing the athletes and in some cases also their coaches; 2) had been examined to be otherwise healthy with a clinical examination and several laboratory and physiological measurements; and 3) had increased their training volume and intensity progressively for up to 6 months before the overtraining symptoms and had continued training with the symptoms without sufficient recovery. The training logbooks of the previous months were checked to confirm the training history of the athletes. The chronic stress of the overtrained athletes was also verified with higher perceived stress when compared to the control athletes (Table 1). Half of the OA group, 6 athletes were able to compete again in 6-12 months after the diagnosis of OTS at the same level as they had been competing before developing the syndromes of overtraining. The other 6 athletes did not compete again in the following 24 months after the diagnosis of OTS. These observations support the diagnosis of OTS in all 12 overtrained athletes in this study. The athletes represented different sporting events, but most of them took part in endurance sports. Besides endurance athletes, two male athletes and one female athlete in OA were ice hockey players, and one female athlete in CA was a sprint runner.

4.1.2 Chronic psychological stress (V)

To examine the effects of chronic psychological stress, subjects with high incidence of stress symptoms were recruited. They were recruited from several workplaces, one rehabilitation centre and through a newspaper advertisement. Individuals were eligible to be selected for the study if they were between 20 and 60 years old, had a daytime job and were employed for more than 30 hours per week. The single-item measure of self-reported stress symptoms was used for the classification of the participants into different categories on self-reported

stress. This single-item stress-symptoms measure invented by Elo at al. (2003) has showed satisfactory content, criterion and construct validity for group-level analysis. In fact, they (Elo et al. 2003) have suggested that the longer scales used to measure psychological stress can be replaced by it in survey research. In line with these suggestions, positive correlations were found between this item and the Perceived Stress Scale ($r = 0.694$, $p < 0.001$) as well as the somatic symptoms scale ($r = 0.779$, $p < 0.001$). The following instruction was used for this item: "Distress results when the body over-reacts to events of life. When you are stressed, you may feel yourself restless, nervous, or anxious. You may also have problems with sleeping. Please, indicate how often you feel like this." Response choices were recorded on a 5-point scale from 1 to 5 accordingly: "I never feel like this", "I almost never feel like this", "I sometimes feel like this", "I fairly often feel like this" and "I very often feel like this". The chronic stress of the subjects of the high stress group was also verified with higher perceived stress when compared to the subjects of the low stress group (Table 1).

4.2 Procedure

Autonomic modulation was investigated during rest and during responses to physical and psychological challenges. Resting measurements were done at home and included the RRI recordings during night sleep, during supine rest immediately after awakening and nocturnal urine collections. The RRI recordings were done also during acute physical and psychological challenges. Table 2 shows the procedures included in the different original studies (I-V).

TABLE 2 Study procedures included in the original papers.

STUDY	I	II	III	IV	V
Resting measurements	X		X	X *	X
Light physical challenge		X	X		X
Moderate and heavy physical challenge				X	
Light psychological challenge		X			
Heavy psychological challenge			X		

* Only nocturnal HR and HRV analysis.

4.2.1 Data collection

RRI collections were done with an RR-Recorder (Polar Electro Ltd., Kempele, Finland) (I-III, V) and Memory Belt (Suunto Ltd., Vantaa, Finland) (IV) with a sampling frequency of 1000 Hz (Ruha et al. 1997, Loimaala et al. 1999, Weippert

et al. 2010). As low sampling rate may produce inaccuracy of the R-wave detection, optimal sampling rate range is 250 to 500 Hz or higher to provide reliable data (Task Force 1996). Therefore, with the digital devices used in this study, the high accuracy of the R-wave detection of 1 ms enables highly reliable analysis of HRV. The RRI collections were done at home and the recording device was brought to the laboratory the next morning. Nocturnal urine samples (I, III, V) were collected between 7 p.m. and 7 a.m., and brought to the laboratory at the same time with the RRI collections. After the subject's arrival in the laboratory, the urine volume was determined and a sample for cortisol analysis was stored at -80°C. Another urine sample for adrenaline and noradrenaline analysis was acidified with 6n-hydrochlorid acid to a pH of 3, before storing it at -80°C.

4.2.2 Light acute physical challenge (II-III, V)

An active orthostatic test served as a light physical challenge (II-III, V). Immediately after collecting the resting RRI (5-min period) after awakening, subjects stood up and kept standing for a 3-min period. The subjects pressed the "marker" button of the recording device before starting the orthostatic test and at the time of the change of posture to identify the correct moments in the RRI collection.

4.2.3 Moderate and heavy acute physical challenge (IV)

Nocturnal RRI collections were done after a rest day (no exercise session during the rest day), after a moderate endurance exercise session (on average 52 ± 26 min, average heart rate 133 ± 9 bpm equaling 72 % of HR_{max}) and after a marathon run (on average 217 ± 28 min, average heart rate 156 ± 5 bpm equaling 85 % of HR_{max}). All the exercise sessions were performed during afternoon, but no exact standardization was used. The moderate endurance exercise session was perceived as light and easy (3 ± 1 on scale 0-10) and marathon as very hard (8 ± 2 on scale 0-10). The three test conditions (rest, moderate exercise, marathon) were not randomized, but the nocturnal HRV were measured tens of times during the 33 week training period. These three conditions presented here were measured during the last two weeks of the project, and therefore, all the subjects were familiar with the measurements.

4.2.4 Light acute psychological challenge (II)

A Stroop Color Word Test (Stroop) served as a light acute psychological challenge. The athletes arrived again at the laboratory at approximately 8 a.m. The athletes first rested in a sitting position for at least 15-min to guarantee stationary physiological values. To identify the correct moments in the RRI collection during the following events, the marker button was pressed and a stop watch was started at the same time. The times of the following events were then manually recorded to help later analysis. Then the athletes performed the Stroop, succeeded by a progressive relaxation period guided by a psychologist from a

tape recorder while sitting in a quiet laboratory room. In the Stroop, the subject had to respond "match" or "non-match" (by pressing a button) to a color word shown on the computer screen in the same (match) or in another (non-match) color. The Stroop began with a familiarization period, after which the test with intermittently decreasing response times was done twice. The initial maximal response time was 2000 ms per word and it decreased by 100 ms after every 18 words until the shortest response time, which was 600 ms per word. The threshold for increasing mistakes was defined as the response time where the mistakes exceeded the individual level during the previous longer response time tasks. At this threshold 5-10 % of all responses were mistakes. Following the intermittently decreasing response time test, three 6-min constant velocity Stroop-tasks were carried out as follows: "slow speed" at 2000 ms for everyone, "moderate speed" at threshold + 300 ms, and "fast speed" at the individual threshold velocity. There was only a little variation in the response times at the threshold, which were 900 ± 100 ms in OA and 900 ± 50 ms in CA.

4.2.5 Heavy acute psychological challenge (III)

A parachute jump served as a heavy acute psychological challenge. Resting measurements and light acute physical challenge measurements were done in the night and morning preceding the jump. The measurements were repeated 1-2 months later when the subjects had no known stress factors affecting the autonomic modulation. The subjects arrived at the parachuters' clubhouse close to the local airport 3-5 h after waking up and at least 1.5 h before boarding for the jump. Approximately 1 h before the jump the subjects sat down and filled in a multisection questionnaire. The marker button was again pressed to synchronize the time of the RR-recorder to the "official time". The subjects boarded the plane and after reaching altitude of 1000 m performed the parachute jump over the drop zone. All the parachute jumps in this study were so called "hop and pop" jumps, which mean that the parachute was opened immediately after exiting the plane. The novices used automated opening of the parachute and the experienced jumpers opened their parachute by pulling the handgrip. At all times there was a radio contact between the plane and the researcher on the landing zone. The official times of exit and landing were marked manually in the data table by the researcher based on the radio contact and visual observation. The RRI collection was stopped and the recording device removed a few minutes after the landing.

4.3 HRV measurements and analysis

For nocturnal HR and HRV analysis, a continuous 4-h period from 0-4 a.m. (I-III, V) or starting from half an hour after going to bed for sleep (IV) were selected. HR and HRV during the light physical challenge immediately after awakening were analyzed as a continuous 4-min period, between 0:30 and 4:30 of the 5-min

supine rest period and the last 2-min of the 3-min standing period. The first and the last 30-s periods of supine rest were excluded because of many artifacts related to "starting and finishing" the rest period. The first standing minute was excluded because of biphasic heart rate response just after assuming the upright position. During the light psychological challenge, HR and HRV analysis were done as a continuous 6-min period of each task. As recordings of approximately 1 min are needed to assess the HFP and 2 min are needed for the LFP assessment, the analyzed periods in this study provided valid results.

Care was taken to remove abnormal RRI caused by movement artifacts or any other artifacts of unknown origin. Both automated artifact detection by the software and visual inspection and control were used to correct the data. In addition to average HR, the following HRV parameters were analyzed: standard deviation of RRI (SDNN), root-mean-square of differences between adjacent RRI (RMSSD), as well as autoregressive calculations including: total power (TP), low frequency power (LFP; 0.04 – 0.15 Hz), high frequency power (HFP; 0.15 – 0.40 Hz) and low to high frequency ratio (LF/HF). To minimize the heart rate dependency of HRV, and to reduce the influence of inter individual differences in SDNN, the coefficients of variation (CVRRI = 100 · SDNN / average RRI) were calculated. HRV parameters are subject to considerable day-to-day variations with intraclass correlation coefficients between 0.76-0.86 for spontaneous breathing and absolute HRV indices (Pinna et al. 2007). Random error however, represents a limited part of the between-subject variability (Pinna et al. 2007). Our own unpublished data of 4-hour nocturnal HRV analysis during two consecutive nights after similar training days in 23 recreational endurance runners showed higher reproducibility in intraclass correlation coefficients (0.80 – 0.93) and intra-individual coefficients of variation (4.2 – 6.1 %).

4.4 Maximal oxygen uptake

Maximal oxygen uptake (VO_{2max}) of each subject (study IV) was assessed by a graded maximal treadmill test. This test consisted of an initial speed of 8 km· h^{-1} (gradient 0.5°) followed by increments of 1 km· h^{-1} every three minutes until exhaustion. Breath-by-breath data of oxygen uptake (VO_2) and carbon dioxide production (VCO_2) (Oxycon Mobile, Viasys Healthcare GmbH, Hoechberg, Germany) as well as RRIs (Suunto t6 heart rate monitor, Suunto Oy, Vantaa, Finland) were collected continuously during the test. The highest 60-s VO_2 value was considered as VO_{2max}, and the highest 10-s HR value as HR_{max}.

4.5 Urinary stress hormones

Nocturnal urinary stress hormone samples (cortisol, adrenaline, noradrenaline) were analyzed by radioimmunoassay and liquid chromatography (RIA and

HPLC; Yhtyneet Laboratoriot, Helsinki, Finland). Due to different lengths in collection times (from 10 to 13 hrs), the actual concentration of hormones in the urine sample was first multiplied by the volume of the whole urine collection, then divided by the collection time in hours, and finally multiplied by 12 to represent a 12-h collection for everyone. Intra-assay CV of the adrenaline was 2.3 % and of the noradrenaline 4.2 %, and the inter-assay CVs were 9.1 % and 11.4 %, respectively.

4.6 Statistical analysis

Statistical analyses were performed with SPSS for Windows statistical software (SPSS, Inc., Chicago, ILL, USA). Values are expressed as mean ± sd. Prior to the statistical analyses of the cardiac indices, log-transformation was used with variables that were not normally distributed. However, to help the comparison of the results, absolute values are provided in the results. Probability level of $p \leq 0.05$ was applied as an indicator of statistically significant results.

In the overtraining studies (I-II), Student's *t*-test for nonpaired samples was used for statistical testing. To compare the responses to awakening, 2 × 2 ANOVA for repeated measurements, with *t*-test as the post hoc test were also used. To compare HRV responses to standing up and the Stroop, ANOVA for repeated measurements, with least significant difference (LSD) as the post hoc test was also used. For bivariate correlation analysis, Pearson's correlation coefficients were calculated.

In the parachuting study (III), Student's *t*-test for paired samples was used to analyze the differences between the jump night and the control night. To analyze the differences between the groups (novice vs. experienced) and time (jump vs. control), 2 × 2 ANOVA with Bonferroni as a post-hoc test was used.

In the marathon study (IV), ANOVA for repeated measurements was used for statistical testing. Pearson's correlation coefficient was used to analyze the connection between nocturnal HRV after rest day, changes in nocturnal HRV after moderate and heavy exercises and aerobic power and performance.

In the study of incidence of stress symptoms (V), a two-category stress level variable (STRESS) was constructed on the basis of the self-reported stress by combining the response choices 1 and 2 (never and almost never) as well as 3, 4 and 5 (sometimes, fairly often and very often). The resulting categories were labeled as low (n = 40), and high (n = 59). Background differences between the two stress groups were analyzed with Student's *t*-test for independent samples. Differences between the two stress groups on cardiac indices and stress hormone secretion were analyzed for significance with MANOVA. The Wilks's test was used as the multivariate test. The design was 2 × 2 × 2; the GENDER (male vs. female), AGE (20–40 years vs. 41–60 years), and STRESS (low vs. high) served as independent variables. To examine changes in HR and HRV indices over time (changes from night to after awakening), ANOVA for repeated measures was used.

5 RESULTS

5.1 HR, HRV and stress hormones in association with chronic physical stress (I-II)

5.1.1 Nocturnal HR, HRV and stress hormone secretion

There were no differences found between overtrained and control athletes in nocturnal HR, HRV and urinary stress hormone secretion (Table 3).

TABLE 3 Nocturnal HR, HRV and urinary stress hormones in overtrained and control athletes.

	OA	CA
HR_{ave} (bpm)	54±6	52±6
SDNN (ms)	132±48	145±39
RMSSD (ms)	77±36	98±51
TP (ms^2)	14781±14070	18491±14956
LFP (ms^2)	2820±2393	4236±3913
HFP (ms^2)	2452±1898	3538±3338
LF/HF	1.30±0.51	1.62±1.42
CVRRI (%)	11.8±3.3	11.9±1.8
Adrenaline (µmol)	0.010±0.008	0.011±0.005
Noradrenaline (µmol)	0.076±0.030	0.080±0.030
Cortisol (nmol)	75.8±29.3	68.8±28.6

Values are in mean±sd. OA, overtrained athletes; CA, control athletes; HR_{ave}, average HR; SDNN, standard deviation of RRI; RMSSD, square root of the mean of the sum of the squares of differences between adjacent RRI; TP, total power; LFP, low-frequency power; HFP, high-frequency power; LF/HF, low- to high frequency ratio; CVRRI, coefficient of variation of RRI.

5.1.2 HR and HRV after awakening

Lower SDNN, LFP and CVRRI were found during supine rest after awakening in OA than in CA (Table 4). As a response to awakening (from night sleep to supine rest after awakening), CVRRI decreased more in OA than in CA (Figure 2).

TABLE 4 Supine HR and HRV after awakening in overtrained and control athletes.

	OA	CA
HR_{ave} (bpm)	56±8	52±6
SDNN (ms)	84±31	116±41 [a]
RMSSD (ms)	76±39	98±57
TP (ms^2)	4857±4598	8966±7968
LFP (ms^2)	2153±2232	4286±2904 [a]
HFP (ms^2)	2704±2607	4680±6607
LF/HF	1.13±0.93	2.09±2.96
CVRRI (%)	7.7±2.5	10.0±2.5 [a]

Values are in mean±sd. OA, overtrained athletes; CA, control athletes; HR_{ave}, average HR; SDNN, standard deviation of RRI; RMSSD, square root of the mean of the sum of the squares of differences between adjacent RRI; TP, total power; LFP, low-frequency power; HFP, high-frequency power; LF/HF, low- to high frequency ratio; CVRRI, coefficient of variation of RRI. Significant difference at [a] $p < 0.05$ compared to OA.

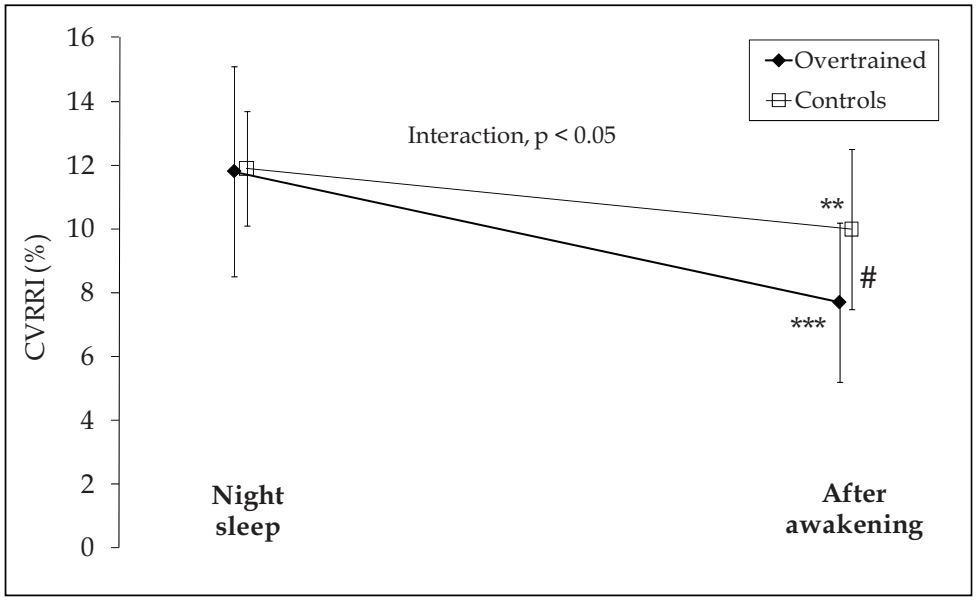

FIGURE 2 Change in the coefficient of variation of RR interval (CVRRI) from night sleep to after awakening. ***, ** indicate significant differences between night sleep and after awakening ($p < 0.001$, $p < 0.01$, respectively). # indicates a significant difference between the groups ($p < 0.05$).

5.1.3 HR and HRV responses to light physical challenge (an orthostatic test)

There was no difference between the groups in average HR during standing. LFP during standing was lower in overtrained athletes (Table 5), but no other differences between the groups in HRV were found. No differences between the groups were found in the RRI or HRV responses from supine rest to standing up.

TABLE 5 HR and HRV during standing in overtrained and control athletes.

	OA	CA
HR_{ave} (bpm)	84±12	82±12
SDNN (ms)	63±36	67±15
RMSSD (ms)	22±12	27±11
TP (ms^2)	1532±1095	2454±1104
LFP (ms^2)	1322±955	2262±1029 [a]
HFP (ms^2)	210±296	192±225
LF/HF	43.61±52.44	21.96±17.56
CVRRI (%)	8.4±4.4	9.0±1.6

Values are in mean±sd. OA, overtrained athletes; CA, control athletes; HR_{ave}, average HR; SDNN, standard deviation of RRI; RMSSD, square root of the mean of the sum of the squares of differences between adjacent RRI; TP, total power; LFP, low-frequency power; HFP, high-frequency power; LF/HF, low- to high frequency ratio; CVRRI, coefficient of variation of RRI. Significant difference at [a] $p < 0.05$ compared to OA.

5.1.4 HR, HRV and cognitive performance during light psychological challenge (the Stroop and relaxation)

In the Stroop, cognitive performance of OA (relative mistakes of 1.0 ± 0.8 %) was similar to CA (0.7 ± 1.0 %) in the slow speed. After increasing the speed of the Stroop, OA made more mistakes than CA during the moderate (2.9 ± 1.7 % vs. 1.7 ± 1.1 %, $p = 0.046$) and the fast speeds (9.7 ± 6.5 % vs. 5.4 ± 3.0 %, $p = 0.045$). During the Stroop, no differences between the groups were found in any HR or absolute HRV parameters and the changes in HR during the Stroop were insignificant. However, RMSSD, TP, LFP and HFP decreased significantly in CA from slow speed to fast speed while no changes were found in OA.

5.1.5 Relative HR and HRV changes from supine rest to light physical and psychological challenges

The relative HR changed from supine rest to standing, to the Stroop and to relaxation similarly in both groups (Figure 3a). During the fast speed, OA had higher relative TP than CA (50 ± 43 vs. 19 ± 14 %, $p = 0.028$, respectively) (Figure 3b). Compared to CA, OA also had higher relative HFP (Figure 3d) and tended to have higher relative LFP (Figure 3c) during the fast speed (38.5 ± 9.4 % vs. 13.5 ± 2.3 %, $p = 0.035$ and 74.1 ± 20.6 % vs. 30.7 ± 8.8 %, $p = 0.077$, respectively). In addition, significant group × task interaction from standing to fast

speed task in the Stroop was found in relative TP, and the decrease in relative TP and LFP from standing to fast speed in CA was significant (Figures 3b and 3c). Increase in relative (from 50 ± 43 to 86 ± 67 % vs. from 19 ± 14 to 96 ± 101 %, respectively, p = 0.020) (Figure 3b) and absolute (from 1831 ± 2508 to 3642 ± 3779 ms^2 vs. from 1150 ± 751 to 4992 ± 3287ms^2, respectively, p = 0.039) TP from the fast speed to the relaxation were smaller in OA than in CA. Also LFP alone (Figure 3c) increased less from the fast speed in the Stroop to relaxation in OA than in CA (p = 0.018). Similarly, RMSSD increased significantly in CA from the Stroop to relaxation while no changes were found in OA, but SDNN increased significantly from the Stroop to relaxation in both groups. In all subjects, lower change in absolute TP from the fast speed to the relaxation correlated with higher percentage of mistakes during the fast speed (r = – 0.588, p = 0.003) in the Stroop.

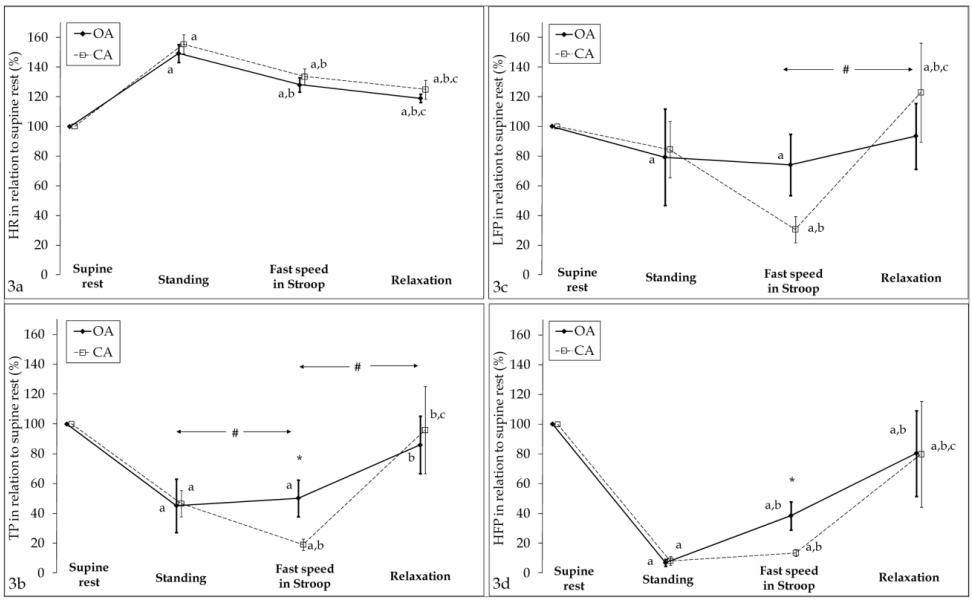

FIGURE 3 Average HR (3a), Total Power (TP) (3b), Low Frequency Power (LFP) (3c), and High Frequency Power (HFP) (3d) during standing in the orthostatic test in the morning, during the fast speed in the Stroop, and in the relaxation immediately after the Stroop in relation to corresponding values during supine rest (mean ± SE). See absolute resting values in Table 3. # indicates statistically significant interaction, group × time, p < 0.05; * = different from controls, p < 0.05; a = different from supine rest, p < 0.05; b = different from standing, p < 0.05; c = different from the Stroop, p < 0.05.

5.2 Nocturnal HR and HRV in association with acute physical stress of endurance exercises (IV)

5.2.1 Nocturnal HR and HRV after moderate and heavy endurance exercise

Compared to control night, nocturnal HR was higher after the moderate endurance exercise session and after the marathon ($p < 0.001$). Most HRV indices were decreased after the moderate endurance exercise session and after the marathon. Most HRV indices were also lower after the marathon than after the moderate endurance exercise session (Table 6). Similar results were found also in relative nocturnal HR and HRV (Figure 4).

TABLE 6 Nocturnal HR and HRV after the rest day, the moderate endurance exercise and the marathon.

	Rest	Moderate	Marathon
HR_{ave} (bpm)	47±2	50±3^{aaa}	61±6^{aaa,bbb}
SDNN (ms)	142±16	126±15^{aa}	91±19^{aaa,bbb}
RMSSD (ms)	61±19	53±19^{aa}	34±18^{aaa,bbb}
TP (ms^2)	1798±543	1725±780	1031±667^{aa,bb}
LFP (ms^2)	1214±920	952±754^{aa}	445±612^{aaa,bbb}
HFP (ms^2)	3011±1230	2677±1408^{a}	1476±1255^{aaa,bbb}
LF/HF	2.56±2.38	3.17±3.51	3.75±2.57
CVRRI (%)	11.0±1.0	10.6±1.1	9.2±1.7^{aa,b}

Values are in mean±sd. HR_{ave}, average HR; SDNN, standard deviation of RRI; RMSSD, square root of the mean of the sum of the squares of differences between adjacent RRI; TP, total power; LFP, low-frequency power; HFP, high-frequency power; LF/HF, low- to high frequency ratio; CVRRI, coefficient of variation of RRI. Significant differences at ^{aaa} $p < 0.001$, ^{aa} $p < 0.01$, ^{a} $p < 0.05$ compared to Rest, and at ^{bbb} $p < 0.001$, ^{bb} $p < 0.01$, ^{b} $p < 0.05$ compared to Moderate.

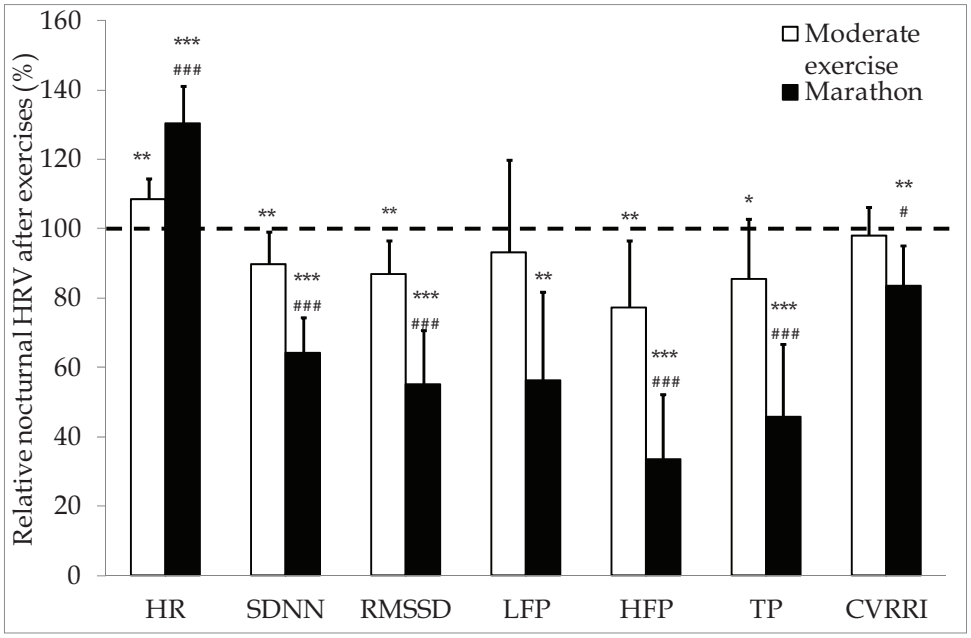

FIGURE 4 The relative changes in nocturnal HR and HRV after the moderate exercise and the marathon. 100 % line corresponds to the respective nocturnal values after the rest day. HR, average heart rate; SDNN, standard deviation of RRI; RMSSD, square root of the mean of the sum of the squares of differences between adjacent RRI; TP, total power; LFP, low-frequency power; HFP, high-frequency power (mean±sd). *, **, *** Statistically significant differences from rest, $p < 0.05$ $p < 0.01$, $p < 0.001$, respectively, and #, ### Statistically significant differences from moderate training, $p < 0.05$, $p < 0.001$, respectively.

5.3 HR, HRV and stress hormones in association with chronic psychological stress (V)

5.3.1 Nocturnal HR, HRV and stress hormone secretion in low and high STRESS groups

No differences in nocturnal HR or HRV between the STRESS groups were observed (Table 7). Neither was there any difference in nocturnal adrenaline, noradrenaline or cortisol secretions between the STRESS groups (Table 7). However, both gender and age had main effects on nocturnal HR and HRV, and gender × age interaction effect for HRV was also significant. Women had higher nocturnal HR and lower nocturnal HRV than men and the same differences were found between older and younger groups, respectively.

TABLE 7 Nocturnal HR, HRV and urinary stress hormones in the low and high STRESS groups.

	Low STRESS N = 40	High STRESS N = 59
HRave (ms)	57±10	61±7
SDNN (ms)	119±40	101±33
RMSSD (ms)	65±34	48±29
TP (ms^2)	12178±7471	8696±6881
LFP (ms^2)	2465±1725	1824±1532
HFP (ms^2)	1813±1911	1398±1726
LF/HF (ratio)	2.47±2.92	2.83±1.73
CVRRI (%)	11.0±3.1	10.1±2.8
Adrenaline (µmol)	0.009±0.005	0.009±0.006
Noradrenaline (µmol)	0.097±0.045	0.096±0.039
Cortisol (nmol)	80.4±45.1	93.9±49.2

Values are in mean±sd. HR_{ave}, average heart rate; SDNN, standard deviation of RRI; RMSSD, square root of the mean of the sum of the squares of differences between adjacent RRI; TP, total power; LFP, low-frequency power; HFP, high-frequency power; LF/HF, low- to high frequency ratio; CVRRI, coefficient of variation of RRI.

5.3.2 HR and HRV during light physical challenge in the STRESS groups

The low STRESS group had higher HRV than the high STRESS group during both supine rest (Table 8) and standing (Table 9). Both age ($p = 0.005$) and gender ($p < 0.001$) had significant main effect on HR and HRV during both supine rest and standing, but none of the interactions reached statistical significance. In other words, the relationships between STRESS and HR and HRV indices were not dependent on age or gender.

TABLE 8 Supine HR and HRV after awakening (p-values are significances in univariate analysis of variance, STRESS as an independent variable).

	Low STRESS N = 40	High STRESS N = 59	p –values
HR (bpm)	58±9	63±9	0.066
SDNN (ms)	86±40	62±29	0.017
RMSSD (ms)	59±32	39±25	0.019
TP (ms^2)	4813±5271	2281±2433	0.016
LFP (ms^2)	3006±3038	1373±1429	0.013
HFP (ms^2)	1807±2402	908±1190	0.065
CVRRI (%)	8.1±3.3	6.3±2.5	0.050

Values are in mean±sd. HR_{ave}, average heart rate; SDNN, standard deviation of RRI; RMSSD, square root of the mean of the sum of the squares of differences between adjacent RRI; TP, total power; LFP, low-frequency power; HFP, high-frequency power; LF/HF, low- to high frequency ratio; CVRRI, coefficient of variation of RRI.

TABLE 9 HR and HRV during standing in the orthostatic test. (p-values are significances in univariate analysis of variance, STRESS as an independent variable.)

	Low STRESS N = 40	High STRESS N = 59	p –values
HR (bpm)	83±14	88±13	0.132
SDNN (ms)	64±25	42±19	< 0.001
RMSSD (ms)	25±17	14±9	0.001
TP (ms^2)	2030±2155	855±1013	0.005
LFP (ms^2)	1846±1895	797±954	0.007
HFP (ms^2)	236±573	58±91	0.017
CVRRI (%)	8.6±3.1	6.0±2.6	< 0.001

Values are in mean±sd. HR_{ave}, average HR; SDNN, standard deviation of RRI; RMSSD, square root of the mean of the sum of the squares of differences between adjacent RRI; TP, total power; LFP, low-frequency power; HFP, high-frequency power; LF/HF, low- to high frequency ratio; CVRRI, coefficient of variation of RRI.

ANOVA for repeated measures showed that group × time interaction was significant for CVRRI between night and supine rest (p = 0.050) and between night and standing (p = 0.006). Thus, as can be seen in Figure 5, higher STRESS was associated with a greater decrease of HRV from night sleep to the orthostatic test.

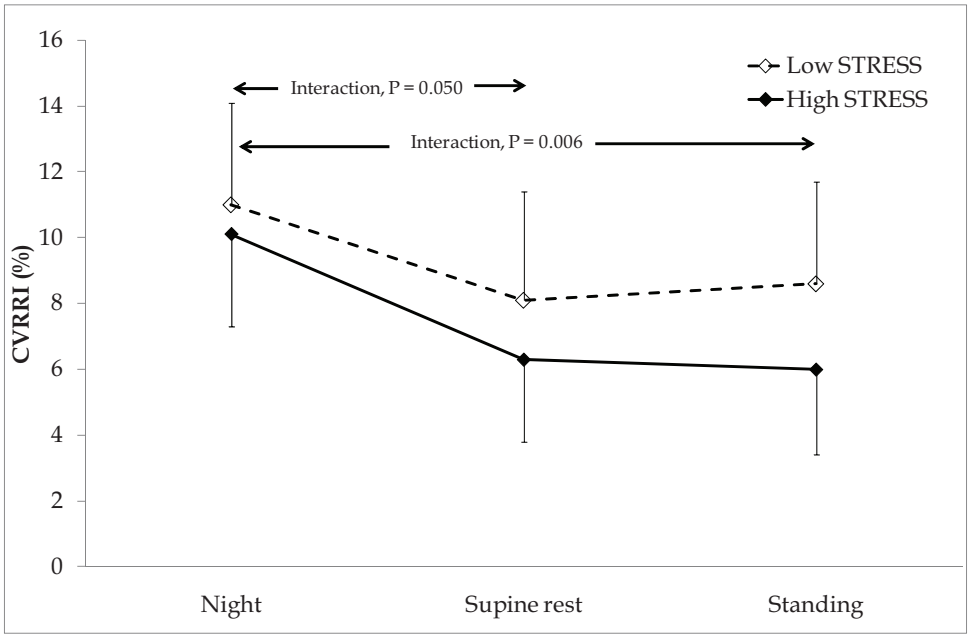

FIGURE 5 Changes in coefficient of variation of RRI (CVRRI) from night rest to orthostatic test in the morning.

5.4 Autonomic modulation in association with acute psychological stress (III)

5.4.1 HR, HRV and stress hormone secretion in parachute jumpers

Heart rate peaked a few seconds after exit from the aircraft (177 ± 18 bpm which equals to 91 ± 9 % of the age-predicted maximum HR) in all subjects (N = 20). There were no differences in HR or any HRV indices between the night before jump and the control night (Table 10). Neither were there any differences in any HRV indices or reactions during the light physical challenge between the jump and control mornings (Tables 11 and 12). Nocturnal adrenaline secretion was found to be slightly higher during the jump night than during the control night, but no differences were found in noradrenaline or cortisol levels (Table 10).

TABLE 10 Nocturnal HR, HRV and urinary stress hormones in jump night and control night.

	Jump night	Control night
HR_{ave} (bpm)	59±9	59±9
SDNN (ms)	110±33	115±38
RMSSD (ms)	65±29	67±37
TP (ms^2)	10056±4061	11786±6956
LFP (ms^2)	2135±1103	2226±1279
HFP (ms^2)	1841±1374	1909±2049
LF/HF	2.19±1.99	1.88±1.72
CVRRI (%)	10.4±2.6	11.0±2.4
Adrenaline (µmol)	0.013±0.007	0.008±0.004*
Noradrenaline (µmol)	0.080±0.030	0.081±0.037
Cortisol (nmol)	92.6±40.0	87.9±44.6

Values are in mean±sd. HR_{ave}, average HR; SDNN, standard deviation of RRI; RMSSD, square root of the mean of the sum of the squares of differences between adjacent RRI; TP, total power; LFP, low-frequency power; HFP, high-frequency power; LF/HF, low- to high frequency ratio; CVRRI, coefficient of variation of RRI. * = Statistically significant difference between the nights, $p<0.05$.

TABLE 11 Supine HR and HRV after awakening in the morning preceding the parachute jump and in the control morning.

	Jump morning	Control morning
HR (bpm)	61±11	61±10
SDNN (ms)	90±37	88±37
RMSSD (ms)	67±40	65±43
TP (ms^2)	5555±4803	4595±4828
LFP (ms^2)	3259±2358	2136±1515
HFP (ms^2)	2295±2652	2459±4072
CVRRI (%)	8.8±3.3	8.6±3.1

Values are in mean±sd. HR_{ave}, average heart rate; SDNN, standard deviation of RRI; RMSSD, square root of the mean of the sum of the squares of differences between adjacent RRI; TP, total power; LFP, low-frequency power; HFP, high-frequency power; LF/HF, low- to high frequency ratio; CVRRI, coefficient of variation of RRI.

TABLE 12 HR and HRV during standing in the orthostatic test in the morning preceding the parachute jump and in the control morning.

	Jump morning	Control morning
HR (bpm)	93±13	88±13
SDNN (ms)	62±27	42±19
RMSSD (ms)	21±10	14±9
TP (ms^2)	2067±2021	2163±2396
LFP (ms^2)	1984±1954	2054±2289
HFP (ms^2)	83±73	109±114
CVRRI (%)	9.4±3.9	9.6±3.1

Values are in mean±sd. HR_{ave}, average HR; SDNN, standard deviation of RRI; RMSSD, square root of the mean of the sum of the squares of differences between adjacent RRI; TP, total power; LFP, low-frequency power; HFP, high-frequency power; LF/HF, low- to high frequency ratio; CVRRI, coefficient of variation of RRI.

5.4.2 HR and HRV in experienced and novice jumpers

HR was higher in novices just before the jump, during the jump and during landing when compared to the experienced jumpers (Figure 6). After controlling for age-predicted maximum heart rate, the differences between the groups were still statistically significant during exit (86 ± 9 in the novices vs. 77 ± 9 % of age-predicted maximum heart rate in the experienced, $p = 0.024$), and during landing (86 ± 7 vs. 75 ± 10 %, $p = 0.004$).

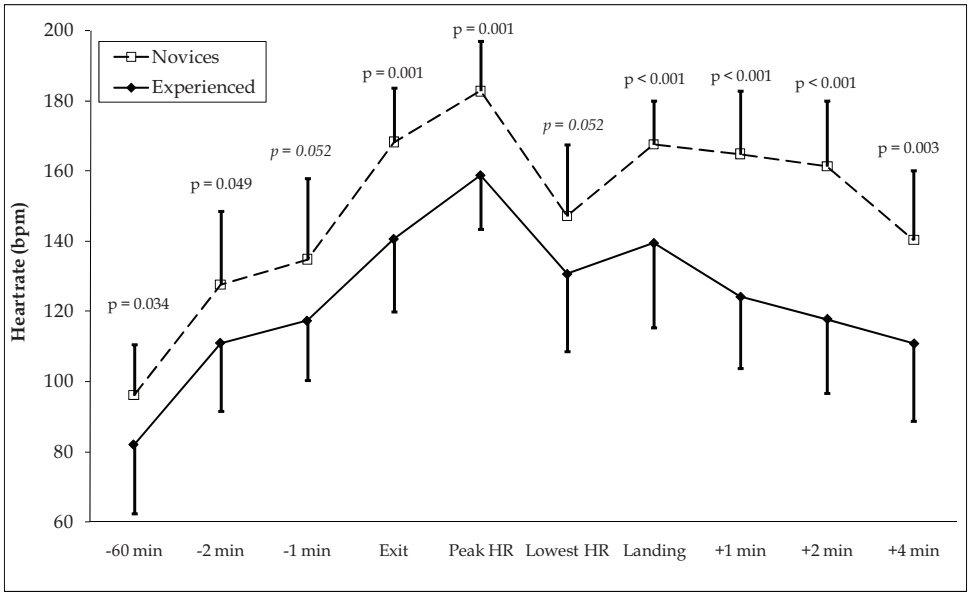

FIGURE 6 Heart rate curves before, during and after a parachute jump. Differences between the groups are presented above the HR curve of the novices.

The statistical analysis showed no significant main effect between the groups (p = 0.434) or between the nights within the groups (p = 0.533) in nocturnal HR or HRV indices. The group x night interactions were also not significant (p = 0.984). No significant main effect was found between the nights within the groups (p = 0.117) or in the groups x night interaction (p = 0.900) in nocturnal stress hormone secretions. There was also no difference in the perceived stress between the groups (16 ± 6 vs. 15 ± 5, p = 0.691).

The statistical analysis of HR indices in the orthostatic test indicated a significant main effect of the groups (p = 0.003), but no effect of day (p = 0.969) or interaction of group x day (p = 0.870) (Tables 13 and 14). In the orthostatic test after awakening the peak HR after standing up was higher in novices than in experienced jumpers (108 ± 10 vs. 95 ± 9 bpm, p = 0.005) and the instantaneous increase in heart rate was higher in novices than in experienced jumpers (47 ± 9 vs. 39 ± 8 bpm, p = 0.004). After controlling for age-predicted maximum heart rate, the difference between the groups in the peak HR after standing up was not statistically significant (40 ± 8 in novices vs. 38 ± 8 % of age-predicted maximum heart rate in experienced, p = 0.219). Furthermore, no significant main effect of the groups (p = 0.906) or time (p = 0.927) were found in HRV indices during the orthostatic test, nor was the interaction of group x day significant (p = 0.969).

TABLE 13 Supine HR and HRV after awakening in novice and experienced jumpers.

	Novice, 9♂ & 4♀		Experienced, 6♂ & 1♀	
	Jump morning	Control morning	Jump morning	Control morning
HR (bpm)	63±11	61±9	57±11	60±12
SDNN (ms)	95±38	91±42	98±41	79±22
RMSSD (ms)	74±42	70±49	78±45	53±27
TP (ms^2)	5811±5664	5457±5591	4999±2352	2729±1674
LFP (ms^2)	3146±2762	2332±1610	4042±1698	1712±1314
HFP (ms^2)	2786±2727	3125±4810	3347±1018	1018±727
CVRRI (%)	9.3±3.5	9.0±3.6	8.0±2.8	7.6±1.0

Values are in mean±sd. HR_{ave}, average heart rate; SDNN, standard deviation of RRI; RMSSD, square root of the mean of the sum of the squares of differences between adjacent RRI; TP, total power; LFP, low-frequency power; HFP, high-frequency power; LF/HF, low- to high frequency ratio; CVRRI, coefficient of variation of RRI.

TABLE 14 HR and HRV during standing in the orthostatic test in novice and experienced jumpers.

	Novice, 9♂ & 4♀		Experienced, 6♂ & 1♀	
	Jump morning	Control morning	Jump morning	Control morning
HR (bpm)	97±12	93±15	84±12	87±16
SDNN (ms)	65±28	67±24	64±26	59±19
RMSSD (ms)	23±12	24±12	22±10	21±8
TP (ms^2)	2078±2109	2249±2765	2042±2006	1978±1505
LFP (ms^2)	2252±2192	2130±2637	1992±1763	1890±1457
HFP (ms^2)	109±124	119±134	100±80	88±53
CVRRI (%)	9.7±4.3	10.2±3.4	8.7±3.3	8.3±1.8

Values are in mean±sd. HR_{ave}, average HR; SDNN, standard deviation of RRI; RMSSD, square root of the mean of the sum of the squares of differences between adjacent RRI; TP, total power; LFP, low-frequency power; HFP, high-frequency power; LF/HF, low- to high frequency ratio; CVRRI, coefficient of variation of RRI.

6 DISCUSSION

The purpose of this thesis was to investigate the association of chronic and acute, as well as physical and psychological stress with autonomic modulation. The main findings showed, that nocturnal HR, HRV and stress hormone secretion were not affected by chronic physical or psychological stress or acute psychological stress. On the contrary, acute physical stress diminished nocturnal HRV during the following night sleep. Both chronic physical and psychological stress induced lower HRV after awakening. Also, the change in HRV from the night sleep to after awakening was found to be associated in the chronic physical and psychological stress. Autonomic responses to either physical (orthostatic test) or psychological (the Stroop and relaxation) challenges were found to be more sensitive to chronic stress in comparison to nocturnal autonomic modulation.

6.1 HR, HRV and stress hormone secretions in association with chronic physical stress (I-II)

The purpose of these overtraining studies was firstly to compare the autonomic modulation during night sleep and after awakening, and secondly the cardiac autonomic responses to physical and psychological challenges, as well as the cognitive performance between severely overtrained and control athletes. It was hypothesized, that the overtrained athletes would exhibit lower HRV and nocturnal urinary stress hormone concentrations, as well as lower HR and HRV responses to light physical and psychological challenges when compared to the control athletes. The main findings showed that the cardiac autonomic modulation was disturbed in OA after awakening, but not during night sleep. In addition, both cardiac autonomic response to psychological challenge and the cognitive performance were significantly attenuated in OA.

The number of overtrained athletes was higher than has usually been reported in overtraining studies, which increased the possibilility of the study to evaluate the effects of overtraining on athletes. Athletes considered to be in an

overtrained state were diagnosed based on tight criteria. Athletes suffering from the symptoms of overtraining first contacted a physician and after careful exclusion of other possible explanations to their symptoms, they were diagnosed to be in an overtrained state. The higher perceived stress in OA than CA and the very slow recovery of performance capacity in OA, as suggested previously (Uusitalo 2001, Meeusen et al. 2006), were considered to support the overtraining state diagnosis of the OA. Actually, the overtraining state was so severe that only six out of twelve overtrained athletes could recover to their earlier competitive level in 24 months after the diagnosis of the overtraining state. OA also had lower VO_{2max} than CA, but it cannot be used to support the overtraining diagnosis. After all, we do not have baseline values for the athletes, and this difference may be related to overtraining, differences in body mass and/or training status as well. In addition, the symptoms of overtraining had forced the overtrained athletes to reduce the intensity and volume of their training and at least some of them may have already begun the recovery phase.

6.1.1 Autonomic modulation in overtrained athletes during sleep and at supine rest after awakening

In the contrary to the hypothesis, there were no differences between the groups in nocturnal HR, HRV and stress hormone secretion. However, there was a non-significant trend of lower nocturnal HRV in OA, especially in LFP, which tended to be 50 % lower in OA than in CA (Table 4). Cardiac autonomic modulation after awakening was disturbed in OA, as seen in lower supine HRV in OA than in CA. Previous controlled overtraining studies concerning HRV are scarce and the results are controversial (Uusitalo et al. 2000, Mourot et al. 2004). In these studies, HRV has been measured during supine rest a few hours after awakening in the laboratory (Uusitalo et al. 2000, Mourot et al. 2004). In the present study, HRV during sleep and immediately after awakening was collected at home in real-life conditions. Regardless of the methodological differences, the results of Mourot et al. (2004) were almost similar to the present findings in the overall HRV (SDNN). In their study, the control athletes had approximately 50 % higher values in SDNN during supine rest (awake) than the overtrained athletes. There were no significant differences in LFP, but the overtrained athletes had lower HFP than the control athletes did. This led to a higher LF to HF ratio in overtrained athletes, which the authors suggested to indicate relatively higher sympathetic activation in overtrained than in control athletes (Mourot et al. 2004). Uusitalo et al. (2000) found a significant increase in LFP during supine rest induced by heavy endurance training. However, there were no differences between the whole training group and the subgroup of overtrained athletes (Uusitalo et al. 2000).

In the real-life situations at home, the response to awakening provided enhanced sensitivity of HRV measurements to differentiate OA from CA (Table 4 and Figure 2). The effect sizes between the groups were approximately 1 SD in both SDNN and LFP after awakening (Table 4). This encourages further investigation of the phenomenon. The awakening response in secretion of cortisol

indicates an additional enhancement of the ongoing circadian activation of hypothalamic-pituitary-axis soon after awakening (Dodt et al. 1997, Wilhelm et al. 2007). Findings of lower SDNN in OA may suggest that the autonomic balance was changed to sympathetic predominance by parasympathetic withdrawal. The findings of lower LFP with almost similar but not significant difference on average HFP may support slight parasympathetic withdrawal. Awakening response in HRV has not been studied previously, but with respect to the present findings, the response to some kind of a challenge seems to provide better sensitivity than simple resting measurements.

A previous study has presented a connection between training load and HR and HRV during nocturnal sleep (Pichot et al. 2000). The athletes were not diagnosed as overtrained, but they were monitored during a 3-week intensive training period, followed by a relative rest period of one week. Their results demonstrated decreases in HRV during the intensive training period, and a recovery of HRV to the initial values after a relative rest period of one week. These findings (Pichot et al. 2000) may demonstrate the acute and reversible effects of hard training or overreaching on HRV. In the present study, there were no differences in any HR or HRV parameters during night sleep between the groups. As the OA were in a severely overtrained state or may have started the recovery phase after reducing the training, acute responses like in the study of Pichot et al. (2000) had vanished.

No differences in nocturnal urinary stress hormones were found between the groups in the present study. This indicates that basal sympathetic tone was similar in these groups, which was against our hypothesis, but agrees with the present findings of nocturnal HRV. Previous literature has found decreased nocturnal urine catecholamine concentrations after high-volume training in overtrained endurance athletes (Lehmann et al. 1992), but the results have been controversial (Uusitalo et al. 1998). There have been some methodological differences in nocturnal urinary stress hormone collections and study setups (cross-sectional vs. follow-up) between the different studies, which may explain the different findings. Lehmann et al. (1992) reported that successfully trained and competed athletes had low nocturnal urinary stress hormone concentrations, but the lowest values (a decrease by at least 50 %) were found among the group of overtrained athletes. Again, if recovery after the overtraining state had already started in the OA of the present study, the recovery may have been first visible during night sleep.

6.1.2 Autonomic responses in overtrained athletes during light physical and psychological challenges

With respect to supine rest values, great decreases were found in TP and HFP during standing suggesting that standing up induced mainly parasympathetic withdrawal. However, the responses to standing up were not different between the groups. Instead, OA had significantly lower LFP during standing in the active orthostatic test as found previously (Mourot et al. 2004). Furthermore, overtraining induced changes in standing LFP have been found to correlate with

changes in VO_{2max} (Uusitalo et al. 2000). It has been suggested that LFP, especially in normalized units reflects mostly the sympathetic activation (Furlan et al. 1993, Task Force 1996, Iellamo et al. 2002). However, recent studies have shown that HRV, regardless of the frequency band, is affected mainly by parasympathetic and only slightly by sympathetic activation (Aubert et al. 2003, Martinmäki et al. 2006a, Martinmäki et al. 2006b).

The HRV-responses to the Stroop and to the relaxation were smaller in OA when compared to CA. These differences between the groups were due to both LFP and HFP, which may suggest that overtraining attenuates not only parasympathetic but possibly also sympathetic responses. Even though there were no significant differences between the groups in absolute HRV variables during the Stroop, only CA reacted to the increase in the Stroop test speed by significantly reducing their HRV. Previous studies have found lower HR, higher HRV and stronger cardiac autonomic responses to cognitive tasks, like the Stroop in this study, in trained subjects than in untrained subjects (Boutcher et al. 1998, Franks and Boutcher 2003). Nevertheless, there is currently no literature available concerning responses to cognitive tasks or cognitive performances in overtrained athletes. Unfortunately, the present experimental design had no sedentary control group, and we cannot know whether the response level of OA was attenuated to an untrained level or not.

During the relaxation immediately after the fast speed in the Stroop, TP and LFP of both OA and CA recovered close to the values recorded during supine rest in the morning while relative HFP recovered to only 80 % of the supine rest. Taking into account that CA had significantly lower relative TP and HFP and tended to have lower relative LFP during the Stroop, they were able to recover to a greater extent than OA after the task. This finding is supported by significant interactions found in the dynamics of TP and LFP suggesting that an inability to respond to a demanding task and to recover after it might be one indicators of overtraining.

Cognitive performance of OA was diminished, as indicated by more mistakes made during the Stroop than the controls. Franks and Boutcher (2003) found that trained adolescent boys had higher parasympathetic reactivity and they also made fewer errors in the Stroop than untrained boys did. In the present study, greater autonomic recovery from low values of HRV during the Stroop was related to better performance in the Stroop, suggesting greater and more effective autonomic responses in athletes with better performances in the Stroop. No correlative calculations between autonomic reactivity and performance were performed in the study of Franks and Boutcher (2003), but they speculated that the trained subjects may have possessed a more competitive attitude towards the tasks. In contrast to that study, all the present subjects were accustomed to competitive situations, suggesting that differences in the basic attitudes towards the tasks could not explain the differences found in cognitive performance – unless OTS results in attitude acquiescence. It should be noted, that unfortunately the cause and the effect cannot be identified here due to the cross-sectional study design.

The following interesting and important question can be raised: Is the lower cognitive performance a result of overtraining and deteriorated ability to respond to cognitive challenge, or are the athletes with lower cognitive performance more vulnerable to developing OTS? At the moment, there seems to be no literature supporting the latter alternative, and therefore, the lower cognitive performance of OA in the Stroop is most probably related to the overtraining state. Instead, some recent findings support the first hypothesis: Liebermann et al. (2006) have reported that during sustained military operations lasting 84 hours, the cognitive performance has declined more extensively than physical performance. Furthermore, Rietjens et al. (2005) have reported an increase in reaction time after two weeks of intensified training, which may also be called intentional (Rusko 2003) or functional (Meeusen et al. 2006) overreaching. Also Nederhof et al. (2007) have reported a trend toward psychomotor slowness in group of overreached athletes after high load training period in comparison to a control group. These findings give some support to our finding of reduced cognitive performance due to overtraining. After all, there seemed to be a tendency that the longest reaction times also produced the most errors (Rietjens et al. 2005). Even though the physical performance seemed to increase in their study, intensified training may lead to non-functional overtraining or OTS if it is continued for too long without enough recovery time (Uusitalo 2001, Meeusen et al. 2006). Unfortunately, autonomic function was not measured in these two studies (Rietjens et al. 2005, Lieberman et al. 2006).

At rest, parasympathetic modulation is dominant and the fast responses of cardiac autonomic modulation to an acute stressor take place by parasympathetic withdrawal (Akselrod et al. 1981, Task Force 1996, Martinmäki et al. 2006b). The higher parasympathetic modulation is at rest before a challenging task, the higher subsequent parasympathetic response can be (Porges 1992). In other words, resting HRV might represent an "autonomic resource". As a matter of fact, previous literature has suggested that the parasympathetic nervous system acts as the modulator of stress vulnerability and reactivity (Porges 1992). According to finding of decreased HRV in response to awakening in overtrained athletes, the awakening in the morning seems to induce disturbance to the homeostasis in OA. Consequently, smaller further changes in HRV were available for response to a cognitive task such as the Stroop. After all, many HRV variables were decreased during the Stroop in CA, but not in OA, and simultaneously the cognitive performance in the Stroop was better in CA than in OA. Based on the previous studies and the present findings, it seems that chronic physical stress decreases the autonomic resources at rest and the ability of autonomic nervous system to respond to different challenges.

In conclusion, autonomic modulation during night sleep did not differ between the overtrained and control athletes, whereas the cardiac autonomic modulation after awakening was disturbed in overtrained athletes. The results suggest that parasympathetic cardiac modulation after awakening was slightly diminished in the overtraining state. Furthermore, the cardiac autonomic modulation during light physical challenge after awakening and responses to the

light psychological challenge, as well as cognitive performance in the severely overtrained athletes were attenuated. Severe overtraining seems to have an effect especially on parasympathetic, but perhaps also on the sympathetic branch of the autonomic nervous system. The present study is the first one to show that A) overtrained athletes had lower cognitive performance than control athletes and B) that decreased cognitive performance and lower autonomic reactivity were connected. Consequently, decreased cognitive performance may be a result of overtraining, but due to the cross-sectional study design this relationship cannot be further evaluated.

6.2 HR and HRV in association with acute physical stress (IV)

The effects of acute moderate and heavy physical stress were studied in longitudinal design in recreational endurance runners. The main findings of increased nocturnal HR and decreased nocturnal HRV suggest prolonged changes in autonomic modulation after both endurance exercise sessions. This was expected after the marathon, but the changes in nocturnal HR and HRV after moderate endurance exercise were against the hypothesis. In addition, the HR and HRV responses acted in a dose-response manner. Furthermore, the absolute decrements in nocturnal HFP after the exercise sessions were related to the nocturnal HFP after the rest day but neither of them was related to performance on marathon or VO_{2max}.

Previously it has been shown that immediate or short-time recovery of HRV after exercise to the pre-exercise level takes place in minutes after low-to moderate intensity exercise, but a significant delay in post-exercise HRV recovery has been observed following moderate- to high-intensity exercise (Seiler et al. 2007, Kaikkonen et al. 2008, Martinmäki and Rusko 2008, Kaikkonen et al. 2010). Hautala et al. (2001) reported a decrease in normalized nocturnal HFP after a 75 km ski-race. A non-significant decreasing trend in absolute HFP when compared to the night before the ski-race was also found. However, the control measurements were done during the very last night before the ski-race and anticipatory mental stress might have influenced HRV, as suggested by Hall et al. (2004). In addition, the subjects of Hautala et al. (2001) had more time to recover after the race than the subjects of this study, as the start of the ski-race was at 9 a. m. and the endurance exercises in this study were done during afternoon. The differences in the actual sport event may also have had some effect on the responses, but on the other hand the ski-race lasted approximately 1 h longer than the marathon run in the present study. Decreased nocturnal HRV after the heavy endurance exercise in this study is in line with Furlan et al. (1993), who reported decreased HRV 24 h after maximal exercise.

It was also hypothesized that nocturnal HRV would not be influenced by a moderate exercise session, but it occurred and the differences between moderate and heavy exercises suggested a dose-response relationship. Recently, Kaikkonen et al. (2010) found that increasing both intensity and duration of a

moderate exercise session reduces the immediate post-exercise HRV. The first ventilatory threshold has been proposed as a threshold of intensity for perturbation of the autonomic nervous system (Seiler et al. 2007). In the present study, the moderate exercise session was performed under the intensity of the first ventilatory threshold, but an attenuated HRV was still found. The subjects in Seiler et al. (2007) study however, were more fit than the present subjects, which may have sped up the rate of recovery of the subjects. In another study on high level endurance athletes, there was no difference in nocturnal LFP and HFP after easy and hard training days but approximately 7 % higher nocturnal HR after hard training in comparison to easy training days (Hynynen et al. 2007). Nocturnal levels of testosterone have been found to decrease while nocturnal profiles of growth hormone and cortisol concentrations change with increasing intensity of daytime exercise, which suggests that intensity of exercise seems to be a key factor in the disturbance of homeostasis (Kern et al. 1995). Furthermore, excess post-exercise oxygen consumption has been suggested to reflect the exercise induced disturbance of homeostasis (Brooks and Fahey 1985), which is strongly dependent on the intensity of the exercise. This is probably due to physiological changes that persist in recovery including "elevation in tissue temperatures, changes in intra- and extracellular ion concentrations, and changes in metabolite and hormone levels" (Brooks and Fahey 1985). These same changes were most probably persistent during nocturnal rest after endurance exercises leading to decreased nocturnal HRV.

The marathon run was used as a heavy physical stressor to investigate the upper limits of changes induced by a single endurance exercise session. When compared to previous literature on nocturnal HRV during different training periods (Pichot et al. 2000, Iellamo et al. 2002, Pichot et al. 2002b, Hynynen et al. 2007, Nummela et al. 2010), the present study's observed decreases of up to 66 % in nocturnal HFP seem to be the greatest ever reported. Details of the training programs used in previous literature were not provided, but it is not presumable that a marathon run or equivalent would have been used. On the other hand, the findings of Hautala et al. (2001) indicated only a non-significant 10 % decline in nocturnal HFP after the 75 km ski-race. This small difference is difficult to explain and further studies on nocturnal HRV after different exercises are needed.

The finding of positive correlation between the nocturnal HFP in non-stressed condition (after the resting day) and the responses to the stress (decrements in nocturnal HFP) after the moderate and heavy exercise sessions was as hypothesized. This may indicate that the amount of the adaptability of the autonomic nervous system, especially the parasympathetic branch, varied between individuals as a function of their resources (Porges 1992, Rusko et al. 2007, see also Figure 1). The adaptation process itself however, was similar between different individuals regardless of nocturnal HFP after the resting day, since the nocturnal HFP after the resting day was not related to the relative changes of nocturnal HFP. It remains unclear what the benefit of these greater resources and responses are, since these autonomic responses were not related

to the absolute performance in marathon running. Furthermore, the differences in the aerobic capacity of the subjects were not related to the nocturnal HRV, suggesting that this autonomic resource is also dependent on some other factor(s) than physical fitness. On the contrary, in a recent study of previously sedentary subjects, it was found that changes in nocturnal HRV were associated with changes in endurance running performance after 4 weeks of endurance training (Nummela et al. 2010). Recently, it was shown that monthly training load had a dose-response relationship with HRV in recreational marathon runners (Iwasaki et al. 2003, Manzi et al. 2009), but no effects of single endurance exercises were reported. In previously sedentary men and women, most of the increase in HRV occurred after 3 months of moderate doses of endurance training while a more prolonged and intense training program may not provide any more enhancements in HRV (Iwasaki et al. 2003).

In conclusion, both moderate exercise session and a marathon as a heavy exercise led to decreased nocturnal HRV and increased nocturnal HR in ten healthy, physically active men when compared to nocturnal HRV and HR after a rest day. These findings extend previous knowledge on acute HRV responses to stress of physical exercise suggesting prolonged parasympathetic withdrawal hours after the effect of the stressor itself had ceased. These HRV responses had a dose-response relationship which may be applied in practice by physically active persons, athletes and coaches in obtaining information on the extent of the disturbance of homeostasis.

6.3 HR, HRV and stress hormone secretions in association with chronic psychological stress (V)

Workers with different incidence of stress symptoms were investigated to find out the association of chronic psychological stress with HR, HRV and stress hormones at rest as well as the responses of HR and HRV to light physical challenge after awakening. Low HRV and stress hormone secretion during night sleep and light physical challenge were hypothesized to be associated with a high incidence of stress symptoms. The main finding of the present study was that regardless of age and gender, the incidence of stress symptoms was negatively associated with HRV during the light physical challenge but not during night sleep. Previous literature has suggested that accumulated physical work-stress (Pichot et al. 2002a) and athletic training (Pichot et al. 2000, Hynynen et al. 2007) may lead to decreased nocturnal HRV. Contrary to our expectations, no differences were found between the present STRESS groups in nocturnal HR, HRV and stress hormone secretions. However, we examined mainly psychological stress while the previous studies (Pichot et al. 2000, Pichot et al. 2002a, Hynynen et al. 2007) focused mainly on physical stress. In addition, the previous studies were follow-up studies, whereas the present study can be regarded as a cross-sectional study. Previous literature on stress hormone responses to psy-

chological stress have been controversial, but there appears to be a tendency that during the acute phase of psychological stress, hormone secretion increases whereas decreased secretion can be detected after exposure to chronic stress (Melamed et al. 2006). Especially awakening response in saliva cortisol have been found to be positively related to anxiety (Vreeburg et al. 2010) and job strain (Maina et al. 2009), but negatively to incidence of stress symptoms (Chida and Steptoe 2009, O'Connor et al. 2009). Unfortunately, whether basal nocturnal secretion of stress hormones is adjusted under the influence of psychological stress is unclear.

The hypothesis that the higher incidence of stress symptoms would be associated with lower HRV after awakening received support. Furthermore, the decrement of HRV in response to awakening was found to be greater in the high stress group in comparison to the low stress group. These findings suggest parasympathetic withdrawal in the presence of stress symptoms. Awakening itself is known to enhance the sympathetic tone (Dodt et al. 1997). Therefore, findings of chronic psychological and physiological stress are in line, suggesting that the decrement in HRV from night sleep to after awakening in the low stress group may have been a normal reaction, but the high stress group may have overreacted with a greater decrease in HRV.

The present findings expand on the observations that there exists an inverse relationship between perceived emotional stress during the past week and normalized HF in physically fit men and women (Dishman et al. 2000). Lucini et al. (2002) have reported changes in autonomic modulation in relation to real life stress of a university examination. In their studies, HRV was measured during supine rest (Dishman et al. 2000) and during an orthostatic test (Lucini et al. 2002) in a laboratory, whereas the measurements in the present study were carried out in a real-life setting at home. However, Dishman et al. (2000) found no difference in absolute HRV indices between low and high stress groups, which may be related to the smaller variability in Perceived Stress Scale values (Cohen et al. 1983) than in this study: 14 ± 6 vs. 22 ± 7 (Dishman et al. 2000) and 15 ± 5 vs. 27 ± 8 in the present study for the low and high stress groups, respectively. The overall stress level was adjusted according to different types of questionnaires in the Lucini et al. (2002) study, limiting the comparison with the present study.

Together with previous literature, the present findings suggest that nocturnal autonomic modulation is relatively stable, except during acute "negative" psychological stress (Hall et al. 2004) or cumulative physically stressful periods (Pichot et al. 2000, Pichot et al. 2002a, Hynynen et al. 2007). Given that the stress measures used in our study captured mainly mild global (i.e. context-free) stress, further studies focusing more directly on chronic work stress are needed. For example, Vrijkotte at al. (2000) have suggested that work stress measured by an effort-reward-imbalance model of Siegrist (1996) is related to increased autonomic and cardiovascular reactivity to stressful situations which in turn may lead to harmful health effects in the long run. Specifically, they found that low night time HRV was predictive of mild hypertension in workers.

The hypothesis that after waking up, high incidence of stress symptoms would diminish HRV during both supine rest and standing during an orthostatic test was also supported. This was associated with greater decreases in CVRRI from night sleep to the orthostatic test. These findings lend support to Porges (1992), who suggested that the parasympathetic nervous system acts as the modulator of stress vulnerability and reactivity. As the stress symptoms were related to HRV and parasympathetic activity after waking up, stress vulnerability and reactivity may have increased. If the nocturnal rest however, is stable and vagal activation high enough for recuperation and recovery, this increased stress vulnerability during wakefulness may not be hazardous for health.

As a practical implication of this study, the effects of self-reported stress on autonomic modulation may be easily estimated with simple HRV measurements in a real-life situation. In a case of remarkably decreased HRV, one might consider several alternatives. For example: one could organize time for relaxation or an exercise program and/or modify the cause inducing the stress symptoms. Previous literature has shown, that higher incidence of burnout (followed by prolonged exposure to work stress) is positively associated with age among employees (Ahola et al. 2006) while HRV is negatively associated with age, respectively (De Meersman 1993). It is therefore worth noticing that the high stress participants were on average 6 years older than their low stress counterparts. Based on these cross-sectional findings it is difficult to say whether high stress leads to low HRV or whether low HRV reflects higher stress vulnerability (Porges 1992) and lower autonomic resources. In either case, if high HRV is seen during rest, reflecting high parasympathetic modulation of heart which is favorable to our health, it is good to remember that physical activity (De Meersman 1993) and stress management programs (Lucini et al. 2007) may be advantageous in reducing the harmful cardiovascular effects of stress. Furthermore, the simple stress question of the incidence of stress symptoms was found to be practical in evaluation of global perceived stress.

In conclusion, contrary to our hypothesis, there were no differences between the STRESS groups in nocturnal HR, HRV and stress hormone secretions. However, higher incidence of self-reported stress symptoms was found to be associated with lower HRV after awakening in real-life conditions suggesting parasympathetic withdrawal. HRV measurements may be useful tools in analyzing stress in real-life conditions together with subjective evaluations of stress.

6.4 HR, HRV and stress hormone secretions in association with acute psychological stress (III)

Twenty parachute jumpers were examined to find the association of high acute psychological stress of a forthcoming parachute jump with HR, HRV and stress hormones during the preceding night and responses of HR and HRV to light

physical challenge after awakening. The main hypothesis was that the forthcoming parachute jump would attenuate HRV and accentuate HR and stress hormone secretion, and these responses would be greater in novice than experienced jumpers. The findings, however, did not support the hypothesis.

6.4.1 Anticipatory Stress During Sleep and After Awakening

As previous literature has shown (Falk and Bar-Eli 1995), the parachute jumpers had a high peak HR of over 90 % of the age-predicted maximum HR during the parachute jump, indicating that it was a highly stressful event. The acceleration of HR beyond 110-120 bpm is mediated by sympathetic part of autonomic nervous system (Robinson et al. 1966, Orizio et al. 1988, Hainsworth 1998), and these findings reflect high sympathetic activity. Anticipatory stress from the forthcoming parachute jump did not have any effects on HR or HRV during the preceding night and morning, which was contrary to what was hypothesized. Instead, these findings are in line with the study of stress of competition in pentathlon, where no differences were found in HRV between control and competition mornings (Iellamo et al. 2003). Slightly elevated adrenaline secretion during the jump night could be related to elevated mental stress (Richter et al. 1996), but this effect was small and physiologically insignificant and may be explained by seasonal variation (Hansen et al. 2001). The parachute jumps were performed during summer, where basal adrenaline secretion is highest, while the control measurements were done 1-2 months later. The present results then, are not in line with the laboratory study of Hall et al. (2004), who reported decreased nocturnal high-frequency power and increased LF/HF ratio during the night preceding a stressful morning. In this previous study of Hall et al. (2004), an oral speech shortly following waking up was used as a stressful task whereas in the present study the parachute jump was performed several hours after waking up. This time difference may at least partly explain the differences between these two studies during the night; despite the fact that during the parachute jump HR increased more than 100 bpm above the resting HR, whereas in a speech task the increase in HR can be approximately 20 bpm above the resting HR.

It was expected to find a decrease in HRV reflecting an anticipatory stress effect, but no difference was found between the jump and control nights. Previously, many types of prolonged or chronic stress have been found to reduce HRV during nighttime sleep (Pichot et al. 2000, Vrijkotte et al. 2000, Pichot et al. 2002a, Brosschot et al. 2007), which may be interpreted to mean that prolonged effects of stress can be negative on health by changing the autonomic modulation of the heart in an unfavorable manner. As the parachute jump induced a very high acute stress, but did not have any effect on nocturnal autonomic modulation, it is probable that changes in nocturnal autonomic modulation are related to different kinds of stress. This may be related to the "positive" and "negative" stress. For example, the parachute jump was expected to be a positive experience, but the stress of public speech (Hall et al. 2004) was considered as a negative one.

6.4.2 Novices vs. Experienced Jumpers

Similarly to previous literature (Schedlowski and Tewes 1992, Falk and Bar-Eli 1995, Roth et al. 1996), HR was found to be higher in novices throughout the parachute jump procedure starting from 1 h before the jump up to a few minutes after landing, when the RRI-collection was stopped. These findings may be interpreted as higher sympathetic activation in novices than in experienced jumpers. The difference between novice and experienced jumpers in the present study was a bit greater than in the previous literature (Schedlowski and Tewes 1992, Falk and Bar-Eli 1995, Roth et al. 1996), which may be partly related to the fact that the jump under investigation was the very first one the novices had done, whereas the novices in the previous studies (Schedlowski and Tewes 1992, Falk and Bar-Eli 1995, Roth et al. 1996) had already done a few jumps. However, the differences in HR responses to parachute jumping between these studies seem to be relatively small and therefore, experience seems to have negligible effect on the response to this kind of high acute stress.

Even though the high acute stress of parachute jumping had been repeated many times during the summer by the experienced jumpers, it had no effect on the nocturnal autonomic modulation. So, this kind of repeated acute stress most probably does not induce any negative health effects related to chronic stress (Pichot et al. 2000, Vrijkotte et al. 2000, Pichot et al. 2002a, Pieper and Brosschot 2005, Brosschot et al. 2007). Instead, higher peak HR together with higher instantaneous increase in HR was found in the orthostatic test in novices when compared to experienced jumpers. Spangler (1997) has reported higher HR and lower HRV as anticipatory effects just a few minutes before a stressful event (an exam vs. control situation), but unfortunately they did not do any measurements during the preceding night or morning. In the present study however, the difference between the groups was similar in the control measurements suggesting that the hypothesized higher acute stress of parachute jumping in novices had no effect on the orthostatic test results in the morning of the jump. Instead, the difference in orthostatic test results between novice and experienced jumpers seemed to be due to the slight age difference between the groups and, therefore, lends no further support to the previous findings (Spangler 1997).

In conclusion, the high acute stress of a parachute jump resulted in very high sympathetic activation during the jump, but it did not have any anticipatory stress effects on autonomic modulation during nocturnal sleep and after the awakening in the morning. Experience had only a minor effect on the responses during the highly stressful event, but not during the preceding night and morning.

6.5 Limitations

Respiration is known to affect HRV, but in a real-life situation and especially during night sleep it is not possible to control respiration (STUDIES I-V). Our pretest observations of breathing pattern during the Stroop also indicated that the breathing frequency did not change or induce any remarkable drift to the LF or HF components of the power spectrum (non-published observations). If this kind of shift existed, it is only a minor limitation since almost all HRV at any frequency band has been proven to reflect mainly vagal activity (Taylor et al. 1998, Martinmäki et al. 2006a).

The practical problems in fitting the time schedules of athletes and researchers generated some time differences between the diagnosis and measurements (STUDIES I-II). This delay may have given some more time for the OA to recover before the measurements, but on the other hand, recovery after severe overtraining state is known to take a long time (Kreider et al. 1998, Uusitalo 2001, Meeusen et al. 2006). As a matter of fact, previous (Kreider et al. 1998) and recent (Meeusen et al. 2006) reviews conclude that it is possible to recover from overreaching within a few weeks or months but recovery from overtraining state may take months or possibly years. The fastest recovery in the present group of overtrained athletes to competitions was 6 months, and half of the group could not compete in the following 24 months. Actually, our observations of slow recovery during this exceptionally long 24-month follow-up also verify previous conclusions of Kreider et al. (1998) and Meeusen et al. (2006). The higher perceived stress reported by the OA athletes during the measurements also gives support to the fact that the overtrained athletes were still in overtraining state during the measurements. Finally, the measurements were done within 3-6 weeks after the diagnosis during which period the athletes continued to train at a reduced intensity and volume. The previous studies suggest that a recovery period ranging from a week to a few weeks after overreaching most probably increases HRV (Pichot et al. 2000, Uusitalo et al. 2000) instead of the present finding of decreased HRV.

There are some limitations related to study III. Firstly, the order of the measurements was the same for everyone, first the jump and later the control situation. The recruitment of novices was done from a parachuting course. After recruitment there was less than 2 wk to their first jump and a non-stressful control situation could not be arranged before the jump. We expected that the parachuting course might have some influence on the autonomic modulation and we wanted to be sure that the control measurements were free of known stressors. Secondly, the experienced jumpers were older than the novices and age is known to have some influence on HR. However, getting experience of hundreds of parachute jumps takes years and, therefore there are not many young and experienced parachute jumpers. Therefore, an attempt was also made to adjust the HR results to age-related HRmax. Thirdly, in comparison to the other parts of this study the stress effects were expected to be anticipatory

and thus no recordings after the acute stress of parachute jumping were done. This was done based on expectations from previous literature and also for practical reasons: the novices wanted to celebrate their first jump, which would have had negative influences on HRV indices.

The studies I and II were cross-sectional studies. Therefore, we cannot definitely know if the findings of lower VO_{2max} in overtrained athletes result from overtraining, nor can we relate the present HRV findings to the athletes' own "well-being" values. As the overtrained athletes had reduced the intensity and volume of their training due to the overtraining symptoms, they may not have been in the worst possible state at the time of these measurements. The same limitation is relevant in study V, where cross-sectional data groups of different stress statuses were investigated. To add to the generalizability of results of STUDY V, the study should be replicated using representative samples of different occupational groups. Also a group of burnout subjects should be included, as the subjects in the high stress group were still able to work even though the incidence of stress symptoms was high. In both of these studies of chronic stress, there was a non-significant tendency of lower nocturnal HRV in the high stress group, which raises a question whether a larger number of subjects would have led to significant difference This is however, the biggest group of overtrained athletes reported in scientific literature, as far as we know. In the study of chronic psychological stress increasing the number of subjects studied is possible and might give different picture of the effects of chronic stress on the nocturnal HRV.

Finally, the recordings of RRIs were done with automated equipment and thus no ECG data was available (STUDIES I-V). Therefore, it is impossible to know if erroneous beats were caused by measurement error or extra beats. This limitation was minimized by carefully checking the RRI data with the help of automated detection software and in the case of probable error the correction was confirmed by visual inspection. Furthermore, the negligible difference between Holter (ECG) recordings and the method used in this study has been previously shown to be clinically irrelevant in healthy adults (Loimaala et al. 1999). Furthermore, HRV reflects mainly parasympathetic modulation of heart and therefore, information of sympathetic cardiac autonomic modulation was not available. As the initial increase in HR up to approximately 100 bpm is mainly due to reduction of parasympathetic activity (Robinson et al. 1966, Orizio et al. 1988, Hainsworth 1998), sympathetic activity was supposed to be negligible in the most situations in this study. However, this should be taken into account in the interpretation of the autonomic modulation in this thesis – it is mainly of parasympathetic origin. Furthermore, one should keep in mind that even though HRV reflects parasympathetic activity well, it cannot be regarded as a direct measure of vagal tone (Stein 2005).

7 MAIN FINDINGS AND CONCLUSION

The main findings of this study were as follows:

1) Chronic stress did not have an influence on nocturnal HR, HRV and stress hormone secretion. On the contrary, chronic stress induced lower resting HRV immediately after awakening. Also the change in HRV from the night sleep to after awakening was found to be associated with chronic physical and psychological stress. The findings of HR, HRV and perceived stress were similar in both chronic physical and chronic psychological stress.
2) In comparison to nocturnal autonomic modulation, autonomic modulation during either a physical (orthostatic test) or a psychological (the Stroop and relaxation) challenge were found to be more sensitive to chronic stress.
3) Acute physical stress diminished nocturnal HRV during the following night sleep in a dose-response manner.
4) Acute psychological stress did not have effects on HR and HRV indices during the preceding night and morning.

Awakening itself enhances the sympathetic tone (Dodt et al. 1997), and both chronic physical and psychological stress seemed to provide an extra enhancement to it. Burnout patients have shown elevated cortisol levels during the first hour after awakening (De Vente et al. 2003). Similar overreaction to awakening may also be seen in the enhanced decrease in HRV of chronic physical and chronic psychological stress subjects. Interestingly, there is a markedly increased risk of malignant ventricular arrhythmias, sudden cardiac death, and stroke in the hours around and just after awakening (Elliot 2001, Guo and Stein 2003) – the same time that disturbed autonomic modulation was found under influence of chronic stress in this study.

In conclusion, this study showed that nocturnal autonomic modulation in real-life situation is relatively stable, except during a recovery phase from an acute physical stress. The awakening period in the morning was found to be

sensitive to different stressors and the effects of both physical and psychological stress were found to be similar: vagal withdrawal and possibly a slight increase of sympathetic activity. This is in accordance with the previous stress literature starting from Selye's general adaptation syndrome, but the novel finding in this study was that the possible effects of different stressors on autonomic modulation can be easily estimated with HRV measurements in real-life condition. The HRV method used in this study is available practically for anyone. The association of chronic stress with overall HRV is concluded in the Figure 7.

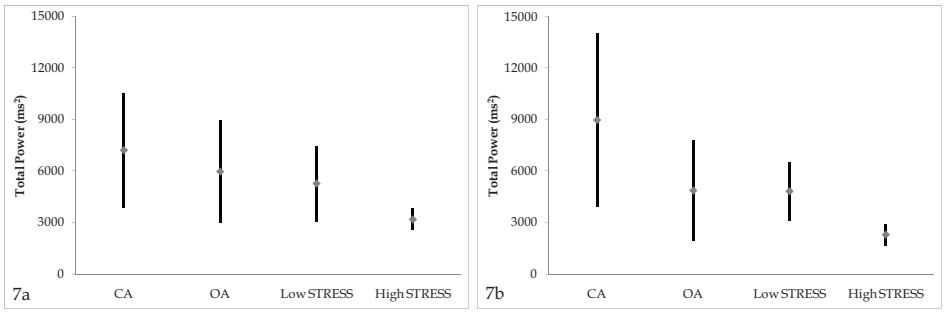

FIGURE 7 The association of chronic stress and overall HRV during night sleep (7a) and immediately after awakening (7b). The figures show 95 % confidence intervals and means of control athletes (CA), athletes with chronic stress (OA), workers with only minor stress symptoms (LowSTRESS), and workers with chronic stress (HighSTRESS).

Finally, according to the findings of this study, the autonomic resource model (Figure 1 in page 14) can be modified as seen in Figure 8. The original idea of Rusko et al. (2007) is widened to show the "whole range" of the functional area of the autonomic modulation, not only the area that is usually needed to random, not heavy challenge. In case of a heavy challenge, not only vagal resources, but also the sympathetic resources are activated. In Figure 8 the updated parts are highlighted to show, that:

1. In chronic stress, both vagal and sympathetic resources are limited, but during sleep the limitations are not as great as after awakening. This was seen in both chronic physical and chronic psychological stress subjects, but especially the chronic psychological stress seems to have diminished the autonomic resources, as seen also in Figure 7.
2. When the recovery is optimal or typical, the sympathetic resources are also great or only slightly decreased.

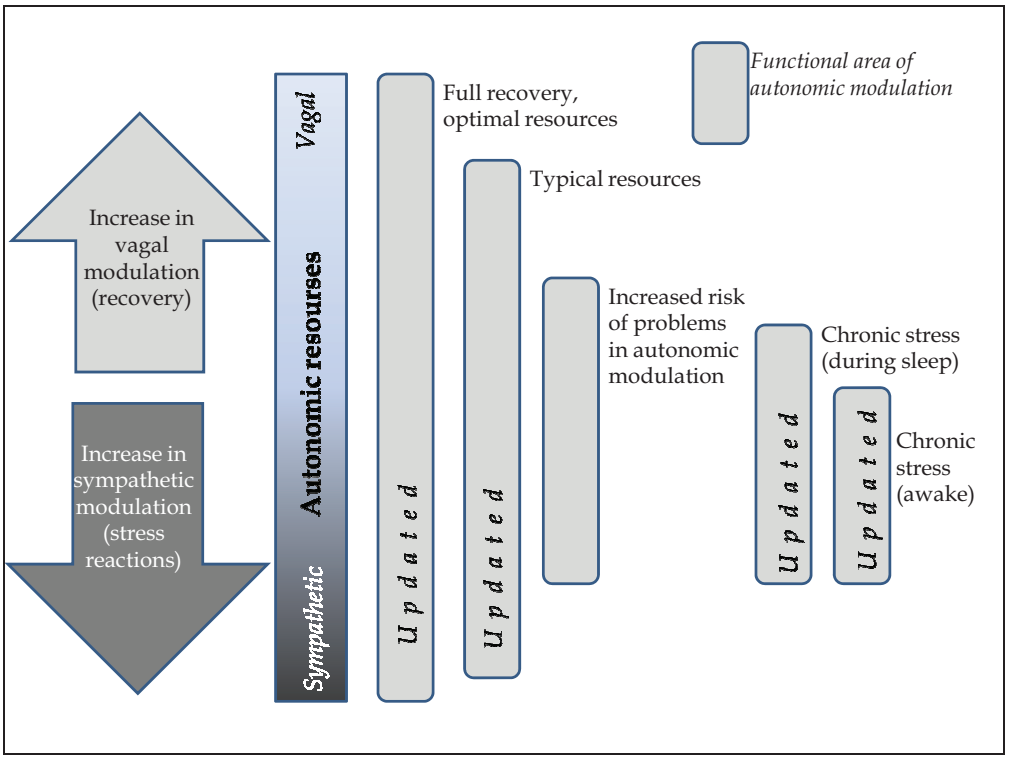

FIGURE 8 Updated model of the autonomic resources. Modified from Rusko et al. (2007).

YHTEENVETO

Sykevariaatiomittaukset kroonisen ja akuutin stressin seurannassa käyttäen hyväksi yöunen ja akuuttien tehtävien aikaisia vasteita

Stressi mielletään helposti negatiiviseksi asiaksi, mutta toisaalta se voidaan nähdä myös ärsykkeenä tai haasteena, johon ihmisen on vastattava. Stressitutkimukset ovat selvittäneet mm. erilaisia akuutteja fysiologisia vasteita psykologiseen stressiin, mikä on aiheutettu esim. matemaattisilla tehtävillä tai julkisella esiintymisellä. Myös arkielämän stressit, kuten työperäinen stressi ja surutyö ovat olleet tutkimuksen kohteina. Näissä tilanteissa stressireaktio ilmentää ihmisen kykyä käsitellä ärsykettä tai haastetta. Negatiiviseksi stressireaktio muuttuu yleensä vasta, kun se pitkittyy, eikä esim. viikonlopun palautuminen riitä työviikon rasituksista.

Urheiluvalmennus pohjautuu pitkälti ylikuormitusperiaatteeseen, jonka mukaan urheilijaa kuormitetaan fyysisellä harjoituksella siten, että elimistön tasapainotila järkkyy. Palautumisen aikana suorituskyky palautuu ennalleen, jopa hieman korkeammalle tasolle kuin ennen kuormitusta. Elimistö valmistautuu siten kohtaamaan uuden kuormituksen aiempaa valmiimpana. Ilmiönä fyysinen ja psyykkinen stressi voidaan nähdä samanlaisina haasteina, joihin ihminen reagoi akuutisti valmiustilaa nostaen ja akuutin kuormituksen mentyä ohi ennalleen palautuen tai jopa uutta resurssia rakentaen.

Autonominen hermosto mukautuu erilaisiin haasteisiin säätelemällä sympaattisen ja parasympaattisen haaransa aktiivisuutta. Nämä haarat täydentävät toisiaan ja niillä on periaatteessa toistensa vastakkaiset vaikutukset; esim. sympaattinen aktiivisuus supistaa verisuonia ja kohottaa sykettä ja parasympaattinen aktiivisuus päinvastoin. Autonomisen hermoston toimintaa on tutkittu aiemmin paljon hormonimääritysten avulla, mutta sykevariaation avulla arviointi on mahdollista myös noninvasiivisesti. Sykevariaatio kuvastaa pääasiassa sydämen parasympaattista säätelyä, joten se soveltuu parhaiten tilaan, missä parasympaattinen aktiivisuus on voimakkainta tai sen aktiivisuutta muutetaan jollain ärsykkeellä.

Tämän tutkimuksen tarkoituksena oli selvittää autonomisen hermoston säätelyn yhteyksiä krooniseen ja akuuttiin fyysiseen ja psyykkiseen stressiin syke-, sykevariaatio- ja stressihormonimääritysten avulla.

Kroonisen fyysisen stressin vaikutuksia tutkittiin käyttämällä poikkileikkausasetelmaa. Vakavaan ylikuormitustilaan ajautuneita urheilijoita verrattiin kontrolliurheilijoihin molempien ryhmien koostuessa puoliksi miehistä, puoliksi naisista ja edustaen eri urheilulajeja. Tulokset osoittivat, että krooninen fyysinen stressi ei vaikuttanut yön aikaiseen autonomiseen säätelyyn, mutta aamulla heti heräämisen jälkeen tehdyssä ortostaattisessa kokeessa sykevariaatio oli madaltunut ylikuormittuneilla urheilijoilla. Laboratorio-olosuhteissa tehdyssä Stroop tehtävässä ylikuormittuneet urheilijat tekivät enemmän virheitä ja suhteessa aamun leposykevariaatioon muutokset Stroopin ja sitä seuranneen rentoutuksen aikana olivat pienempiä kuin kontrolliurheilijoilla. Sykevariaatiovas-

teen havaittiin lisäksi korreloivan kognitiivisen suorituskyvyn kanssa. Tulokset viittasivat siihen, että krooninen fyysinen stressi vaimensi parasympaattista säätelyä aamulla heti heräämisen jälkeen ja siten supisti parasympaattista resurssia reagoida tuleviin haasteisiin.

Akuutin fyysisen stressin vaikutuksia sykkeeseen ja sykevariaatioon tutkittiin kuntoilijamiehillä pitkittäisasetelmalla kohtalaisen kestävyysharjoituksen sekä maratonin jälkeisenä yönä. Tulokset osoittivat, että vaikka kohtalainen kestävyysharjoitus koettiin helpoksi, myös sen jälkeisenä yönä syke oli hieman kohonnut ja sykevariaatio hieman laskenut lepopäivän jälkeiseen yöhön verrattuna. Maratonin jälkeisen yön syke oli kohonnut ja sykevariaatio laskenut edelleen myös kohtalaiseen kestävyysharjoitukseen verrattuna. Akuutin fyysisen stressin aiheuttamat vasteet yösykevariaatiossa olivat yhteydessä lepopäivän jälkeiseen yösykevariaatioon siten, että absoluuttinen muutos oli suurinta niillä, joiden yösykevariaatio oli lepopäivän jälkeen suurinta. Tulokset viittasivat siihen, että sydämen parasympaattinen säätely vaimeni ja sympaattinen aktiivisuus saattoi olla lievästi kohonneena akuutisti kestävyysharjoitusta seuranneena yönä. Vasteiden annos-vastesuhde viittaa siihen, että vastetta voidaan hyödyntää kestävyysvalmennuksessa yksittäisten harjoitusten kuormittavuutta arvioitaessa.

Kroonisen psyykkisen stressin vaikutuksia tutkittiin käyttämällä poikkileikkausasetelmaa. Tutkittavat olivat työelämässä olevia miehiä ja naisia, jotka jaettiin koettujen stressioireiden toistuvuuden perusteella matalan ja korkean stressin ryhmiin. Lisäksi huomioitiin ikä ja sukupuoli, sillä tässä aineistossa myös niillä havaittiin olevan oma yhteytensä sykevariaatiomuuttujiin. Tulokset osoittivat, että krooninen psyykkinen stressi ei vaikuttanut yön aikaiseen autonomiseen säätelyyn, mutta aamulla heti heräämisen jälkeen tehdyssä ortostaattisessa kokeessa sykevariaatio oli madaltunut kroonisen psyykkisen stressin ryhmällä. Madaltunut sykevariaatio ortostaattisessa kokeessa viittaa siihen, että krooninen psyykkinen stressi vaimensi sydämen parasympaattista säätelyä aamulla heti heräämisen jälkeen.

Akuutin psyykkisen stressin vaikutuksia tutkittiin laskuvarjohyppääjillä sekä poikittais- että pitkittäisasetelmalla. Sekä aloittelijoissa että kokeneissa hyppääjissä oli sekä miehiä että naisia. Akuutti sykevaste laskuvarjohyppyyn oli aloittelijoilla voimakkaampi kuin kokeneilla hyppääjillä sekä absoluuttisena että suhteutettuna iän mukaiseen maksimisykkeeseen. Sekä hyppyaamun että kontrolliaamun ortostaattisessa kokeessa aloittelijoiden akuuttisykevaste seisomaan nousuun oli korkeampi kuin kokeneilla hyppääjillä. Tämä havainto kuitenkin liittynee poikkileikkausasetelmaan, sillä iän mukaiseen maksimisykkeeseen suhteutettu sykevaste ei eronnut ryhmien välillä. Pitkittäisasetelman tulokset osoittivat, ettei laskuvarjohypyn aiheuttama kova akuutti psykologinen stressi vaikuttanut yöunen tai aamun ortostaattisen kokeen aikaiseen sykkeeseen tai sykevariaatioon. Itse hypyn aikainen korkea syke osoitti voimakasta sympaattista aktivaatiota. Hyppy-yön aikainen adrenaliinin eritys oli hieman suurempaa kuin kontrolliyön, mutta selittynee vuodenaikojen aiheuttamalla kausivaihtelulla, eikä siten liity akuuttiin stressiin.

Tutkimuksen perusteella voidaan todeta, että yksinkertaisilla kotioloissa suoritettavilla syke- ja sykevariaatiomittauksilla voidaan tutkia sekä fyysisen että psyykkisen stressin vaikutuksia sydämen autonomiseen säätelyyn: Sykevaihtelun mittaaminen yön aikana näyttäisi toimivan fyysisen harjoittelun aiheuttaman stressin vaikutusten arvioinnissa ja aamulla tehty ortostaattinen koe taas enemmän kroonista fyysistä ja/tai psyykkistä alkuperää olevan stressin vaikutusten arvioinnissa. Mittaustuloksia voidaan hyödyntää toisaalta stressitutkimuksissa, mutta myös yksilöllisessä seurannassa, kuten urheilussa ja kuntoilussa sekä erilaisissa stressinhallintaohjelmissa. Tässä tutkimuksessa käytettyjen mittausten erityinen etu on niiden non-invasiivisyyden ohella edullisuus ja yksinkertaisuus, sillä kalliita mittalaitteita ja määritysmenetelmiä ei tarvita. Mittaukset soveltuvat siten kotioloissa suoritettavina lähes kenelle vain ja yksinkertaisimmat laskennatkin onnistuvat esim. taulukkolaskentaohjelmalla.

REFERENCES

Ahola K, Honkonen T, Kivimäki M, Virtanen M, Isometsä E, Aromaa A & Lönnqvist J. 2006. Contribution of burnout to the association between job strain and depression: The health 2000 study. Journal of Occupational and Environmental Medicine / American College of Occupational and Environmental Medicine 48 (10), 1023-1030.

Akselrod S, Gordon D, Ubel FA, Shannon DC, Berger AC & Cohen RJ. 1981. Power spectrum analysis of heart rate fluctuation: A quantitative probe of beat-to-beat cardiovascular control. Science 213 (4504), 220-222.

Antelmi I, de Paula RS, Shinzato AR, Peres CA, Mansur AJ & Grupi CJ. 2004. Influence of age, gender, body mass index, and functional capacity on heart rate variability in a cohort of subjects without heart disease. The American Journal of Cardiology 93 (3), 381-385.

Armstrong LE & VanHeest JL. 2002. The unknown mechanism of the overtraining syndrome: Clues from depression and psychoneuroimmunology. Sports Medicine 32 (3), 185-209.

Aubert AE, Seps B & Beckers F. 2003. Heart rate variability in athletes. Sports Medicine 33 (12), 889-919.

Barantke M, Krauss T, Ortak J, Lieb W, Reppel M, Burgdorf C, Pramstaller PP, Schunkert H & Bonnemeier H. 2008. Effects of gender and aging on differential autonomic responses to orthostatic maneuvers. Journal of Cardiovascular Electrophysiology 19 (12), 1296-1303.

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 Sport Medicine : Official Journal of the Canadian Academy of Sport Medicine 16 (5), 412-417.

Bernard C 1865. Introduction à létude de la médecine expérimentale. Paris: J.B.Bailliere et Fils.

Bernardi L, Keller F, Sanders M, Reddy PS, Griffith B, Meno F & Pinsky MR. 1989. Respiratory sinus arrhythmia in the denervated human heart. Journal of Applied Physiology 67 (4), 1447-1455.

Berntson GG, Bigger JT,Jr, Eckberg DL, Grossman P, Kaufmann PG, Malik M, Nagaraja HN, Porges SW, Saul JP, Stone PH & van der Molen MW. 1997. Heart rate variability: Origins, methods, and interpretive caveats. Psychophysiology 34 (6), 623-648.

Bigger JT,Jr, Fleiss JL, Steinman RC, Rolnitzky LM, Kleiger RE & Rottman JN. 1992. Frequency domain measures of heart period variability and mortality after myocardial infarction. Circulation 85 (1), 164-171.

Biondi M & Picardi A. 1999. Psychological stress and neuroendocrine function in humans: The last two decades of research. Psychotherapy and Psychosomatics 68 (3), 114-150.

Bosquet L, Papelier Y, Leger L & Legros P. 2003. Night heart rate variability during overtraining in male endurance athletes. Journal of Sports Medicine & Physical Fitness 43 (4), 506-512.

Boutcher SH, Nugent FW, McLaren PF & Weltman AL. 1998. Heart period variability of trained and untrained men at rest and during mental challenge. Psychophysiology 35 (1), 16-22.

Brooks G and Fahey T. 1985. Metabolic response to exercise. In G. Brooks and T. Fahey (Eds) Exercise physiology; human bioenergetics and its applications. Vol. New York: Macmillan Publishing Company, 189-219.

Brosschot JF, Van Dijk E & Thayer JF. 2007. Daily worry is related to low heart rate variability during waking and the subsequent nocturnal sleep period. International Journal of Psychophysiology 63 (1), 39-47.

Brosschot JF, Pieper S & Thayer JF. 2005. Expanding stress theory: Prolonged activation and perseverative cognition. Psychoneuroendocrinology 30 (10), 1043-1049.

Byrne EA, Fleg JL, Vaitkevicius PV, Wright J & Porges SW. 1996. Role of aerobic capacity and body mass index in the age-associated decline in heart rate variability. Journal of Applied Physiology 81 (2), 743-750.

Cannon WB. 1914. The emergency function of the adrenal medulla in pain and the major emotions. Am.J.Physiol. 33 356-372.

Carney RM, Steinmeyer B, Freedland KE, Blumenthal JA, Stein PK, Steinhoff WA, Howells WB, Berkman LF, Watkins LL, Czajkowski SM, Domitrovich PP, Burg MM, Hayano J & Jaffe AS. 2008. Nighttime heart rate and survival in depressed patients post acute myocardial infarction. Psychosomatic Medicine 70 (7), 757-763.

Chida Y & Steptoe A. 2009. Cortisol awakening response and psychosocial factors: A systematic review and meta-analysis. Biological Psychology 80 (3), 265-278.

Clays E, De Bacquer D, Crasset V, Kittel F, de Smet P, Kornitzer M, Karasek R & De Backer G. 2010. The perception of work stressors is related to reduced parasympathetic activity. International Archives of Occupational and Environmental Health DOI 10.1007/s00420-010-0537-z

Cohen S, Kamarck T & Mermelstein R. 1983. A global measure of perceived stress. Journal of Health and Social Behavior 24 (4), 385-396.

De Meersman RE & Stein PK. 2007. Vagal modulation and aging. Biological Psychology 74 (2), 165-173.

De Meersman RE. 1993. Heart rate variability and aerobic fitness. American Heart Journal 125 (3), 726-731.

De Vente W, Olff M, Van Amsterdam JG, Kamphuis JH & Emmelkamp PM. 2003. Physiological differences between burnout patients and healthy controls: Blood pressure, heart rate, and cortisol responses. Occupational and Environmental Medicine 60 Suppl 1 i54-61.

Dishman RK, Nakamura Y, Garcia ME, Thompson RW, Dunn AL & Blair SN. 2000. Heart rate variability, trait anxiety, and perceived stress among physically fit men and women. International Journal of Psychophysiology: Official Journal of the International Organization of Psychophysiology 37 (2), 121-133.

Dodt C, Breckling U, Derad I, Fehm HL & Born J. 1997. Plasma epinephrine and norepinephrine concentrations of healthy humans associated with nighttime sleep and morning arousal. Hypertension 30 (1 Pt 1), 71-76.

Eckberg DL. 1983. Human sinus arrhythmia as an index of vagal cardiac outflow. Journal of Applied Physiology: Respiratory, Environmental and Exercise Physiology 54 (4), 961-966.

Elliot WJ. 2001. Cyclic and circadian variations in cardiovascular events. American Journal of Hypertension 14 (9 Pt 2), 291S-295S.

Elo AL, Leppänen A & Jahkola A. 2003. Validity of a single-item measure of stress symptoms. Scandinavian Journal of Work, Environment & Health 29 (6), 444-451.

Falk B & Bar-Eli M. 1995. The psycho-physiological response to parachuting among novice and experienced parachutists. Aviation, Space, and Environmental Medicine 66 (2), 114-117.

Franks PW & Boutcher SH. 2003. Cardiovascular response of trained preadolescent boys to mental challenge. Medicine and Science in Sports and Exercise 35 (8), 1429-1435.

Fry AC, Kraemer WJ & Ramsey LT. 1998. Pituitary-adrenal-gonadal responses to high-intensity resistance exercise overtraining. Journal of Applied Physiology 85 (6), 2352-2359.

Fry RW, Morton AR & Keast D. 1991. Overtraining in athletes. an update. Sports Medicine 12 (1), 32-65.

Furlan R, Piazza S, Dell'Orto S, Gentile E, Cerutti S, Pagani M & Malliani A. 1993. Early and late effects of exercise and athletic training on neural mechanisms controlling heart rate. Cardiovascular Research 27 (3), 482-488.

Furlan R, Guzzetti S, Crivellaro W, Dassi S, Tinelli M, Baselli G, Cerutti S, Lombardi F, Pagani M & Malliani A. 1990. Continuous 24-hour assessment of the neural regulation of systemic arterial pressure and RR variabilities in ambulant subjects. Circulation 81 (2), 537-547.

Grossman P & Kollai M. 1993. Respiratory sinus arrhythmia, cardiac vagal tone, and respiration: Within- and between-individual relations. Psychophysiology 30 (5), 486-495.

Guo YF & Stein PK. 2003. Circadian rhythm in the cardiovascular system: Chronocardiology. American Heart Journal 145 (5), 779-786.

Guyton AC & Hall JE. 1996. Textbook of medical physiology. Philadelphia, USA: W.B. Saunders Company.

Hainsworth R. 1998. Physiology of the cardiac autonomic system. In M Malik (Ed) Clinical guide to cardiac autonomic tests, Vol. AA Dordrecht, The Netherlands: Kluwer Academic Publishers, 3-28.

Häkkinen K, Pakarinen A, Alen M, Kauhanen H & Komi PV. 1988. Daily hormonal and neuromuscular responses to intensive strength training in 1 week. International Journal of Sports Medicine 9 (6), 422-428.

Hales S. 1733. Haemastatics. In S. Hales (Ed) Statistical essays, Vol. London: Innys and Manby, 1-86.

Hall M, Vasko R, Buysse D, Ombao H, Chen Q, Cashmere JD, Kupfer D & Thayer JF. 2004. Acute stress affects heart rate variability during sleep. Psychosomatic Medicine 66 (1), 56-62.

Hansen AM, Garde AH, Skovgaard LT & Christensen JM. 2001. Seasonal and biological variation of urinary epinephrine, norepinephrine, and cortisol in healthy women. International Journal of Clinical Chemistry 309 (1), 25-35.

Hartikainen JEK, Tahvanainen KUO and Kuusela TA. 1998. Short-time measurement of heart rate variability. In M Malik (Ed) Clinical guide to cardiac autonomic tests, Vol. AA Dordrecht, The Netherlands: Kluwer Academic Publishers, 149-176.

Hautala A, Tulppo MP, Mäkikallio TH, Laukkanen R, Nissilä S & Huikuri HV. 2001. Changes in cardiac autonomic regulation after prolonged maximal exercise. Clinical Physiology 21 (2), 238-245.

Hedelin R, Kenttä G, Wiklund U, Bjerle P & Henriksson-Larsen K. 2000. Short-term overtraining: Effects on performance, circulatory responses, and heart rate variability. Medicine and Science in Sports and Exercise 32 (8), 1480-1484.

Hirsch JA & Bishop B. 1981. Respiratory sinus arrhythmia in humans: How breathing pattern modulates heart rate. The American Journal of Physiology 241 (4), H620-9.

Hohnloser SH and Klingenheben T. 1998. Basic autonomic tests. In M Malik (Ed) Clinical guide to cardiac autonomic tests, Vol. AA Dordrecht, The Netherlands: Kluwer Academic Publishers, 51-65.

Hon EH & Lee ST. 1963. Electronic evaluation of the fetal heart rate. viii. patterns preceding fetal death, further observations. American Journal of Obstetrics and Gynecology 87 814-826.

Hon EH. 1958. The electronic evaluation of the fetal heart rate; preliminary report. American Journal of Obstetrics and Gynecology 75 (6), 1215-1230.

Huikuri HV, Mäkikallio TH, Airaksinen KE, Seppänen T, Puukka P, Räihä IJ & Sourander LB. 1998. Power-law relationship of heart rate variability as a predictor of mortality in the elderly. Circulation 97 (20), 2031-2036.

Huikuri HV, Linnaluoto MK, Seppänen T, Airaksinen KE, Kessler KM, Takkunen JT & Myerburg RJ. 1992. Circadian rhythm of heart rate variability in survivors of cardiac arrest. The American Journal of Cardiology 70 (6), 610-615.

Huikuri HV, Kessler KM, Terracall E, Castellanos A, Linnaluoto MK & Myerburg RJ. 1990. Reproducibility and circadian rhythm of heart rate variability in healthy subjects. The American Journal of Cardiology 65 (5), 391-393.

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

Iellamo F, Pigozzi F, Parisi A, Di Salvo V, Vago T, Norbiato G, Lucini D & Pagani M. 2003. The stress of competition dissociates neural and cortisol ho-

meostasis in elite athletes. The Journal of Sports Medicine and Physical Fitness 43 (4), 539-545.

Iellamo F, Legramante JM, Pigozzi F, Spataro A, Norbiato G, Lucini D & Pagani M. 2002. Conversion from vagal to sympathetic predominance with strenuous training in high-performance world class athletes. Circulation 105 (23), 2719-2724.

Israel S. 1976. The problems of overtraining in a medical and sports physiological point of view (zur problematik des übertrainings aus internisticher und leistungsphysiologischer sicht). Medizin Und Sport 16 (1), 1-12.

Iwasaki K, Zhang R, Zuckerman JH & Levine BD. 2003. Dose-response relationship of the cardiovascular adaptation to endurance training in healthy adults: How much training for what benefit? Journal of Applied Physiology 95 (4), 1575-1583.

Johnson TA, Gray AL, Lauenstein JM, Newton SS & Massari VJ. 2004. Parasympathetic control of the heart. I. an interventriculo-septal ganglion is the major source of the vagal intracardiac innervation of the ventricles. Journal of Applied Physiology 96 (6), 2265-2272.

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.

Kaikkonen P, Rusko H & Martinmäki K. 2008. Post-exercise heart rate variability of endurance athletes after different high-intensity exercise interventions. Scandinavian Journal of Medicine & Science in Sports 18 (4), 511-519.

Karsner HT & Denis W. 1914. A further study of nitrogen retention in the blood in experimental acute nephritis. The Journal of Experimental Medicine 19 (3), 259-269.

Katona PG & Jih F. 1975. Respiratory sinus arrhythmia: Noninvasive measure of parasympathetic cardiac control. Journal of Applied Physiology 39 (5), 801-805.

Kellmann M. 2010. Preventing overtraining in athletes in high-intensity sports and stress/recovery monitoring. Scandinavian Journal of Medicine & Science in Sports 20 Suppl 2 95-102.

Kern W, Perras B, Wodick R, Fehm HL & Born J. 1995. Hormonal secretion during nighttime sleep indicating stress of daytime exercise. Journal of Applied Physiology 79 (5), 1461-1468.

Keselbrener L and Akselrod S. 1998. Autonomic responses to blockades and provocations. In M Malik (Ed) Clinical guide to cardiac autonomic tests, Vol. Dordrecht, Netherlands: Kluwer Academic Publishers, 101-148.

Kivimäki M, Virtanen M, Elovainio M, Kouvonen A, Väänänen A & Vahtera J. 2006. Work stress in the etiology of coronary heart disease--a meta-analysis. Scandinavian Journal of Work, Environment & Health 32 (6), 431-442.

Kreider RB, Fry AC & O'Toole ML 1998. Overtraining in sport. Champaign, IL: Human Kinetics.

Kuipers H & Keizer HA. 1988. Overtraining in elite athletes. review and directions for the future. Sports Medicine 6 (2), 79-92.

Lehmann M, Schnee W, Scheu R, Stockhausen W & Bachl N. 1992. Decreased nocturnal catecholamine excretion: Parameter for an overtraining syndrome in athletes? International Journal of Sports Medicine 13 (3), 236-242.

Lehmann M, Dickhuth HH, Gendrisch G, Lazar W, Thum M, Kaminski R, Aramendi JF, Peterke E, Wieland W & Keul J. 1991. Training-overtraining. A prospective, experimental study with experienced middle- and long-distance runners. International Journal of Sports Medicine 12 (5), 444-452.

Levy MN, Martin PJ, Iano T & Zieske H. 1970. Effects of single vagal stimuli on heart rate and atrioventricular conduction. The American Journal of Physiology 218 (5), 1256-1262.

Lieberman HR, Niro P, Tharion WJ, Nindl BC, Castellani JW & Montain SJ. 2006. Cognition during sustained operations: Comparison of a laboratory simulation to field studies. Aviation, Space, and Environmental Medicine 77 (9), 929-935.

Loimaala A, Sievänen H, Laukkanen R, Parkka J, Vuori I & Huikuri H. 1999. Accuracy of a novel real-time microprocessor QRS detector for heart rate variability assessment. Clinical Physiology 19 (1), 84-88.

Lucini D, Riva S, Pizzinelli P & Pagani M. 2007. Stress management at the worksite: Reversal of symptoms profile and cardiovascular dysregulation. Hypertension 49 (2), 291-297.

Lucini D, Norbiato G, Clerici M & Pagani M. 2002. Hemodynamic and autonomic adjustments to real life stress conditions in humans. Hypertension 39 (1), 184-188.

Lundberg V & Hellström B. 2002. Workload and morning salivary cortisol in women. Work and Stress 16 (4), 356-363.

Maina G, Bovenzi M, Palmas A & Larese Filon F. 2009. Associations between two job stress models and measures of salivary cortisol. International Archives of Occupational and Environmental Health 82, 1141-1150.

Malliani A, Pagani M, Lombardi F & Cerutti S. 1991. Cardiovascular neural regulation explored in the frequency domain. Circulation 84 (2), 482-492.

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 (6), H1733-40.

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 (3), 353-360.

Martinmäki K, Rusko H, Kooistra L, Kettunen J & Saalasti S. 2006a. Intraindividual validation of heart rate variability indexes to measure vagal effects on hearts. American Journal of Physiology. Heart and Circulatory Physiology 290 (2), H640-7.

Martinmäki K, Rusko H, Saalasti S & Kettunen J. 2006b. 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 (6), H2582-9.

Meeusen R, Duclos M, Gleeson M, Rietjens G, Steinacker J & Urhausen A. 2006. Prevention, diagnosis and treatment of the overtraining syndrome. ECSS position statement 'Task force'. European Journal of Sport Science 6 1-14.

Meijman T and Mulder G. 1998. Psychological aspects of work load. In PJ Drenth and H Thierry (Eds) Handbook of work and organizational psychology, Vol. Hove, UK: Psychology Press, 5-33.

Melamed S, Shirom A, Toker S, Berliner S & Shapira I. 2006. Burnout and risk of cardiovascular disease: Evidence, possible causal paths, and promising research directions. Psychological Bulletin 132 (3), 327-353.

Milczarek M, Schneider E & González ER. 2009. European risk observatory report.

Morgan WP, Brown DR, Raglin JS, O'Connor PJ & Ellickson KA. 1987. Psychological monitoring of overtraining and staleness. British Journal of Sports Medicine 21 (3), 107-114.

Mourot L, Bouhaddi M, Perrey S, Cappelle S, Henriet MT, Wolf JP, Rouillon JD & Regnard J. 2004. Decrease in heart rate variability with overtraining: Assessment by the poincare plot analysis. Clinical Physiology and Functional Imaging 24 (1), 10-18.

Munck A, Guyre PM & Holbrook NJ. 1984. Physiological functions of glucocorticoids in stress and their relation to pharmacological actions. Endocrine Reviews 5 (1), 25-44.

Nederhof E, Lemmink K, Zwerver J & Mulder T. 2007. The effect of high load training on psychomotor speed. International Journal of Sports Medicine 28 (7), 595-601.

Nummela A, Hynynen E, Kaikkonen P & Rusko H. 2010. Endurance performance and nocturnal HRV indices. International Journal of Sports Medicine 31 (3), 154-159.

O'Connor DB, Hendrickx H, Dadd T, Elliman TD, Willis TA, Talbot D, Mayes AE, Thethi K, Powell J & Dye L. 2009. Cortisol awakening rise in middle-aged women in relation to psychological stress. Psychoneuroendocrinology 34 (10), 1486-1494.

Orizio C, Perini R, Comande A, Castellano M, Beschi M & Veicsteinas A. 1988. Plasma catecholamines and heart rate at the beginning of muscular exercise in man. European Journal of Applied Physiology and Occupational Physiology 57 (5), 644-651.

Pagani M, Lombardi F, Guzzetti S, Rimoldi O, Furlan R, Pizzinelli P, Sandrone G, Malfatto G, Dell'Orto S & Piccaluga E. 1986. Power spectral analysis of heart rate and arterial pressure variabilities as a marker of sympatho-vagal interaction in man and conscious dog. Circulation Research 59 (2), 178-193.

Parker P, Celler BG, Potter EK & McCloskey DI. 1984. Vagal stimulation and cardiac slowing. Journal of the Autonomic Nervous System 11 (2), 226-231.

Pichot V, Bourin E, Roche F, Garet M, Gaspoz JM, Duverney D, Antoniadis A, Lacour JR & Barthelemy JC. 2002a. Quantification of cumulated physical fatigue at the workplace. European Journal of Physiology 445 (2), 267-272.

Pichot V, Busso T, Roche F, Garet M, Costes F, Duverney D, Lacour JR & Barthelemy JC. 2002b. Autonomic adaptations to intensive and overload training periods: A laboratory study. Medicine and Science in Sports and Exercise 34 (10), 1660-1666.

Pichot V, Roche F, Gaspoz JM, Enjolras F, Antoniadis A, Minini P, Costes F, Busso T, Lacour JR & Barthelemy JC. 2000. Relation between heart rate variability and training load in middle-distance runners. Medicine and Science in Sports and Exercise 32 (10), 1729-1736.

Pieper S & Brosschot JF. 2005. Prolonged stress-related cardiovascular activation: Is there any? Annals of Behavioral Medicine 30 (2), 91-103.

Pinna GD, Maestri R, Torunski A, Danilowicz-Szymanowicz L, Szwoch M, La Rovere MT & Raczak G. 2007. Heart rate variability measures: A fresh look at reliability. Clinical Science 113 (3), 131-140.

Porges SW. 1995. Cardiac vagal tone: A physiological index of stress. Neuroscience and Biobehavioral Reviews 19 (2), 225-233.

Porges SW. 1992. Vagal tone: A physiologic marker of stress vulnerability. Pediatrics 90 (3 Pt 2), 498-504.

Pruessner JC, Hellhammer DH & Kirschbaum C. 1999. Burnout, perceived stress, and cortisol responses to awakening. Psychosomatic Medicine 61 (2), 197-204.

Ramaekers D, Ector H, Aubert AE, Rubens A & Van de Werf F. 1998. Heart rate variability and heart rate in healthy volunteers. is the female autonomic nervous system cardioprotective? European Heart Journal 19 (9), 1334-1341.

Richter SD, Schurmeyer TH, Schedlowski M, Hadicke A, Tewes U, Schmidt RE & Wagner TO. 1996. Time kinetics of the endocrine response to acute psychological stress. The Journal of Clinical Endocrinology and Metabolism 81 (5), 1956-1960.

Rietjens GJ, Kuipers H, Adam JJ, Saris WH, van Breda E, van Hamont D & Keizer HA. 2005. Physiological, biochemical and psychological markers of strenuous training-induced fatigue. International Journal of Sports Medicine 26 (1), 16-26.

Ritvanen T, Louhevaara V, Helin P, Väisänen S & Hänninen O. 2006. Responses of the autonomic nervous system during periods of perceived high and low work stress in younger and older female teachers. Applied Ergonomics 37 (3), 311-318.

Ritvanen T, Laitinen T & Hänninen O. 2004. Relief of work stress after weekend and holiday season in high school teachers. Journal of Occupational Health 46 (3), 213-215.

Robinson BF, Epstein SE, Beiser GD & Braunwald E. 1966. Control of heart rate by the autonomic nervous system. studies in man on the interrelation between baroreceptor mechanisms and exercise. Circulation Research 19 (2), 400-411.

Rosengren A, Hawken S, Ounpuu S, Sliwa K, Zubaid M, Almahmeed WA, Blackett KN, Sitthi-amorn C, Sato H, Yusuf S & INTERHEART investigators. 2004. Association of psychosocial risk factors with risk of acute myocardial infarction in 11119 cases and 13648 controls from 52 countries (the INTERHEART study): Case-control study. Lancet 364 (9438), 953-962.

Roth WT, Breivik G, Jorgensen PE & Hofmann S. 1996. Activation in novice and expert parachutists while jumping. Psychophysiology 33 (1), 63-72.

Ruha A, Sallinen S & Nissilä S. 1997. A real-time microprocessor QRS detector system with a 1-ms timing accuracy for the measurement of ambulatory HRV. IEEE Transactions on Bio-Medical Engineering 44 (3), 159-167.

Rusko H, Feldt T & Kinnunen M-L. 2007. Stressin ja palautumisen mittaaminen sykevälianalyysin avulla. (Heart rate variability in measurements of stress and recovery). Yksityislääkäri (3), 39-41.

Rusko H 2003. Cross country skiing. Malden, MA, USA: Blackwell Science.

Saul JP, Berger RD, Albrecht P, Stein SP, Chen MH & Cohen RJ. 1991. Transfer function analysis of the circulation: Unique insights into cardiovascular regulation. The American Journal of Physiology 261 (4 Pt 2), H1231-45.

Saul JP, Rea RF, Eckberg DL, Berger RD & Cohen RJ. 1990. Heart rate and muscle sympathetic nerve variability during reflex changes of autonomic activity. The American Journal of Physiology 258 (3 Pt 2), H713-21.

Schedlowski M & Tewes U. 1992. Physiological arousal and perception of bodily state during parachute jumping. Psychophysiology 29 (1), 95-103.

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 (8), 1366-1373.

Selye H. 1983. The stress concept: past, present, and future. In C. L. Cooper (Ed) Stress research, Vol. Chichester: John Wiley & Sons, 1-20.

Selye H 1956. The stress of life. New York: McGraw-Hill.

Siegrist J. 1996. Adverse health effects of high-effort/low-reward conditions. Journal of Occupational Health Psychology 1 (1), 27-41.

Singh JP, Larson MG, O'Donnell CJ, Tsuji H, Evans JC & Levy D. 1999. Heritability of heart rate variability: The framingham heart study. Circulation 99 (17), 2251-2254.

Spangler G. 1997. Psychological and physiological responses during an exam and their relation to personality characteristics. Psychoneuroendocrinology 22 (6), 423-441.

Stansfield SA & Marmot MG 2002. Stress and the heart: Psychosocial pathways to coronary heart disease. BMJ Publishing Group.

Stein PK. 2005. Vagal tone: Myths and realities. Journal of Cardiovascular Electrophysiology 16 (8), 870-871.

Steptoe A. 2000. Stress, social support and cardiovascular activity over the working day. International Journal of Psychophysiology : Official Journal of the International Organization of Psychophysiology 37 (3), 299-308.

Task Force. 1996. Heart rate variability: Standards of measurement, physiological interpretation and clinical use. Task force of the European Society of

cardiology and the North American Society of pacing and electrophysiology. Circulation 93 (5), 1043-1065.

Taylor JA, Myers CW, Halliwill JR, Seidel H & Eckberg DL. 2001. Sympathetic restraint of respiratory sinus arrhythmia: Implications for vagal-cardiac tone assessment in humans. American Journal of Physiology.Heart and Circulatory Physiology 280 (6), H2804-14.

Taylor JA, Carr DL, Myers CW & Eckberg DL. 1998. Mechanisms underlying very-low-frequency RR-interval oscillations in humans. Circulation 98 (6), 547-555.

Thayer JF, Verkuil B, Brosschot JF, Kampschroer K, West A, Sterling C, Christie IC, Abernethy DR, Sollers JJ, Cizza G, Marques AH & Sternberg EM. 2010. Effects of the physical work environment on physiological measures of stress. European Journal of Cardiovascular Prevention and Rehabilitation DOI: 10.1097/HJR.0b013e328336923a.

Tsuji H, Larson MG, Venditti FJ,Jr, Manders ES, Evans JC, Feldman CL & Levy D. 1996. Impact of reduced heart rate variability on risk for cardiac events. the framingham heart study. Circulation 94 (11), 2850-2855.

Umetani K, Singer DH, McCraty R & Atkinson M. 1998. Twenty-four hour time domain heart rate variability and heart rate: Relations to age and gender over nine decades. Journal of the American College of Cardiology 31 (3), 593-601.

Uusitalo AL. 2001. Overtraining: Making a difficult diagnosis and implementing targeted treatment. The Physician and Sportsmedicine 29 (5), 35-50.

Uusitalo AL, Uusitalo AJ & Rusko HK. 2000. Heart rate and blood pressure variability during heavy training and overtraining in the female athlete. International Journal of Sports Medicine 21 (1), 45-53.

Uusitalo AL, Huttunen P, Hanin Y, Uusitalo AJ & Rusko HK. 1998. Hormonal responses to endurance training and overtraining in female athletes. Clinical Journal of Sport Medicine 8 (3), 178-186.

Uusitalo AL, Tahvanainen KU, Uusitalo AJ & Rusko HK. 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 (6), 575-588.

Vreeburg SA, Zitman FG, van Pelt J, Derijk RH, Verhagen JC, van Dyck R, Hoogendijk WJ, Smit JH & Penninx BW. 2010. Salivary cortisol levels in persons with and without different anxiety disorders. Psychosomatic Medicine 72 (4), 340-347.

Vrijkotte TG, van Doornen LJ & de Geus EJ. 2000. Effects of work stress on ambulatory blood pressure, heart rate, and heart rate variability. Hypertension 35 (4), 880-886.

Weippert M, Kumar M, Kreuzfeld S, Arndt D, Rieger A & Stoll R. 2010. Comparison of three mobile devices for measuring R-R intervals and heart rate variability: Polar S810i, suunto t6 and an ambulatory ECG system. European Journal of Applied Physiology 109 (4), 779-786.

Wilhelm I, Born J, Kudielka BM, Schlotz W & Wust S. 2007. Is the cortisol awakening rise a response to awakening? Psychoneuroendocrinology 32 (4), 358-366.

Wolf S. 1967. The end of the rope: The role of the brain in cardiac death. Canadian Medical Association Journal 97 (17), 1022-1025.

Wust S, Federenko I, Hellhammer DH & Kirschbaum C. 2000. Genetic factors, perceived chronic stress, and the free cortisol response to awakening. Psychoneuroendocrinology 25 (7), 707-720.

Zhang J. 2007. Effect of age and sex on heart rate variability in healthy subjects. Journal of Manipulative and Physiological Therapeutics 30 (5), 374-379.

Zulfiqar U, Jurivich DA, Gao W & Singer DH. 2010. Relation of high heart rate variability to healthy longevity. The American Journal of Cardiology 105 (8), 1181-1185.

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