HEART RATE VARIABILITY –BASED RECOVERY DURING SLEEP COMPARED TO MOVEMENT ANALYSIS, SUBJECTIVE RATINGS AND CORTISOL AWAKENING RESPONSES

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ABSTRACT

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Prolonged work stress of employees or heavy training without adequate recovery of athletes can alter the balance between the sympathetic and the parasympathetic branch of the autonomic nervous system (ANS) towards sympathetic dominance, which is a risk factor for cardiovascular health. Heart rate (HR) and heart rate variability (HRV) recordings have been used as indicators of the function of ANS. A new method has been developed to evaluate stress and recovery from ambulatory RR-interval recordings. Firstbeat PRO Heartbeat Analysis Software (FBT-PRO) combines information on HR and HRV indices to evaluate the state of the body, i.e. whether the body of a person is e.g. under stress reactions, recovering (in relaxation state), or doing physical exercise. The aims of this study were to assess stress and relaxation during awake and sleep, and to compare FBT-PRO indices during sleep to indices of movement based Actiwatch Sleep Analysis® (ASA), to subjective ratings of the quality of sleep, and to cortisol awakening responses, which all have been used a lot in stress research.

The subjects were 14 nurses from the Central Hospital of Central Finland (age: 44±10 years, height: 163±5 cm, and weight: 63±10 kg). RR-intervals and wrist movements were recorded for 24 hours during workday 1 (WD1), workday 2 (WD2) and day off (DO) and analyzed for stress and relaxation with FBT-PRO and for sleep quality with ASA, respectively. The subjects took three saliva samples (at awakening, 30 min and 60 min after) each morning for cortisol analyses (raw values and cortisol awakening responses during 60 minutes from awakening) and rated whether they had slept well, moderately or badly (1–3). The two workday sleeps were categorized into better or worse sleep based on subjective ratings, fragmentation of the sleep, actual sleep time (AST), cortisol at awakening, and cortisol awakening response. In addition, HRV variables were analyzed with time and frequency domain methods and calculated for each 5-min segment. Nonparametric Wilcoxon and Marginal Homogeneity tests were used in the statistical analysis.

Significantly more stress during awake (65±20% vs. 25±19%, p=0.008) and relaxation during sleep (1±3% vs. 65±19%, p=0.002) was detected with FBT-PRO. During sleep, HR was lower (84±6 vs. 60±5 btm, p=0.002), and RMSSD and HF power greater (p=0.003 for both), whereas LF/HF ratio was lower (p=0.002) than during awake. AST was significantly longer (6:34±1:05 vs. 7:55±0:41 h:min, p=0.005), and there was more relaxation time detected (4:29±1:38 vs. 5:25±1:58, p=0.037) during DO than during WD2 sleep, which did not differ from WD1 sleep. The DO sleep was also rated to be subjectively better (p=0.034) than WD2 sleep. When better and worse workday sleeps were compared, there were no differences in stress or relaxation during subjectively different sleeps. Relaxation time was longer during less than more fragmented sleep (5:05±2:01 vs. 4:35±1:48 h:min, p=0.026). Relaxation time was longer (5:06±1:37 vs. 4:25±1:37 h:min, p=0.026) and there was greater RMSSD (p=0.050) during workday sleep with longer than with shorter AST. The increase and change in cortisol after awakening (measured with area under curve) were smaller (p=0.050 for both) during workday sleep with longer than with shorter AST. Lower awakening cortisol was accompanied by a greater increase and change in cortisol (p=0.050 and 0.041, respectively), and there tended to be less stress time (p=0.075) during sleep with lower awakening cortisol. There was longer AST (p=0.050) and greater sleep efficiency measured with ASA (p=0.029) but no differences in stress or relaxation variables during sleep with a smaller cortisol awakening response.

The results show that stress and relaxation states can be detected from HR and HRV, i.e. there was more stress during awake and more relaxation during sleep. Parasympathetic modulation seems to be dominating sleep and sympathetic activity awake-time. During workdays with longer actual sleep time and during day off, more relaxation time during sleep was achieved through longer sleep. Restlessness during sleep was related to reduced relaxation time and to a shift in favour of stress in relaxation/stress - balance. Sleep with lower awakening cortisol tended to include less stress time, but cortisol awakening response did not seem to differentiate stress or relaxation times during sleep. These results give further evidence that both sleep quantity and quality are important factors for adequate physiological recovery. In conclusion, the new HR and HRV –based method seems to allow the analysis of stress and recovery during sleep and awake in healthy working population. More studies are needed with greater number of subjects and with different subject groups (e.g. athletes) to find out more specifically the connections between this new method and the other methods used in measuring recovery.

Keywords: Heart Rate Variability, Recovery, Stress, Sleep, Cortisol, Actigraphy, Firstbeat PRO

ABBREVIATIONS

ANS	Autonomic nervous system. Divided to the sympathetic (SNS) and parasympathetic nervous system (PNS).
HPA axis	Hypothalamic-pituitary-adrenal axis.
SAM system	Sympatho-adrenal medullary system.
HRV	Heart rate variability, the variations in heart rate around its mean value or differences in times between adjacent heart beats.
R-R interval (ms)	Time between adjacent heart beats.
SDNN (ms)	Standard deviation of R-R intervals, estimate of overall HRV.
RMSSD (ms)	The square root of the mean squared differences of successive R-R intervals, estimate of short-term components of HRV.
HF (ms^2)	High frequency variation of R-R intervals (0.15-0.40 Hz).
LF (ms^2)	Low frequency variation of R-R intervals (0.04-0.15 Hz).
LF/HF	Ratio of high to low frequency variation in R-R intervals.
FBT-PRO	Heart rate and heart rate variability -based method for analyzing stress and recovery in ambulatory settings.
Actigraphy	A method to study activity and sleep patterns based on body movement recordings.
ASA	Actiwatch Sleep Analysis Software. A sleep analysis method based on actigraphy.
$AC_{0/30/60\ min}$	Cortisol value at awakening (0 min), 30 minutes after (30 min), and 60 minutes after awakening
AUC	Area under curve representing another way to express the magnitude of cortisol awakening response.
$AUC_{g\ 0\text{-}60min}$	Area under curve with respect to ground from awakening to 60 minutes after awakening.
$AUC_{i\ 0\text{-}60min}$	Area under curve with respect to increase from awakening to 60 minutes after awakening.
$AUC_{c\ 0\text{-}60min}$	Area under curve with respect to change from awakening to 60 minutes after awakening.
$CAR_{0\text{-}30\ min\ /\ 0\text{-}60\ min}$	Cortisol awakening response. Calculated by subtraction of the awakening value from the value 30/60 min after awakening.
WD1	Workday 1. A workday at the beginning of the working period.
WD2	Workday 2. A workday at the end of the working period, 3-4 days after workday 1.
DO	Day off. A first leisure day after working period.

CONTENTS

1 INTRODUCTION

Homeostasis of the human body is constantly challenged by intrinsic or extrinsic, real or perceived, physical or mental factors, which are called stressors. The body and mind react to stressors by activating a complex physiological and behavioural central nervous system and peripheral responses. If these responses are inadequate or excessive and/or prolonged, it may affect behaviour and personality development as well as numerous physiological functions, such as growth, metabolism, circulation, reproduction, and the immune system. (Charmandari et al. 2005.) The stressors cause immediate activation of the autonomic nervous system (ANS), resulting in increased activity of the sympathetic and decreased activity of the parasympathetic nervous system (SNS and PNS, respectively), but also in activation of the hypothalamic-pituitary-adrenal axis (HPA axis) and the sympatho-adrenomedullary system (SAM system) in which the adrenal gland plays a crucial role (Pignatelli et al. 1998). The changes in the activity of the bodily systems are useful and needed in short-term, but if the reactions are prolonged or excessive or there are problems in switching off the stress response, it can have serious adverse health effects (McEwen 1998).

Nowadays, most of the stress experienced by a person has its origin in working life. Work has changed during the past decades towards more demands, mostly because of higher mental requirements rather than physical workload (European Foundation for the Improvement of Living and Working Conditions 2005). It was noticed already in 1992 that heart rate is elevated when a person experiences stress in real life (Dobkin & Pihl 1992). Later it has been shown that work stress in employees or heavy training in athletes can alter the balance of ANS towards sympathetic dominance (Vrijkotte et al. 2000, Uusitalo et al. 2000). Therefore, it is not surprising that work stress has also been connected to a higher risk of cardiovascular health problems in recent studies. Increased heart rate reactivity and sympathetic tone and decreased parasympathetic modulation caused by work-related or other stress can all partly cause hypertension and other cardiovascular diseases, i.e. serious health outcomes (McEwen 1998, Vrijkotte et al. 2000, Landsbergis et al. 2001). It has been noticed that employees who are stressed out have higher blood pressure during the workday but also at leisure time than control

subjects (Schnall et al. 1998, Vrijkotte et al. 2000), they may have alterations in hormone secretion (e.g. cortisol) (Lundberg & Hellström 2002, Dahlgren et al. 2005, Pruessner et al. 1999), problems in maintaining sleep quality (Edéll-Gustafsson et al. 2002, Kudielka et al. 2004b), psychological disorders and mood changes (Tennant 2001), and decreased productivity at work as well as increased absenteeism from work for sickness (European Foundation for the Improvement of Living and Working Conditions 2005).

It has also been speculated that the negative effects of work stress would affect not only during work but especially during leisure time and sleep, making recovery more difficult, but these findings still require more evidence (Vrijkotte et al. 2000). However, it seems obvious that adequate recovery between working periods and during leisure time is essential for maintaining health, well-being, and quality of life. Recovery during off-work time can be achieved via rest, sleep, and changing activities. (Rook & Zijlstra 2006, Taris et al. 2006, Sonnentag & Kruel 2006.) Physiological recovery occurs when the activity and arousal levels of physiological systems are allowed to return again to the baseline level (Craig & Cooper 1992). The recovery process is also reflected in psychological detachment from work and decreased feelings of fatigue (Zijlstra & Sonnentag 2006).

Heart rate variability (HRV) means fluctuations in heart rate around its mean value or the differences in times between successive heart beats. Heart rate and heart rate variability recordings have been used as indicators of the function of ANS (Task Force 1996). HRV analysis has been proposed as a non-invasive technique for the evaluation of job-related cardiovascular stressors (Kristal-Boneh et al. 1995) and it provides an excellent tool for ambulatory measurement of work stress. There are still limited number of studies dealing with work stress and HRV available, but e.g. Vrijkotte et al. (2000) and van Amelsvoort et al. (2000) have studied the connections between HRV and work stress. Vrijkotte et al. (2000) reported that high work stress was associated with lower 24-h parasympathetic modulation than low work stress, and van Amelsvoort et al. (2000) that in high work demands group sympathetic modulation was higher during work and parasympathetic modulation lower during night-sleep compared to low work demands group. However, it has been shown that HRV values are very much individual, and therefore, the traditional HRV variables have only modestly been

associated with the stress experienced by a person. The factors that could explain this variability include differences in autonomic modulation among subjects, different curves relating autonomic effects to HRV for different subject, and/or age-related, sex-related, and physical activity -related differences (Goldberger et al. 2001).

Firstbeat Technologies Ltd (Jyväskylä, Finland, www.firstbeat.fi) has developed a new method to evaluate stress and recovery based on heart rate (HR) and HRV. Firstbeat PRO Heartbeat Analysis Software (FBT-PRO) combines information on HR and HRV indices based on ambulatory RR-interval recordings to describe the function of ANS, and to evaluate different bodily states. The method detects different bodily states of a person, such as stress reactions, recovery (relaxation), physical activity, and recovery from physical activity during the measured period, and takes into account the individual level and range of HR and HRV during the analysis. The aims of this master's thesis were to evaluate how FBT-PRO functions in measuring stress and recovery during awake and sleep, and to compare FBT-PRO indices during sleep to indices of movement based actigraphy (Actiwatch Sleep Analysis Software®, Cambridge Neurotechnology Ltd, Cambridge, UK, www.camntech.com), to subjective ratings of the quality of sleep and cortisol awakening responses, which all have been used a lot in stress research.

This study was a part of a currently ongoing larger research project "Heart Rate and Work Stress" in the University of Jyväskylä. The aim of the project is to validate the new method for detecting work-related stress and to provide models and advice for the use of the method in occupational health care settings. With a practicable and an objective tool for the measurement of stress and recovery of an individual, in addition to widely used psychological questionnaires, it would be easier to detect signs of increased need for recovery, decreased ability to work, and to provide means for maintaining the health and well-being of an individual, but also productivity in occupational settings.

2 WORK STRESS, HEALTH AND RECOVERY

2.1 Work stress and recovery

Stress can be defined in a number of different ways. From Cannon's (1914) description, where the stress response was seen as a 'fight or flight' response, the concept of stress has come through Selye's (1946) model of General Adaptation Syndrome, where the stress was seen as the sum of non-specific changes in the body caused by function or damage, to the most popular approach nowadays, the transactional model of stress, based on Lazarus' studies (1966). In this approach, a person experiences stress when the perceived demands of the environment are greater than his/her perceived ability to cope with them. This highlights the meaning of the psychological aspect of stress and means that the same stressor can be experienced in a number of ways - not everyone experience stress in the same situations. (Perrewé & Zellars 1999). Firstbeat Technologies defines stress reaction to be "an increase in the activity level of the body caused by internal or external stressors, where the fundamental parts are the reflective and continuous responses in the autonomic nervous system and the hormonal responses. Then sympathetic activity of the autonomic nervous system is predominant, and parasympathetic activity is recessive. The stressors can be experienced as both positive and negative." (Handbook of Wellness Analysis Software 1.4.0.14, in Finnish.)

Work-related stress has been under growing interest across Europe in recent years. The workplace has changed dramatically during the past decades due to globalization of economy, organizational restructuring, use of new technology, change towards older and more educated employees, increase in women participation and increased mental workload. (European Foundation for the Improvement of Living and Working Conditions 2005, Sparks et al. 2001, Tennant 2001.) An imbalance between work demands and environmental or personal resources can cause a number of reactions. These may include physiological (e.g. increase in heart rate and blood pressure), emotional (e.g. feeling nervous or irritated), and cognitive responses (e.g. reduced attention), as well as behavioural reactions (e.g. aggressive behaviour, making

mistakes). (European Foundation for the Improvement of Living and Working Conditions 2005.)

Theoretical models of work stress examining the interaction between health affecting work environment and person's cognitions, emotions, and social environment have been applied in the studies of work stress and health. Several theoretical models exist, such as the job strain (Karasek 1979), the effort-reward imbalance (ERI) (Siegrist et al. 2004), and the effort-recovery (Meijman & Mulder 1998) models. Karasek's (1979) job strain model predicts that mental strain results from the interaction of high job demands and low job decision latitude. The effort-reward imbalance model (Siegrist et al. 2004) integrates structural and personal components of the work, and chronic work stress is defined in the model as an imbalance between high efforts spent (in terms of e.g. high demands and workload, low controllability of the work, personal coping pattern) and low rewards received (in terms of e.g. money, appreciation, and career opportunities). According to the effort-recovery model (Meijman & Mulder 1998), workload requires effort which is responded to by short-term psychophysiological reactions (e.g. increased heart rate and hormone secretion, and mood changes). These responses are adaptive and reversible as long as there is enough time to recover between exposure to workload and within work settings (e.g. to change working strategy, regulate work demands). It is assumed that workers are capable of adjusting their effort and sometimes exceeding their capacity even if fatigued, but this compensatory effort may lead to even more increased fatigue levels. Accumulation of fatigue with insufficient time for recovery may eventually affect an employee's health. (Josten et al. 2003, Geurts et al. 2005, Meijman & Mulder 1998.)

Prolonged work stress has been associated with chronic elevated blood pressure, which has been seen as one of the underlying mechanisms to cardiovascular diseases (e.g. Rau 2006, Tsutsumi et al. 2001, Vrijkotte et al. 2000). Work stress has mostly been connected to high blood pressure or hypertension (e.g. Tsutsumi et al. 2001, Cesana et al. 2003, Kawakami et al. 1998, Landsbergis et al. 1994), but also findings of a higher risk of coronary heart disease have been made (e.g. Kivimäki et al. 2006, Kuper & Marmot 2003). However, these associations seem to be partly mediated by other risk factors for cardiovascular diseases, such as smoking, genetic risk for cardiovascular diseases, and negative emotions (Lee et al. 2002). A study about the health status of

21290 female nurses indicated that adverse working conditions (low job control, high job demands, and low work related social support) were associated with poor health status and significant functional declines over a 4-year follow-up period, which could not be explained by age, BMI, alcohol consumption, smoking status, education level, exercise level, employment status, or marital status (Cheng et al. 2000). There is also a risk for developing a burnout state if a prolonged stress situation at work exists. Recent findings among 30–64-year-old Finnish employees (n=3424) indicate that burnout can develop in all kinds of vocational groups and that susceptibility seemed to increase with age (Ahola et al. 2006.) A model of causes and consequences of work-related stress is presented in Figure 1.

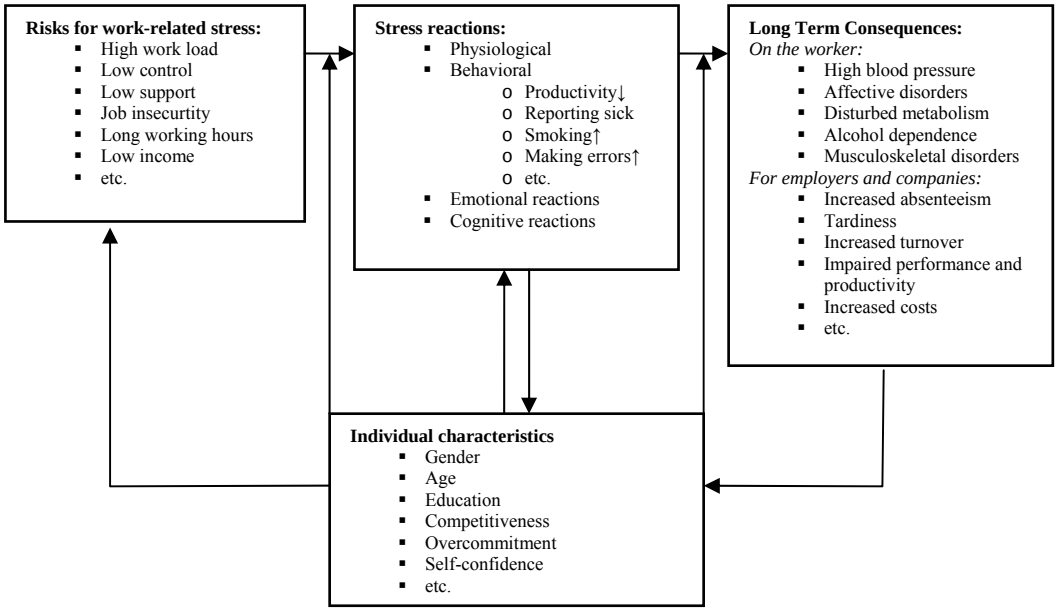

FIGURE 1. Model of causes and consequences of work-related stress (European Foundation for the Improvement of Living and Working Conditions 2005).

The demands of work can also be experienced as a challenge by an employee if work-stressors are in balance with his/her abilities, and this kind of positive stress can have approving effects on well-being (Cavanaugh et al. 2000, Sparks et al. 2001). Occupationally induced fatigue is experienced primarily after a working day. This is not considered to cause problems if enough time for recovery is offered between two periods of work. (Sluiter et al. 1999, Sluiter et al. 2001.) The topic of recovery from work has not, however, received much scientific attention and the process of recovery is

not yet well understood, but it can be viewed as replenishment of both physical and mental resources after fatigue caused by a period of work. (Zijlstra & Sonnentag 2006). Recovery can thus be seen as a combination of physiological and psychological processes, which allow the person to replenish his/her resources through e.g. detachment from work, mental "switch-off", changing activities, as well as rest and sleep (Zijlstra & Sonnentag 2006, Cropley et al. 2006, Rook & Zijlstra 2006, Sonnentag & Kruel 2006). Physiological recovery can be seen as a decreased activity level and possibility to unwind, during which the person can return to a pre-stressor level of functioning (Craig & Cooper 1992). The importance of the parasympathetic system for growth and restoration, i.e. anabolic activities, and the conservation of bodily energy and resting of vital organs has been highlighted by Porges (1995), who further discussed that the autonomic nervous system (for more information, see 3.1) is continuously seeking for balance. According to Porges, the control of physiological stability is primarily mediated by the parasympathetic nervous system. Firstbeat Technologies Oy defines recovery as "the decrease in the activity level of the body in the absence of / decrease in internal and external stressors, such as during relaxation, rest and calm working. Then parasympathetic activity of the autonomic nervous system is predominant." (Handbook of Wellness Analysis Software 1.4.0.14, in Finnish.)

2.2 Importance of night-sleep for recovery

Sleep is one of the most important recovery mechanisms available to humans, allowing recovery from daily stress and strains (Cropley et al. 2006). Sleep causes two major types of physiologic effects, which affect most importantly the nervous system itself, but also other functional systems of the body. It can be assumed that sleep in many ways restores normal levels of brain activity and balance in the central nervous system. (Guyton & Hall 2000.) It is generally known that the quantity and quality of sleep affect the well-being of a person. It has been stated that sleep must be continuous to be restorative (Walsh & Lindblom 2000, Cropley et al. 2006, Ekstedt 2005), and that shortened or fragmented sleep is associated with objective and subjective sleepiness (Bonnet & Arand 2003) and burnout (Ekstedt 2005). It has also been reported that sleep duration above or below the average duration of seven to less than eight hours per night is associated with increased prevalence of hypertension. This increase was more

pronounced in the group that slept less than six hours per night (Gottlieb et al. 2006). Prolonged wakefulness is associated with deterioration of thought processes and even abnormal behavioural activities (Guyton & Hall 2000), learning (Kryger et al. 1989), as well as ability to make correct decisions (Harrison & Horne 2000). Disturbed sleep has been shown to affect endocrinology, immunology and metabolism, and the changes may be linked to diseases, particularly Type 2 diabetes and cardiovascular diseases (Åkerstedt & Nilsson 2003).

Daily sleep duration has, however, been declining in the modern societies over the past decades. For example in the United States, the decline in average sleep duration has been from 8 hours in the 1950's to less than seven hours at present. (Gottlieb et al. 2006, Jean-Louis et al. 2000.) Much of this reduction can be regarded as voluntary, with nearly half of individuals reporting that they restrict night-sleep in order to work, watch television or use Internet (Gottlieb et al. 2006). In Finland, 42% of the working individuals reported in 1997 that they sleep daily at least one hour less than they would need to be fresh (Sallinen et al. 2000). Also aging effects sleep, as normally the quantity and quality of sleep get worse with aging (Walsleben et al. 2004). It has been reported that also subjective sleep quality decreases, and difficulties in awakening and feelings of not being rested after sleep increase with age (Åkerstedt et al. 2002).

High work demands have been associated with sleep disturbances, but despite of that fact, most of the studies in the area of work and health have neglected the topic of sleep (Zijlstra & Sonnentag 2006). There is a lack of studies concerning e.g. spontaneous microarousals during sleep outside the laboratory, which is an important consideration for studies of work stress, sleep and recovery (Ekstedt 2005). Indirect evidence for the harmful effects of stress on sleep comes from studies showing sleep changes immediately after a disturbing life-event, in chronic insomniacs, in shift-workers, in stress-related disorders, or in elderly subjects (Van Reeth et al. 2000). It has been speculated that work stress would affect not only working hours, but also leisure time and the first hours of sleep. Therefore, ambulatory recording during sleep after workday provides an excellent setting to measure recovery processes (Vrijkotte et al. 2000.)

High work demands can keep the arousal level high in the evening or people may have problems in "switching off" from work-related thoughts, and both can cause difficulties

in falling asleep (Zijlstra & Sonnentag 2006). Sluiter et al. (1999) proved that subjective need for recovery after working day was associated with health complaints and sleep quality. Consequently, sleep problems are related to fatigue, causing more need for recovery. If the recovery is insufficient, a vicious cycle can develop, where more effort has to be invested during every working period in order to rebalance a suboptimal psychophysiological state and performance. Therefore, recovery between working periods is essential for avoiding deleterious consequences for mental and physical health in the future. (Sluiter et al. 1999, Sluiter et al. 2001, Rook & Zijlstra 2006, Zijlstra & Sonnentag 2006.)

3 PHYSIOLOGICAL METHODS TO EVALUATE STRESS AND RECOVERY

3.1 Autonomic nervous system (ANS) and stress response

The autonomic nervous system (ANS) can be divided to the parasympathetic (PNS) and sympathetic (SNS) nervous system. These systems help to control many physiological functions, such as cardiovascular function, gastrointestinal emptying, sweating, body temperature and many others. One of the most important characteristics of the ANS is the rapidity and intensity with which it can change visceral function. Normally there is continuous activity in both PNS and SNS, and the basal activity is known as parasympathetic (vagal) and sympathetic tone. This makes it possible for a single nervous system to both increase and decrease the activity of a stimulated organ. (Guyton & Hall 2000.) The basic functions of PNS and SNS are presented in Figure 2.

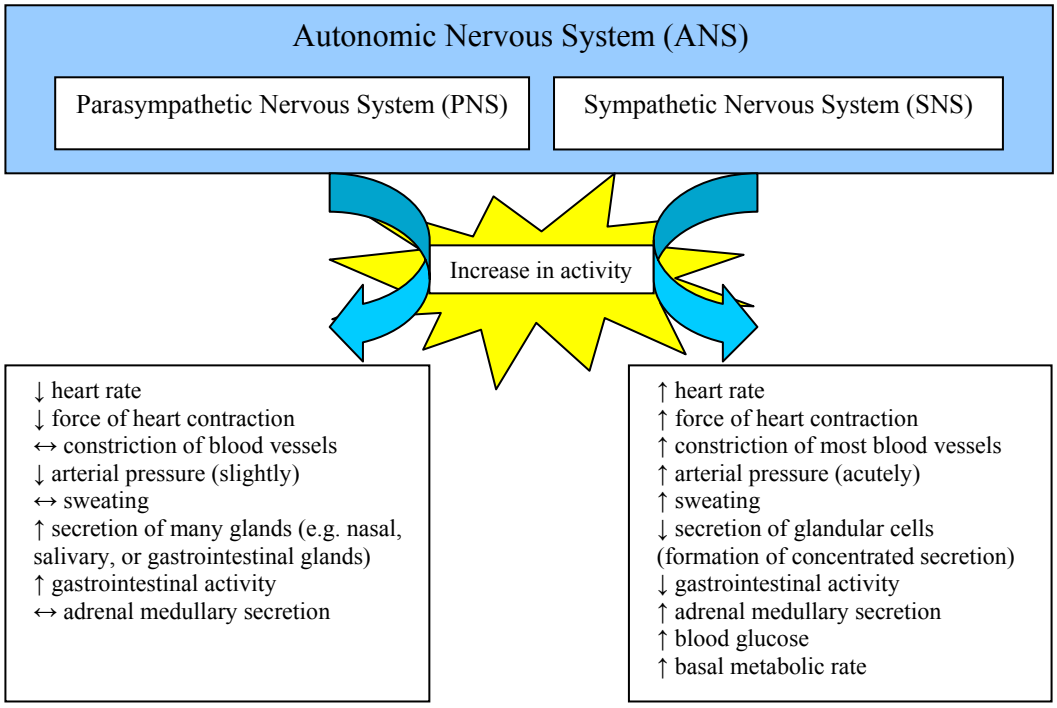

FIGURE 2. Effects of parasympathetic and sympathetic activity on specific organs (based on Guyton & Hall 2000).

Cardiac function is regulated by numerous simultaneous mechanisms for providing substrates to the tissues. The heart has its own intrinsic rhythm (from the sinoatrial node), but it is also regulated via direct neural stimulation and humoral stimulation by catecholamines (epinephrine and norepinephrine in the bloodstream), as well as via autonomic reflexes based on the information from baroreceptors and chemoreceptors, and the vasoconstriction/dilatation caused by chemical circumstances in the tissues. The parasympathetic (vagal) stimulation of the heart is mediated via release of acetylcholine by the vagus nerve, which is responded to by muscarinic receptors causing slowing down of heart rate. The sympathetic nerves release norepinephrine and cause increasing of heart rate and strengthening of heart contraction. (Guyton & Hall 2000.)

The key physiological systems to maintain homeostasis of the body challenged by different stressors include the sympatho–adrenomedullary system (SAM system), the hypothalamic–pituitary–adrenal axis (HPA axis) (for more information see 3.3) along with ANS, as well as cardiovascular, metabolic, and immune systems. The sympathetic branch of ANS promotes the immediate 'fight-or-flight' response to stress along with the SAM system. (Wetherell et al. 2006, McEwen 1998.) The immediate cardiovascular response to a stressful situation is, therefore, an increase in blood pressure and heart rate level in order to support the need for substrates of the organs. The slower humoral response is controlled via the adrenal gland; adrenal cortex secretes corticosteroid hormones (e.g. aldosterone and cortisol) with the main function of affecting electrolyte balance and providing adequate glucose to bloodstream, while the medulla secretes epinephrine and norepinephrine (catecholamines), which function in co-operation with sympathetic nerves to increase sympathetic modulation in the stress response. (Guyton & Hall 2000.) The reactivity to acute stress through cortisol and increased blood pressure seems to be more pronounced in men when compared to women, who seem to react more typically by increased heart rate (Earle et al. 1999).

It is generally known that the parasympathetic branch of ANS is responsible for decreasing the activation level of the body after the stress has ceased, therefore enabling recovery. On the other hand, Porges (1995) has presented a model of the control of stress where parasympathetic activity is responsible for maintaining internal homeostasis of the body and sympathetic activity for responding to external challenges. According to this model, also withdrawal of the parasympathetic tone in response to a

stressful situation may define stress or stress vulnerability and the stress response may occur even without major shifts towards increased sympathetic tone. Stress would, therefore, be defined also as a state of the autonomic system where the vagal tone is decreased. (Porges 1995.)

These described physiological reactions represent attempts to optimize cardiovascular function and substrate availability in order to maintain homeostasis – stress reaction is therefore a useful reaction. The ANS, HPA axis and SAM system have the ability of adjusting the responses to match the external and internal demands at long-term (also called allostasis). These systems have larger boundaries to function within when compared to many other strictly regulated homeostatic systems, such as blood oxygen and pH, or body temperature. Normal stress response is illustrated in Figure 3. Stress causes initiating of adaptive responses, which are "turned on" as long as the stressor is present and then "turned off" when stress ceases, and this is when the recovery occurs. If the magnitude of the response is over- or underestimated or there are problems in turning off the response, stress reactions in the body can become prolonged. Sustained stress reactions can cause changes in the boundaries of function in these systems, or lead to compensatory hyperactivity of other mediators over long-term, therefore causing health problems. (McEwen 1998.)

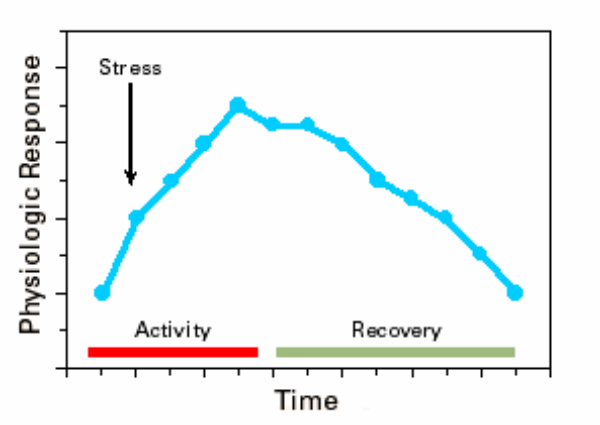

FIGURE 3. Normal stress response (modified from McEwen 1998).

The stress-exhaustion process can be viewed as a three-stage continuum based on Selye's (1946) General Adaptation Syndrome. On the first stage (the alarm reaction), a person reacts to stressors immediately by "fight-or-flight" reaction, which causes the person to be ready for physical action. The second stage is resistance (or adaptation) to

stressors, and during it many physiological systems adapt to the existence of the stressors. If stress prolongs without adequate recovery, the body's resistance may gradually be reduced, and the stage of exhaustion develops. (Selye 1946.) The process from acute stress to exhaustion in autonomic modulation is hypothetically illustrated in Figure 4. Increased sympathetic activity and decreased parasympathetic activity (i.e. lack of recovery) can eventually lead to exhaustion and a chronic state of fatigue. This can cause narrowing of the functional range of the autonomic nervous system probably at least partly through depletion of catecholamine stores (Rusko 2006). This process may get support from a recent animal study, where rats exposed to a stressor (immobilization for 2 h) showed a 39% decrease in catecholamine stores in central and peripheral tissues (Dronjak & Gavrilovic 2006). Moreover, it can be at least speculated that the exhaustion originating from different sources, such as physical stress (e.g. overtraining syndrome) and mental or work-related stress (burnout) can both be preceded by a similar process as described above. This is because both syndromes are associated with similar symptoms, such as an adverse ambulatory profile, e.g. disturbed autonomic modulation of the heart, as well as hormonal and mood changes, even though physiological and psychological symptoms of these syndromes are various and not so well-defined (reference for overtraining e.g. Gleeson 2002, Angeli et al. 2004, and for burnout e.g. De Vente et al. 2003, Ekstedt 2005).

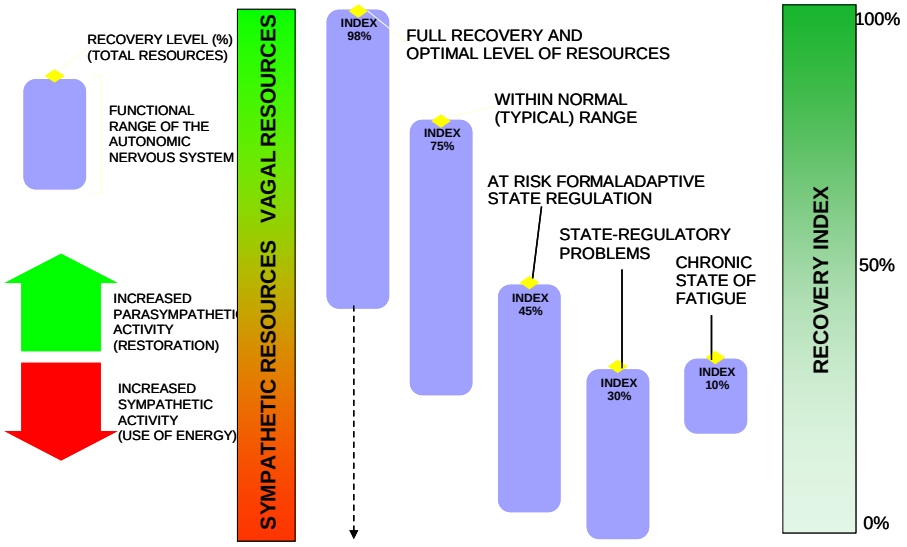

FIGURE 4. Lack of recovery and increased sympathetic activity can lead to exhaustion and chronic state of fatigue, and cause narrowing of the functional range of ANS (Rusko 2006).

3.2 Heart rate variability

Heart rate variability (HRV) means the variations of both instantaneous heart rate and R-R intervals. HRV represents one of the most promising markers of cardiac autonomic activity. (Berntson et al. 1997, Task Force 1996.) The clinical significance of HRV became appreciated in the late 1980's, when it was established that HRV was a strong and independent predictor of mortality after an acute myocardial infarction. (Task Force 1996.) Several studies have shown that decreased HRV is associated with cardiovascular morbidity and mortality (Bigger et al. 1993, Dekker et al. 1997, Tsuji et al. 1996). Numerous factors have been reported to affect HRV, such as age, sex, health status, lifestyle and nutritional factors. Older age, sedentary habits, obesity, hyperglycemia, insulin resistance, coronary artery disease, hypertension, elevated fatty acids, and pollution have been shown to be associated with decreased HRV, but exercise training and improved physical condition mostly with increased HRV. (Singh et al. 2003, Achten & Jeukendrup 2003.) Given that, it is obvious that there is significant individual variation in heart rate variability, which should be taken into account when interpreting it.

Spontaneous HRV is thought to have its background in three physiological origins: regulation of respiration, blood pressure, and body temperature (Perini & Veicsteinas 2003), and at rest HRV represents the fine tuning of the beat-to-beat control mechanism of cardiovascular function (Task Force 1996). It has also been shown that breathing has a significant influence on autonomic neural outflow – fluctuation in beat-to-beat HR is related to respiration because inspiration inhibits parasympathetic modulation (Hautala 2004, Task Force 1996). It should be noticed that HRV measures fluctuations in autonomic inputs rather than the mean level of autonomic inputs to the heart (Task Force 1996.)

The variations in heart rate can be analyzed by a number of methods, including time and frequency domain methods, statistical and geometrical methods, and nonlinear methods. First each QRS complex is detected in a continuous ECG record, and the so-called normal-to-normal (NN) intervals (intervals between adjacent QRS complexes resulting

from sinus node depolarizations) or the instantaneous heart rate is determined. (Task Force 1996.)

Simple time domain methods include for example mean R-R interval and mean heart rate. The standard deviation of the NN intervals (SDNN) expresses an estimate of overall HRV, and the square root of the mean squared differences between successive NN intervals (RMSSD) expresses short-term variations in heart rate. (Task Force 1996.) RMSSD is highly correlated with the frequency domain variable high frequency power (HFP, 0.15-0.40 Hz), and they are both widely accepted as a measure of parasympathetic activity (Berntson et al. 1997, Task Force 1996). The low frequency power of HRV (LFP, 0.04-0.15 Hz) has been more controversial, but mostly it has been connected to both sympathetic and parasympathetic activity of ANS. The ratio of LF to HF (LF/HF) has been used to express the sympathovagal balance (Malliani et al. 1991, Malliani 2005), but criticism considering the existence of this kind of balance has been presented (e.g. Eckberg 1997). The third main spectral component in the frequency domain is the very low frequency power (VLF, ≤0.04 Hz). Its physiological explanation is less defined and the existence of its physiological background might even be questioned. Total power (TP, ≤0.4 Hz) expresses variance of NN intervals over the measured period. (Task Force 1996.) It has been shown in a vagal-blockade study that all HRV, especially HF power, is under parasympathetic control (Martinmäki et al. 2006). The measurement of VLF, LF, and HF is usually made in absolute values, but LF and HF can also be calculated in normalized units, which represent the relative value of each power component in proportion to the total power minus the VLF component (Task Force 1996). To reach the normal distribution of the spectral power values, transformation with natural logarithm (ln) is sometimes applied.

3.2.1 Circadian rhythm for heart rate and heart rate variability

There is significant circadian variation in heart rate (HR), blood pressure (BP), and activity of the autonomic nervous system in humans. Numerous studies have reported circadian variation of HR and HRV (Beckers et al. 2006, Wennerblom et al. 2001, Ramaekers et al. 1998, Carrington et al. 2003). HR and BP are lower during night than during daytime and studies of 24-hour HRV indicate that daytime is associated with

sympathetic dominance, while parasympathetic activity dominates the night. (Carrington et al. 2003.) HR and LF/HF decrease during the night, and HF power as well as LF power in absolute values seem to increase (Wennerblom et al. 2001, Ramaekers et al. 1998). Even though the absolute value of LF increases, its relative contribution decreases during the night, again expressing parasympathetic dominance (Beckers et al. 2006). There are numerous factors which affect this circadian variation, including activity level, posture, sleep-wake-cycle, and light exposure. (Carrington et al. 2003.) Ito et al. (2001) reported that HRV variables and R-R interval exhibited similar patterns during day and night shift in nurses and that they were dependent on activity levels. Variables related to sympathetic control (LF and LF/HF ratio) were the greatest during working period and the smallest during sleep and the opposite was found in variables related to parasympathetic control (HF power). Therefore, autonomic cardiac function seems largely to be under the influence of sleep-wake cycle rather than an internal clock.

3.2.2 HRV and stress

Heart rate variability in relation to stress has been investigated in laboratory settings during short-term acute stress tasks and in ambulatory measurements concerning longer-term job strain or work stress. It has been reported that mental stress in laboratory conditions increases HR and decreases HRV. Shapiro et al. (2000) found that a mental arithmetic task increased HR and decreased HF power. Isowa et al. (2006) reported increased HR, BP, and LF/HF ratio, and decreased HF power during a stress task compared to baseline and control group, indicating increased sympathetic modulation. Guasti and co-workers (2005) reported increased LF power and decreased HF power during stress. Hjortskov et al. (2004) found a reduction in HF power and an increase in BP and LF/HF ratio during computer-work related stressors, but no changes in LF power. The researchers concluded that HRV is a more selective and sensitive measure of mental stress than BP, as BP remained elevated during the recovery period.

Garde et al. (2002) found that an increase in HRV variables expressing sympathetic modulation and a decrease in variables expressing parasympathetic modulation were found in response to a computer task, but no additional effect were elicited on these

variables via addition of mental demands to the task (Stroop color-word test). Recently it has been shown that an arithmetic stress task with verbalization of the answers was associated with an increase in HR, which was achieved trough decreased or, surprisingly, increased modulation in high-frequency (HF) variation. This could reflect the influence of breathing pattern or possibly different co-activation mechanisms of the system. It was concluded that it might be possible that regulation of HR upon mental stress goes through decreased parasympathetic activity but also through an additional anti-adrenergic effect, which are both reflected in high-frequency variation. (Vuksanovic & Gal 2006.) Hall et al. (2004) found that acute psychophysiological stress, which was a standard speech task paradigm, used to elicit acute stress in the immediate pre-sleep period, was associated with decreased levels of HF power during non-REM-sleep and REM-sleep and increased levels of LF/HF ratio during non-REM-sleep. It was concluded that changes in HRV associated with acute stress may represent one pathway to disturbed sleep. According to the study, stress-related changes in HRV during sleep may also be important in association with chronic stressors.

Results which indicate that work stress decreases parasympathetic modulation and increases sympathetic modulation of the heart have also been reported in ambulatory field studies. Vrijkotte et al. (2000) reported that subjects with a high imbalance in effort-reward scale (based on Siegrist's model of work stress) had lower RMSSD on both work and rest day. The study showed that the adverse effects of work stress are partly mediated through increased HR reactivity to a stressful workday, decreased 24-h parasympathetic tone, and increased systolic blood pressure. Most importantly, it was also shown that RMSSD during night-sleep was more predictive of mild hypertension than values during the day. Pichot et al. (2002) also found a large and significant decrease in the total heart rate variability during sleep, especially in the high frequency component of HRV, during a 3-week working period, followed by a rebound in HRV during a resting week. The authors concluded that nocturnal autonomic nervous system activity reflected cumulated physical fatigue and progressive parasympathetic withdrawal during the working period, followed by recovery during the resting period. Lucini et al. (2002) found that mild real life stress (university examinations) raised BP and HR, increased LF power and LF/HF ratio, and decreased HF power, but also elevated salivary cortisol levels (for more information about cortisol see 3.3) on a stress day. Aasa et al. (2006) observed significant differences in HF and LF power in a group

of ambulance personnel with many health complaints between work day and a day off. These differences were mostly due to increased LF and decreased HF power late in the night and in the morning of the workday.

Collins et al. (2005) made a recording of 48 hours and reported that job strain did not predict total HRV (measured by SDNN), but decreased HF power was observed in the high job strain and low control work groups. In the low control group, also reduction in SDNN was observed. Lucini et al. (2005) reported that patients suffering chronic psychosocial stress had elevated LF and LF/HF, and decreased HF at rest, in addition to blunted orthostatic reactions and reduced baroreflex sensitivity. These might represent disadvantageous autonomic changes, which may be a part of the underlying mechanisms of the complex physiological changes in the increased cardiovascular risk related to chronic stress. Conversely, Riese et al. (2004) studied effects of job strain on ambulatory BP, HR and HRV in 159 healthy nurses, and found that job strain was not associated with these variables. It was concluded that high job strain among young female nurses is not connected to an unfavourable ambulatory cardiovascular profile.

3.3 Cortisol

3.3.1 Physiology and circadian rhythm of cortisol secretion

Cortisol is the most important glucocorticoid of the human body secreted by adrenal cortex. The secretion is regulated by adrenocorticotropic hormone (ACTH), which is secreted from the pituitary gland and stimulated by corticotropin-releasing hormone (CRH) from the hypothalamus. Cortisol inhibits the synthesis of both ACTH and CRH, and therefore maintains a balance in hypothalamic-pituitary-adrenal axis (HPA axis) with a negative feedback system. (Bartels et al. 2003.) After the secretion of cortisol, most of it binds to plasma proteins (mainly to corticostreroid binding globulin, CBG, but also albumin) and only about 6% of the cortisol is free and biologically active. The binding proteins restrain cortisol from entering its target cells (Lewis et al. 2005, Bartels et al. 2003), but free cortisol is able to attach to the receptors on the cells and affect their function (Guyton & Hall 2000). Cortisol is mainly metabolized in the liver and the most important physiologic functions of cortisol include (Guyton & Hall 2000):

- Increasing the catabolic part of lipid and protein metabolism
- Stimulation of gluconeogenesis and decreased glucose utilization by the cells
- Antagonism to insulin
- Inhibition of inflammatory and allergenic reactions

Therefore, cortisol affects the metabolic reactions in the cells and the immune system. Excess activity or dysregulation of the HPA axis has been associated with serious health problems such as cardiovascular diseases, Type 2 diabetes, and impaired function of the immune system and cognitive function. Increased cortisol levels cause increased obesity in abdominal region and insulin resistance. (Rosmond 2003.) Dysfunction of HPA axis has been reported in overweight persons and persons suffering anorexia nervosa (Putignano et al. 2001).

Cortisol can be measured in plasma, urine, and nowadays also in saliva. These measures are highly intercorrelated. (King & Hegadoren 2002, Aardal & Holm 1995.) Cortisol in saliva reflects the functional free fraction of cortisol (Bartels et al. 2003, Kirschbaum & Hellhammer 1994). Cortisol measurement in saliva is a great step forward for work stress research because saliva samples can be taken non-invasively, frequently, and without any excess stress (Putignano et al. 2001, King & Hegadoren 2002). Free salivary cortisol reflects plasma and serum cortisol and it has been shown to have the same diurnal rhythm, which typically declines throughout the waking day (Stone et al. 2001, Ice et al. 2004). However, great individual variation in the diurnal rhythm of salivary cortisol has been reported in younger (Stone et al. 2001) as well as older adults (Ice et al. 2004). Typical for secretion of cortisol are, however, 10 to 15 secretion pulses within 24 hours and it is regulated with a clear on-off pattern (Bartels et al. 2003, Stone et al. 2001). Cortisol values are generally highest in the morning, declining during the day, and the lowest at midnight, after which they start to increase rapidly during the first couple of sleeping hours. This circadian rhythm is linked to sleep-wake and light-dark rhythms. (Bartels et al. 2003.) Cortisol values increase remarkably immediately after awakening in healthy adults. For example, saliva cortisol increases 50–160% during the first 30 minutes after awakening, and this has been assumed to be equal to three secretion pulses (Clow et al. 2004.)

3.3.2 Cortisol in stress research

Secretion of cortisol reaches its peak 30 minutes after an exposure to acute stress (Lundberg 2005). Endocrinological responses caused by different stressors have been studied extensively. Increased cortisol values caused by a Trier Social Stress Test (TSST), which has been used as a standard psychological stress test, have been thought to describe the magnitude of the stress reaction (Kudielka et al. 2004a). Cortisol has been used a lot as a physiological measure of work-related stress (Lundberg & Hellström 2002, Grossi et al. 2005). Several different cortisol variables and study approaches have been used in examining the connections between stress exposure and cortisol secretion, including various time points for sampling. Elevated evening cortisol, elevated 24-h cortisol, elevated overnight and morning cortisol, as well as suppression of 24-h cortisol levels have all been presented as potential candidates for markers of stress (Powell et al. 2002).

The most studied cortisol variable has lately been cortisol awakening response (CAR), which means the increase in cortisol values after awakening (e.g. Pruessner et al. 1997, Clow et al. 2004). In the calculations of awakening response, the difference between awakening value from the values shortly after awakening (e.g. 30 or 60 minutes after) has mostly been used, but also areas under the cortisol curve (AUC) as recommended by Pruessner et al. 1997. The cortisol awakening response has been stated to be a discrete and distinctive part of the cortisol circadian cycle (Clow et al. 2004). It has been suggested to be associated with the mobilization of energy stores during the transition from sleep to wake state (Pruessner et al. 1997) and with the function of the immune system (Petrovsky & Harrison 1997). Increases in cortisol after awakening with respect to the awakening value have been thought to indicate short-term reactivity to stress, and cortisol with respect to ground total hormonal output (both measured in areas under the curve) as presented by Pruessner et al. (2003a).

The magnitude of cortisol awakening response has been associated with psychosocial variables, stress, and health (Clow et al. 2004), as well as chronic stress, work stress, and depression (Pruessner et al. 2003b, Kunz-Ebrecht et al. 2004, Schlotz et al. 2004, Williams et al. 2005). Attenuated awakening responses have been reported in weekend

compared to weekday (Kunz-Ebrecht et al. 2004, Schlotz et al. 2004). Cortisol awakening response has also been reported to be linked to e.g. gender (Wüst et al. 2000, Pruessner et al. 1997), light (Leproult et al. 2001), awakening time (Federenko et al. 2004, Edwards et al. 2001), and duration of sleep (Schlotz et al. 2004, Späth-Schwalbe et al. 1992). On the other hand, Wüst et al. (2000) reported that cortisol awakening response is not affected by age, use of oral contraceptives, habitual smoking, time of awakening, total duration of sleep or using/not using an alarm clock. The importance and role of cortisol awakening response is thus under vigorous research (Clow et al. 2004). However, the intraindividual stability of cortisol awakening response over time has been shown to be remarkably high with correlations up to r=.63 (for the area under the curve) (Wüst et al. 2000).

(The cortisol secretion in response to stress seems, however, to be a complicated phenomenon, as indicated by the conflicting results of earlier studies. Increased cortisol values in the morning have been found in burn-out subjects compared to controls (De Vente et al. 2003, Pruessner et al. 1999), but long-lasting psychological stress has also been reported to decrease cortisol values, indicating decreased ability of the body to respond to stimuli (Zarkovic et al. 2003). Hyperactivity of the HPA axis including excess release of cortisol has been seen during chronic stress, while HPA axis hypoactivity with a decrease in cortisol release has been reported in chronic fatigue syndrome and after chronic stress (Kudielka et al. 2006, Gaab et al. 2002). It seems that there is no consensus about the interpretation of cortisol results in the scientific world currently.

3.4 Sleep analysis and subjective ratings of recovery

Sleep is a cyclical process, which alternates with wakefulness and consists of different stages, roughly divided to REM-sleep (rapid eye movement) and NREM-sleep (non-rapid eye movement) (Ekstedt 2005, Härmä & Sallinen 2004). NREM-sleep is further divided to stages 1-4, where the deepness of sleep increases toward stages 3 and 4, which are termed Slow Wave Sleep (SWS). SWS is considered to be the most important phase of sleep for its restorative functions, such as cerebral recovery and secretion of growth hormone, and it is mostly slept during the first 4-5 hours of sleep. (Härmä &

Sallinen 2004, Ekstedt 2005.) There is also a close and strong temporal association between sleep-wake states and activity of the HPA axis and sympathetic system. Sleep onset is associated with decreasing cortisol levels and sympathetic activity, but on the other hand, REM-sleep and awakenings from sleep are followed by a pulse of cortisol secretion. Various components of the immune system (e.g. natural killer cell activity, B and T lymphocytes, cytokines) have an impact on sleep structure, and these components are affected by acute and chronic stressors. (Van Reeth et al. 2000.)

Actigraphy is a method developed to assess sleep-wake patterns and circadian rhythms by measuring movement, most commonly of the wrist, and it has been used for over 20 years (Ancoli-Israel et al. 2003, Littner et al. 2003). It has been proven to be a valid and reliable method to assess sleep in normal and healthy adult populations, but less accurate for detecting sleep when sleep becomes more disturbed. Actigraphy may overestimate sleep and underestimate wake especially during the day compared with polysomnography (Ancoli-Israel et al. 2003.) Actigraphy has also been used to measure daytime physical activity (Kario et al. 1999, Chen & Bassett 2005), but mostly in children (Kelly et al. 2004).

Polysomnography (PSG), which is based on electrical brain activity, is considered the golden standard to measure sleep (Ekstedt 2005, Signal et al. 2005, Tryon 2004). However, the use of actigraphy to identify sleep-wake pattern and to assess sleep disorders has been critically evaluated and accepted (An American Sleep Disorders Association Report 1995, Tryon 2004). Wrist actigraphy is a non-invasive, easy-to-use and low-cost method in ambulatory setting, so it has become widely used (Signal et al. 2005). When comparing PSG and actigraphy, it should be noticed that sleep onset is a gradual process. It has been shown that actigraphy detects sleep onset systematically earlier than PSG because actigraphy is based on movement and not on electrical activity of the brain, thus evoking overestimation of the amount of sleep (Signal et al. 2005, Tryon 2004). When reviewing the role of actigraphy in studying sleep and circadian rhythm, Ancoli-Israel and co-workers (2003) found that compared with PSG, actigraphy is valid for assessing sleep durations and sleep/wake activity, but less reliable for more specific measures such as sleep offset or efficiency. According to them, no “first night effect” has been found with actigraphy in healthy adults.

In addition to objective measurements of night-sleep, it has been reported that sleep quality assessed by subjective ratings gives also valuable information about recovery. As a matter of fact, Pilcher (2000) found that sleep quality reflected sleepiness during daytime better than sleep quantity. It has also been reported that sleep quality is better related to measures of health, well-being, and negative emotions than sleep quantity in a group of college students sleeping seven to eight hours per night (Pilcher et al. 1997). Rook & Zijlstra (2006) reported that subjective sleep quality emerges as important for recovery as sleep quantity, and the quality predicts recovery by reducing the experience of fatigue.

Blagrove et al. (1998) found a correlation of -0.26 between movement index (during both daytime and night-time sleep) assessed by actigraphy and subjective sleep quality. It was concluded that actigraph-detected movement frequency is a reliable, even though weak, indicator of subjective sleep quality and efficiency. Lockley et al. (1999) found good correlations between actigraphy and sleep logs when sleep timing and duration (sleep onset, sleep offset, night sleep duration, daytime nap duration) were compared, but poorer correlations in their assessment of transitions between sleep and wake states (sleep latency, number and duration of night awakenings, number of daytime naps).

Tworoger et al. (2005) reported that going to bed late, medication use, employment, increased daylight hours, longer menstrual cycle length, and higher BMI were associated with poorer actigraphic sleep measures in 20 to 40-year-old women. However, subjective and objective (actigraphy) sleep pattern differed, as having a job and perceived stress was associated with subjectively but not objectively poorer sleep. The authors suggested that both subjective and objective aspects of sleep should be assessed because these two are only modestly correlated (Baker et al. 1999, Vitiello et al. 2002, Vitiello et al. 1997) and may reflect different aspects of the quality of sleep (Tworoger et al. 2005). It was also stated by Tworoger et al. (2005) that subjective sleep reports may partly reflect an individual's perspective or state of mind in addition to some component of their objective sleep patterns. Kushida et al. (2001) also suggested using both subjective and actigraphic measurements compared to PSG.

In the review by Ancoli-Israel et al. (2003), a question was raised about the meaning of subjective and actigraphic analysis of sleep. If subjective reports such as sleep logs are

considered more important (in a sense that it does not matter what the objective methods show, if a person feels that he/she is sleeping well), then there is no need for PSG or actigraphy (since subjective method may not correlate highly with objective methods, and the latter are also time and effort consuming). If, however, objective measurements are preferred, then actigraphy is less expensive and much easier to perform than PSG, and recording of multiple days and nights is possible. (Ancoli-Israel et al. 2003.)

4 RESEARCH QUESTIONS AND HYPOTHESES

It has been shown that the physiological stress reaction involves increased activity of the sympathetic nervous system and decreased parasympathetic modulation, along with increased SAM system and HPA axis activity. As a result, the amount of catecholamines and cortisol in the circulation is increased, and heart rate and blood pressure are elevated. These physiological reactions are useful in short-term but may chronically lead to illnesses such as cardiovascular diseases via sustained arousal of central and peripheral systems. (e.g. Porges 1995, McEwen 1998, Lovallo 2005.) Stress caused by work has been connected to changes in the above-mentioned systems but also to disturbances during sleep (Van Reeth et al. 2000, Åkerstedt et al. 2002, Zijlstra & Sonnentag 2006). Awakenings from sleep have also been associated with a burst of cortisol secretion (Späth-Scwalbe et al. 1991, Dahlgren et al. 2005). Therefore, higher levels of stress, increased level of arousal of physiological systems (e.g. increased SNS and HPA axis activity, and decreased PNS activity), sleeping problems, and psychological changes related to stress are interrelated, but scientific knowledge about the exact mechanisms behind these changes and causal links between these variables are still limited, especially when ambulatory real life measurements are concerned. It seems obvious, however, that recovery during the cycle of work and rest, and especially during night-sleep, is essential to maintain mental and physical health, overall well-being, and the ability to work (e.g. Vrijkotte et al. 2000, Ekstedt 2005, Zilstra & Sonnentag 2006).

It has been shown that HRV offers an excellent tool to non-invasively study the function of ANS. Using different HRV variables it is possible to get information about the two branches of ANS and of their balance, which is a key to elucidate the control of stress and recovery. However, the inter-individual differences in HRV values are high, and therefore, the traditional HRV variables have only modestly been associated with stress experienced by a person. There clearly exists a need for a method which analyzes HRV intra-individually. In order to further improve the use of HRV in studying the prevailing state of the body, Firstbeat Technologies Ltd (www.firstbeat.fi) has developed a method which takes into account the individual level of HRV in the analysis of the ANS function. This new method (FBT-PRO), developed for work-

related research and occupational health assessments, calculates some variables of stress and relaxation of the body based on HR and HRV variables from ambulatory R-R interval recordings. The purpose of the present study was, therefore, to evaluate this new method in assessing stress and recovery during awake and sleep, during sleep on workdays and on a day off, and during better and worse workday sleep. This categorization to better and worse sleep was done based on criterion variables, which were subjective ratings, movement-based sleep analysis, and cortisol – all widely used physiological methods to evaluate work stress and recovery.

It was predicted, based on extensive number of studies (e.g. Beckers et al. 2006, Carrington et al. 2003, Ito et. al. 2001, Wennerblom et al. 2001, Ramaekers et al. 1998), that during awake time HR is elevated and sympathetic modulation increased compared to sleep phase. Therefore, it was also predicted that there would be differences in HR and HRV variables between awake and sleep, and that there would be differences in stress and recovery detected during these time periods, i.e. that there would be more relaxation during sleep and more stress during awake.

It is known that people tend to delay and prolong the sleep during weekend and reduce their sleep time during the week (Valdez et al. 1996). On the other hand, it has been shown that respite during the weekend may not necessarily affect positively the feelings of fatigue at the beginning of the working week (usually Monday morning) (Rook & Zijlstra 2006). However, it was hypothesized that there would occur a compensation of work stress after working period so that more recovery and less stress would be detected, and that the quality of the sleep would be better during a day off than a workday sleep.

Based on previous studies, it seems that sleep should be continuous and long enough to be restorative (Walsh & Lindblom 2000, Cropley et al. 2006, Ekstedt 2005). Arousals during sleep and fragmented sleep are reported to be associated with sleepiness during awake-time (Bonnet & Arand 2003), and increased HPA axis and sympathetic nerve activity (Van Reeth et al. 2000). Despite of contradictory findings, higher cortisol levels at awakening and greater cortisol awakening responses have mostly been connected to higher level of work-related stress (Clow et al. 2004, Eller et al. 2006). Given that, it was hypothesized in the present study that subjectively better night sleep, longer sleep,

and less fragmented sleep would be better for recovery during a working week. It was also predicted that sleep followed by lower awakening cortisol and lower cortisol awakening responses would include more relaxation and less stress than sleep which was followed by greater cortisol values.

Research questions

The specific research questions to be answered in this master's thesis were the following:

1. Is it possible to detect relaxation and stress from HR and HRV during sleep and awake?
2. Are there differences between workday sleeps and between workday and day off sleeps in detected stress and relaxation?
3. Are there differences in detected stress and relaxation between better and worse workday sleeps after categorizing the sleeps with subjective ratings of the quality of sleep, movement-based sleep analysis variables, and cortisol variables?
4. Do the traditional HRV variables give similar information than the new stress and relaxation indices during sleep in the above-mentioned study approaches?

Research hypotheses

The specific hypotheses for the research questions described above were as follows:

Question 1:

Work-hypothesis: It is possible to detect stress and relaxation from HR and HRV so that there is more stress than relaxation during awake-time and more relaxation and less stress during sleep.

0-hypothesis: There are no differences in HR and HRV –based stress and relaxation between awake and sleep.

Question 2:

Work-hypothesis: There is more relaxation and less stress during a day off than during workday 2 sleep.

0-hypothesis: There are no differences in detected stress and relaxation between workday 2 and day off sleep.

Question 3:

Work-hypothesis: More relaxation and less stress is detected in better than in worse workday sleep after categorization based on subjective ratings, sleep analysis variables, and cortisol variables.

0-hypothesis: Detected stress and relaxation do not differ between better and worse workday sleep.

Question 4:

Work-hypothesis: The traditional HRV variables which reflect the function of the parasympathetic branch of ANS (HF, RMSSD) and total HRV (SDNN, LF+HF) give similar information than the new HR and HRV –derived variables related to relaxation state.

0-hypothesis: The traditional HRV variables do not give similar information than the new variables related to relaxation state.

5 METHODS

5.1 Subjects

There were 19 subjects in this study, of which 18 were women. All of the subjects were employees from the Central Hospital of Central Finland and most of them were nurses. We wanted to have high-quality heart rate data in the final analysis, and therefore, only subjects who had less than 5% of error in the HR data were accepted. There were different subjects to match this demand of high-quality HR data in different measured time periods, so all of the subjects are not the same in different study approaches represented later on. After these strict criteria, there were 14 subjects who had high-quality recordings from the three days and who were used in the analysis. They all were women. More exact details of the subjects are shown in Table 1. The subjects were 44±10 years old; their height was 163±5 cm, and weight 63±10 kg.

TABLE 1. Background information of the subjects.

Subjects (n=14)	Average	SD
Age (years)	43.6	9.5
Height (cm)	162.8	5.1
Weight (kg)	62.6	9.9
Body Mass Index (weight/height^2)	23.7	4.2
Waist Circumference (cm)	76.8	8.8
Predicted VO_2 value (ml/kg/min)*	34.3	6.2
Physical Activity Class (0-7)**	4.9	1.6

* Predicted maximal oxygen uptake based on a maximal undirect bicycle ergometer test.
** Activity class based on Ross & Jackson (1990).

As the main focus of the study was to evaluate recovery during sleep, some background information about the subjects' normal sleeping habits during the previous month was questioned with a questionnaire. One subject reported having had abnormal tiredness weekly, three subjects less frequently, and 10 subjects never during the previous month. Two subjects reported having had abnormal insomnia weekly, seven subjects less frequently, and five never during the past month. None of the subjects reported having had these aforementioned symptoms daily.

5.2 Study design

This study was a part of a larger research project ”Heart Rate and Work Stress”, currently in progress at the University of Jyväskylä. The study was completed as a so-called daily-stress design. Two workdays (workday 1 and workday 2) and a day off were recorded. The recorded days were planned so that workday 1 was the first day in a working period (after the day-off days or weekend) and workday 2 was three to four days later at the end of working period. Because all of the subjects did not have long enough working periods, for some of the subjects there was only one day between workday 1 and 2. The study design is described in Figure 5. The measurements were carried out between January and May in 2005.

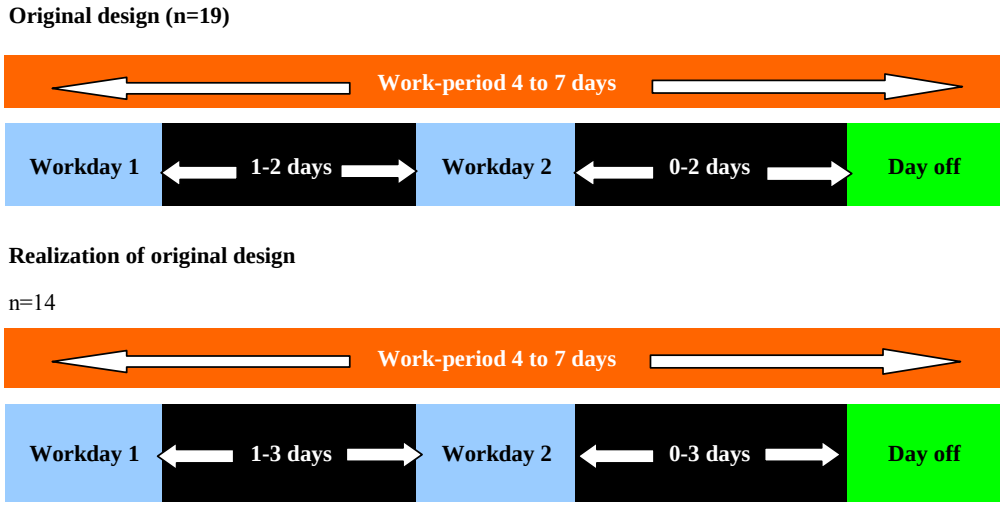

FIGURE 5. Study design.

5.3 Measurements

5.3.1 Heart rate measurements

Heart rate was measured from the subjects for approximately 36 hours with RR-recorder heart rate monitors (Polar Electro, Oulu, Finland) that detect the R-R peaks with an accuracy of 1 ms. Heart rate measurement was started the previous evening prior to the

study day in the subjects' homes or at their workplace. Before starting the measurement the electrodes were dowsed with water, and then the heart rate belt was fitted tightly around the subject's chest. Then the RR-recorder software was set to recognize the R-peaks from the subject's ECG-signal. The RR-recorder was set to measure heart rate data till the morning after the day of interest (workday 1 or 2 or day off). Heart rate and activity recordings (see below) were started at the same time and later adjusted timely by markers set to both devices.

5.3.2 Movement measurements and sleep analysis

In addition to heart rate also movement of the subjects were monitored. In this study, the Actiwatch activity monitoring system® (Cambridge Neurotechnology Ltd, Cambridge, UK, www.camntech.com), which is a small watch-like device, was used. Actiwatch measures activity by the means of a piezo-electric accelerometer which is set up to record the amount, integration of intensity, and duration of movement in all directions. The movement produces an electric current which is converted to activity counts and stored in the memory of the Actiwatch. The maximal sampling frequency is 32 Hz. The Actiwatch unit measures all movement which is greater than 0.05 g and filters values outside the range of 3-11 Hz to eliminate gravitational artefacts. (Cambridge Neurotechnology.)

The subjects wore an Actiwatch in a dominant wrist and it was positioned using a standardized protocol. The sampling epoch of activity counts was set to 0.25 minutes (15 sec). Actiwatch activity & sleep analysis 5 -software (ASA, version 5.32) was used to analyze the night sleep of the subjects. ASA analyzes several variables to describe night sleep using a scoring algorithm for the period between sleep start and end times. In this study, we chose the following variables for further analysis: actual sleep time, sleep efficiency and the fragmentation index. Actual sleep time means the amount of sleep as determined by the algorithm. Sleep efficiency is the percentage of time spent asleep whilst in bed. The fragmentation index is used as an indicator of restlessness; it is the addition of the percentage of time spent moving and the percentage of immobility phases of one minute. (Cambridge Neurotechnology.) Before the analysis, the bed and

get up times had to be set based on the subject's sleep diary. The sensitivity of the analysis was set to medium.

5.3.3 Saliva sampling

The subjects gave three saliva samples during each examined day for the determination of cortisol. Saliva samples were taken with a cotton-chew salivette which was chewed for 1–2 minutes. The instructions for the subjects for how to perform the saliva sampling can be found in appendix 4 (in Finnish). The time-points for sampling were the following:

1) Immediately after wake-up in the morning
2) 30 minutes from wake-up
3) 60 minutes from wake-up

5.3.4 Subjective ratings of the quality of sleep

The subjects filled a diary for the measured period where they wrote their daily routines, i.e. their doings during the day. Subjective ratings about the quality of sleep in the morning after waking up were asked in the diary. A 3-level scale was used (1 = slept well, 2 = slept moderately, 3 = slept badly). The subjects were also asked to report their going-to-bed and wake-up times in this diary.

5.4 Data analysis

5.4.1 Firstbeat PRO and accomplishment of wellness analysis

Physiological variables describing exercise, stress and recovery were analyzed with Firstbeat PRO Heartbeat Analysis Software version 1.4.1 (FBT-PRO, www.firstbeat.fi), based on Heart Rate (HR) and Heart Rate Variability (HRV) indices calculated from ambulatory R-R interval (RRI) data over one day. In addition to HR and HRV, the

software calculates and takes into account respiratory frequency as well as on- and off-response phases (on/off-dynamics) when the response times of HR and VO_2 are known to be different (Bernard et al. 1997, Davies et al. 1972, Pulkkinen et al. 2004). All this information is obtained from HR and HRV indices calculated from the ambulatory RRI data. The individual input parameters of the software are age, sex, weight, height, smoking habits and physical activity class (Ross & Jackson 1990). From these parameters the software predicts maximal HR (ACSM, 2001) and maximum oxygen uptake (VO_{2max}, Jackson et al. 1990, Ross & Jackson 1990). The software uses 60 beats/min as a resting heart rate but the resting and maximal HR are automatically updated from the recorded data if smaller resting or higher maximal HR is observed.

First, the recorded RRI-data are scanned through an artefact detection filter to perform an initial correction of falsely detected, missed and premature heartbeats. The consecutive artefact-corrected RRI's are then resampled at a rate of 5 Hz by using linear interpolation to obtain equidistantly sampled time series (for more detailed information, see Saalasti 2003).

FBT-PRO software calculates second-by-second HRV indices using the short-time Fourier Transform method (STFT), a generalization of the stationary Fourier into non-stationary time series analysis (Oppenheim & Schafer 1989, Saalasti, 2003), and HR- and HRV-derived variables that describe respiration rate, on/off dynamics and oxygen consumption (VO_2) using neural networks (Saalasti 2003, Kettunen & Saalasti 2004, 2005). The spectrum of HRV signal reveals two major components of spectral power which are important for analyzing exercise, stress and recovery: low frequency power (LFP, 0.04–0.15 Hz) and high frequency power (HFP, 0.15–1.00 Hz); the HFP frequency range corresponds to the respiratory frequency range that can be observed at rest and during exercise (Martinmäki et al. 2006, Perini et al. 1990, Perini & Veicsteinas 2003, Tulppo et al. 1996). The software determines the respiratory frequency from the time-frequency distributions based on the frequency distribution of the HFP (Saalasti 2003) and the on/off-dynamics based on the changes in HR and HRV (Kettunen & Saalasti 2005, Saalasti 2003). The calculation of instantaneous VO_2 takes into account HR, respiration rate and on/off dynamics (for more information, see http://www.firstbeat.fi/files/VO2_Estimation.pdf). The software first calculates the percentage from maximum intensity and then multiplies it with the maximum VO_2 of

the subject to calculate final second-by-second VO_2. This VO_2 calculation has been found to be more accurate than methods based only on HR, especially during start of exercise and recovery from exercise (Pulkkinen 2003, Pulkkinen et al. 2004).

The software program also calculates second-by-second indices of stress and relaxation, reflecting activity of the parasympathetic (absolute relaxation vector, ARV) and sympathetic (absolute stress vector, ASV) nervous system. ARV and ASV are calculated from HR, HFP, LFP, and respiration rate (RespR) as follows:

ARV = sqrt(60000*log(HFP+1)/HR)

ASV = (HR/(RespR*log(HFP+1)*log(LFP+1)))

Further, the software calculates the duration of different physiological states of the body during the day or recording period by segmenting the data into stationary segments. The data segments, including movements, physical activity at different intensities (% of VO_{2max}) and recovery from physical activity, are detected first using HR, HRV-derived respiration rate, estimated VO_2 and movement-related ANS responses. Thereafter, for non-exercise data segments, the time when the body is in a relaxation state (RT, Relaxation Time), stress state (ST, Stress Time) or an unrecognized state (URS) is determined. The determination of stress and recovery states is based on the detection of sympathovagal reactivity that exceeds momentary metabolic requirements for the autonomic nervous system (ANS). The relaxation state is defined as a decreased activation in the body during relaxation, rest and/or peaceful working, related to the lack of external and internal stress factors when parasympathetic (vagal) activation is dominating. In the relaxation state HR is close to the basic resting level and HRV is great and regular. The stress state is defined as an increased activation in the body, induced by external and internal stress factors (stressors), during which sympathetic nervous system activity is dominating and parasympathetic (vagal) activation is decreased. The body is in the stress state when heart rate is elevated, HRV is reduced, there are inconsistencies in the frequency distribution of HRV, and respiration rate is low relative to HR and HRV. This definition does not take into account the nature of the stress response, that is, whether it is positive or negative. As the physical activity is determined first, there can not be stress or relaxation state when physical activity or

recovery from physical activity is detected. The FBT-PRO Software also calculates Relaxation-Stress Balance, i.e. the accumulation or decrease of resources during the day or recording period by taking into account the proportion of stress time and relaxation time (Kettunen & Saalasti 2004). For more details, see Kettunen and Saalasti (2004), and the Wellness Analysis Software User Manual (www.firstbeat.fi).

The accuracy of the software can be improved by giving exact personal information, namely person's real maximal and minimal heart rate, maximal oxygen uptake (in METs), maximal breathing frequency, maximal ventilation and vital capacity. The more exact the information added to person profile, the more exact are the results of the analysis. In this study, we wanted to have exact person profile parameters. That is why each subject performed a maximal dynamic exercise test on a cycle ergometer to get the information about subjects' physical fitness, maximal heart rate and maximal breathing frequency. Also all of the measured days were analyzed first with FBT-PRO and if there were updates in physiological background parameters, all of the days were analyzed again with the correct person profile. The lowest heart rate found was used as resting HR. If FBT-PRO found a higher breathing frequency during measured days than found in maximal exercise test, that higher breathing frequency value was ignored because in closer inspection those values were found to arise from errors in heart rate data. For maximal ventilation and tidal volume, no value was given to person profile.

After the analysis has been done, data export function yields information that has been computed during the analysis. The data export contains scalar variables for the whole measured period and vector variables for every second of a measured period. The exact content of data export (scalars and vectors) is shown in appendix 5. One of the vectors is the state vector that tells which one of the nine possible physiological states is detected during each second. These bodily states are the following:

1. Physical Activity > 95 % VO2max
2. Physical Activity 95–75 % VO2max
3. Physical Activity 75–50 % VO2max
4. Physical Activity 50–30 % VO2max
5. Light Physical Activity
6. Relaxation

7. Recovery from Physical Exercise
8. Stress
9. Unrecognized state

After the analysis the program also gives a percentage of error in the analyzed period, which means the percentage of how many R-R intervals had to be corrected by an artifact detection filter. In this study, we wanted to have high-quality HR data, and therefore, only subjects whose percentage of error during awake and sleep was less than 5% were approved.

For awake-sleep comparison, the FBT-PRO variables of interest in this study were the following: session total time, average heart rate, stress and relaxation time (min and %), stress balance, average absolute stress and relaxation index, average absolute stress and relaxation vector, physical exercise time (min and %), recovery time from physical exercise (min and %), and unrecognized time (min and %). For the analyses concerning sleep, the following variables were used in the analyses: session total time, average heart rate, stress and relaxation time (min and %), stress balance, average absolute stress and relaxation index, and average absolute stress and relaxation vector. The explanations for all variables can be found in appendix 5, but shortly session total time refers to total length of the analyzed period, averaged absolute stress and relaxation indices refer to the average absolute level of stress or relaxation of the session during the periods when that specific state was detected, and averaged absolute stress and relaxation vectors to the average of momentary absolute level of stress or relaxation during the session regardless of if the specific state was detected or not.

5.4.2 Heart rate variability calculations

In addition to stress and relaxation indices calculated by FBT-PRO, also traditional HRV variables were calculated from RR-interval data using MATLAB (MathWorks, version 7.0.1) –program. First the RR-interval data was corrected with MATLAB one subject at a time for artefacts (for more information, see Saalasti 2003). RR-interval data was opened and all of the data was first corrected with an automatic correction function (acmex.m). After automatic correction, the data was visually inspected by a researcher. Sections of the data were marked to be corrected again and some parts

which were not good enough for later analyses were marked not to be used in later analyses. After the inspection, the sections of the data which were accepted were once again corrected automatically with the correction function.

HRV was then calculated from corrected and visually inspected RR-interval data for time and frequency domain variables. HRV indices were calculated over 5-minute periods during the night-sleep and averaged for the 4-hour period starting 30 minutes after bed time, and for the whole sleep. Analyzed variables included: HR, R-R interval (RRI), SD, RMSSD, HF power (HF, 0.15–0.40 Hz), LF power (LF, 0.04–0.15 Hz), and LF+HF power (LF+HF, 0.04–0.40 Hz), and LF/HF ratio. Only 5-minute periods in which there was less than 5% error in the data were accepted for calculations (van Amelsvoort et al. 2000). In addition, only subjects whose RR-interval data had totally less than 5% of error (ectopic beats etc.) in the periods of interest (e.g. awake and sleep) were accepted for heart rate variability calculations. In other words, only subjects who had more than 95% of the 5-minute periods, including more than 95% uncorrected data, were approved for HRV calculations. Power spectrum in frequency domain was calculated using the Fast Fourier Transform (FFT) –method with a 512 samples Hanning window and ¾ overlapping. Before the analysis, the original RR-interval data was resampled at a rate of 5 Hz using linear interpolation to obtain equidistantly sampled time series.

5.4.4 Analyses of saliva (cortisol) samples

Saliva samples for cortisol analyses were analyzed in the Finnish Institute of Occupational Health (Helsinki, Finland) with an IBL-Hamburg LIA (Luminescence Immunoassay) kit which measures cortisol hormone from saliva. Normal cortisol values for adults measured in saliva are in the morning peak 13.8–48.9 nmol/l and eight hours after peak 1.4–8.6 nmol/l. The sensitivity of the method was 0.15 ng/ml, intra-assay precision was 2.9–7.7% at 0.96–8.70 ng/ml and inter-assay precision was 6.2-11.5% at 0.75–6.82 ng/ml. Cross-reactivity for corticosterone was 2.5%, for cortisone 2.0% and for prednisone 1.0%. There were two kit controls from the manufacturer and one control from the Finnish Institute of Occupational Health which was pooled saliva. The measured range for the samples was 0.41–110 nmol/l when the manufacturer for the kit

presents a range of 0.83–110 nmol/l. If the measured value was below the range, the concentration was expressed as 0.41 nmol/l and if it was above the range, it was diluted and measured again.

Two types of cortisol variables were calculated. Firstly, cortisol values were analyzed as raw cortisol values, and these were analyzed in the following time points:

1. Immediately after awakening ($AC_{0\ min}$),
2. 30 min after awakening ($AC_{30\ min}$)
3. 60 minutes after awakening ($AC_{60\ min}$)

Secondly, cortisol values were analyzed as awakening responses (CARs) measured with absolute values and with areas under the cortisol curve (AUCs). Cortisol awakening responses were calculated in absolute values by subtraction of the awakening value from the value 30 or 60 minutes after awakening ($CAR_{0\text{-}30\ min}$ and $CAR_{0\text{-}60\ min}$, respectively). In addition, three types of area under curve –values were calculated from awakening to 60 minutes after, and they were with respect to ground, with respect to increase after awakening (could have only positive values), and with respect to change after awakening (could have also negative values, if the trend of the curve was negative after awakening). Therefore, the cortisol awakening response variables were the following:

1. $CAR_{0\text{-}30\ min}$ and $CAR_{0\text{-}60\ min}$
2. AUC with respect to ground 0→60 minutes ($AUC_{g\ 0\text{-}60min}$)
3. AUC with respect to increase 0→60 minutes ($AUC_{i\ 0\text{-}60min}$)
4. AUC with respect to change 0→60 minutes ($AUC_{c\ 0\text{-}60min}$)

5.4.5 Comparison of awake and sleep, and sleep during the working period

Stress, relaxation and HRV variables were compared during awake and sleep-time in 12 subjects (n=12) using sleep and the following working day (awake). HR data of high-quality for both sleep and the following awake-time was for 11 subjects on workday 1 and for one subject on workday 2. Sleep during different days of the cycle of workdays and days off were also compared with each other, so that workday 1 sleep was

compared with workday 2 sleep, and workday 2 sleep with a day off sleep. The analyzed sleep was the sleep which preceded these above-mentioned days. For comparison of workday sleeps there were eleven subjects (n=11) and for comparison of workday 2 and day off sleep there were ten subjects (n=10).

5.4.6 Comparison of better and worse workday sleep

The night-sleep preceding workday 1 and workday 2 were categorized into better and worse sleep with the following five criteria:

1. Subjective ratings
2. Fragmentation of the sleep with Actiwatch Sleep Analysis (ASA)
3. Actual sleep time with ASA
4. Cortisol value at awakening ($AC_{0\ min}$)
5. Cortisol awakening response from awakening to 60 minutes after ($CAR_{0\text{-}60\ min}$)

Subjective ratings after awakening (1=slept well, 2=slept moderately or 3=slept badly) were used as a criterion so that every subject who had answered so that their ratings were different on workday 1 and workday 2 were accepted to the statistical analysis. The night-sleep which was answered to be better was categorized as better sleep and vice versa. There were seven subjects who had a different rating for the two workday sleeps (n=7).

Actiwatch Sleep Analysis (ASA) was used in two different ways in the categorization of the sleeps. Firstly, a 4-hour period starting 30 minutes after going to bed was used. During this period, the subjects should be sleeping properly, and the exact time of going to bed would not influence the results. In addition, as the length of the period was kept constant, differences in the total duration of sleep would not affect results. The criterion for categorization was that the sleep with lower fragmentation index (expresses restlessness during sleep) was defined to be better and the sleep with greater fragmentation index to be worse. Secondly, the sleep analysis was performed for the whole sleep using actual sleep time (true duration of sleep determined by a software algorithm) as a criterion variable. The sleep with longer actual sleep time was

categorized to be better and vice versa. In both categorizations, there were eleven subjects to match the criterion used (n=11).

Cortisol was used in the categorization in two different ways. Firstly, workday 1 and 2 sleeps were categorized to be better or worse by dividing them based on the cortisol value at awakening ($AC_{0\ min}$). The sleep with the lower cortisol value at awakening was defined to be better and the sleep with the greater cortisol value to be worse. The other categorization was done based on cortisol awakening response from awakening to 60 minutes after awakening ($CAR_{0\text{-}60\ min}$). The sleep with the smaller cortisol response in the morning was categorized to be better and the sleep with the greater response to be worse. In both categorizations, eleven subjects were used in the analysis (n=11).

5.4.7 Statistical analysis

Non-parametric Wilcoxon tests (two related samples) were applied to compare variable values at different time periods, and therefore, this study was completed as a within-subject design. With categorical variables (subjective ratings of the quality of sleep), Marginal Homogeneity test was used. The level for statistical significance was determined as $P < 0.05$. In the figures and tables, star symbols are used to express statistical significance as follows: $P \leq 0.05$ = *, $P \leq 0.01$ = **, and $P \leq 0.001$ ***.

6 RESULTS

6.1 Detection of stress and relaxation during sleep and awake-time

A general description of detecting stress and relaxation based on HR and HRV during sleep and awake-time in real life settings is found in this paragraph. The sleep and the following working day (awake-time) were analyzed in 12 subjects, who had less than 5% error in heart rate data during this period. Table 2 gives the information about different FBT-PRO-variables. Physical exercise at different intensities has been combined under the same variable, physical exercise, in Table 2 and in Figure 6.

TABLE 2. FBT-PRO variables from the sleep (sleep) and the following workday (awake).

	SLEEP				AWAKE				
	Minimum	Maximum	Average	SD	Minimum	Maximum	Average	SD	Sign.
Lenght of the period (min)	378	595	452	59	795	1 079	977	69	.002**
Heart rate average (bts/min)	54	70	60	5	75	94	84	6	.002**
Relaxation time (min)	173	426	293	94	0	88	13	25	.002**
Relaxation percentage (%)	29	91	65	19	0	9	1	3	.002**
Absolute relaxation index	66.0	89.5	74.0	6.7	64.9	91.4	76.6	9.2	.249
Absolute relaxation vector	63.1	87.4	71.2	6.4	47.7	60.2	56.0	3.5	.003**
Stress time (min)	0	376	112	102	274	892	636	213	.002**
Stress percentage (%)	0	63	25	19	26	91	65	20	.008**
Absolute stress index	0.05	0.25	0.16	0.05	0.13	0.25	0.18	0.03	.210
Absolute stress vector	0.10	0.19	0.15	0.02	0.18	0.29	0.21	0.03	.003**
Relaxation/stress balance (-1+1)	-0.4	1.0	0.5	0.4	-1.0	-0.4	-0.9	0.2	.002**
Physical Exercise time (min)	1	10	6	3	27	279	106	81	.002**
Physical Exercise percentage (%)	0	3	1	1	3	27	11	8	.002**
Recovery from Physical Exercise time (min)	0	9	1	3	45	441	197	121	.002**
Recovery from Physical Exercise percentage (%)	0	2	0	1	5	42	20	12	.002**
Unrecognized state time (min)	29	52	40	7	0	40	24	12	.012*
Unrecognized state percentage (%)	7	11	9	1	0	4	2	1	.002**

There were statistically significant differences in most of the FBT-PRO variables between sleep and awake. Only relaxation and stress index -variables did not differ significantly. Stress dominated awake time (awake 65±20% vs. sleep 25±19%, p=0.008) and relaxation dominated sleep (awake 1±3% vs. sleep 65±19%, p=0.002). In Figure 6, the proportion of different bodily states determined by FBT-PRO during sleep and awake-time is illustrated. It can be seen that during awake-time the subjects were mostly in stress state and there was very little relaxation during that time. On the other hand, during sleep, the subjects were in relaxation state the majority of the time. However, stress state dominated 25% of the sleep time with a lot of individual variation (SD 19%)

between subjects. The duration of physical activity and recovery from physical activity states during awake was also remarkably high as, on average, the subjects were in physical exercise state 11% of the awake-time and in recovery from physical exercise state 20% of the awake-time.

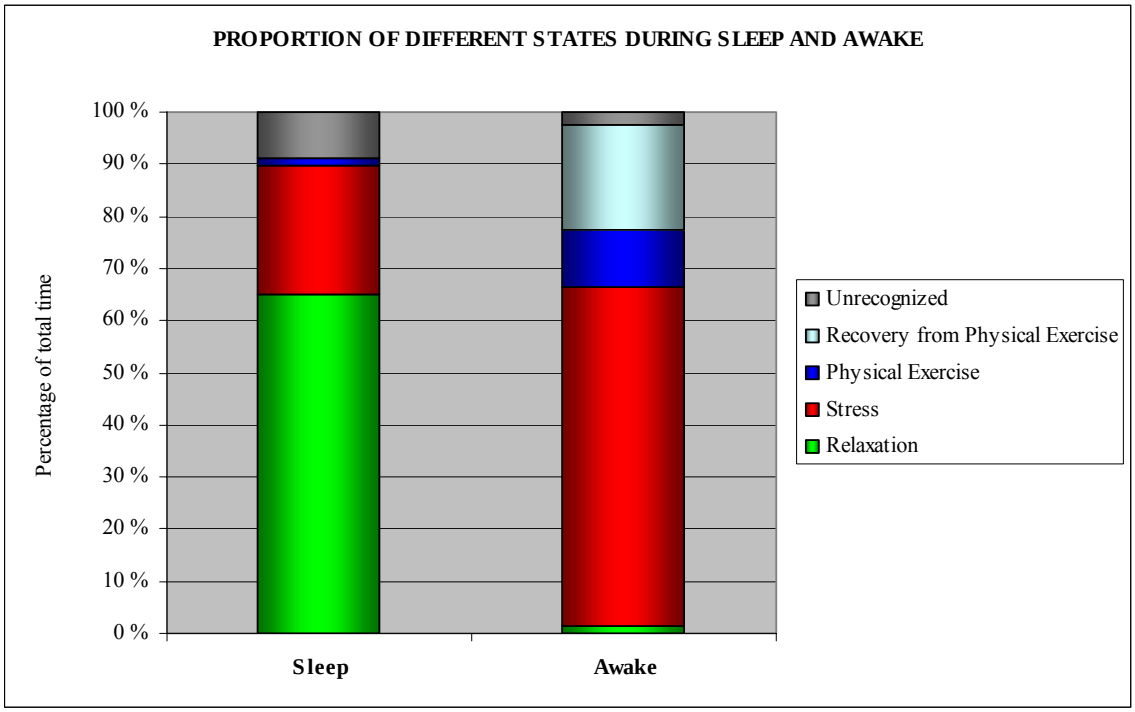

FIGURE 6. Proportion of different bodily states detected during night-sleep and awake time.

Averaged HR and HRV values during sleep and awake are presented in Table 3. It can be seen that during sleep the mean R-R interval was statistically significantly longer ($p=0.002$) than during awake-time (heart rate was lower), as presented also earlier in Table 2. The HRV variable estimating overall HRV (SDNN) and the variable reflecting both sympathetic and parasympathetic activity (LF power) were not different between sleep and awake. The HRV variables linked to parasympathetic modulation (RMSSD and HF power) were significantly higher during sleep than awake-time ($p=0.003$ for both). Also LF to HF ratio differed significantly ($p=0.002$). The percentage of how many acceptable 5-minute segments (i.e. data ok %) there were during sleep and awake used in the analysis also differed significantly, but in both awake and sleep the RRI data was of high quality. During sleep about 99% and during awake about 94% of the 5-min segments could be used in the HRV analysis. There existed a lot of individual variation in HRV values, as shown by the SD-values in Table 3.

TABLE 3. HRV variable values (averaged) during sleep and awake-time.

	SLEEP				AWAKE				
	Minimum	Maximum	Average	SD	Minimum	Maximum	Average	SD	Sign.(p)
Data ok (%)	94.8	100.0	99.2	1.8	76.6	100.0	94.2	6.4	.008**
RRi (ms)	882	1112	1004	77	647	827	731	54	.002**
SDNN (ms)	40.5	111.2	57.7	18.7	44.4	73.8	57.0	9.2	.583
RMSSD (ms)	19.0	83.5	35.0	17.5	11.7	33.4	22.6	6.6	.003**
HF (ms^2)	208	6190	1298	1675	88	1078	498	345	.003**
LF (ms^2)	469	5680	1845	1425	768	4064	1921	960	.388
LF/HF	1.0	5.0	2.4	1.3	3.6	15.2	6.3	3.2	.002**
LF+HF	681	12024	3172	3081	984	5204	2447	1289	.388

6.2 Comparison of workdays and a day off

Workday 1 was at the beginning of the working period and workday 2 at the end of the working period of 3–4 working days. These two workdays were very similar because there were no significant differences in the analyzed variables between these two sleeps. The quality of the sleep measured with ASA seemed to be better on workday 2 because fragmentation index for whole sleep tended to be lower on workday 2 (p=0.075). There were not any statistically significant differences either in FBT-PRO or HRV variables between the sleeps analyzed over the whole sleep or over the 4-hour period.

Workday 2 and day off were also compared, in which the day off was the first leisure day after the working period. Differences between these two sleeps are shown in Table 4. In the day off sleep, session total time (total length of the sleep) was statistically significantly longer than on workday 2 (p=0.005) and also actual sleep time during the whole sleep was significantly longer on day off than on workday 2 (WD2 6:34±1:05 vs. DO 7:55±0:41 h:min, p=0.005). The difference could be seen in awakening time, which was significantly later on the day off morning (WD2: 5:41±0:36, DO: 7:02±0:50, p=0.008), but bed time did not differ between these two days (p=0.649).

When the whole sleep was analyzed, significantly more relaxation time (see Figure 7, page 49) was detected during day off than during workday 2 sleep (WD2 4:29±1:38 vs. DO 5:25±1:58, p=0.037), but no difference was seen in stress time (p=0.314). There were no differences in stress or relaxation during the 4-hour period. The subjects rated their day off sleep to be subjectively better than workday 2 sleep (WD2: 2.00±0.76, DO:

TABLE 4. Comparison of workday 2 and day off sleep.

	WORKDAY 2		DAY OFF		
	Average	SD	Average	SD	Sign. (p)
Cortisol					
Time point for 1st cortisol sample (h:min)	5:41	0:36	7:02	0:50	0.008**
Time point for 2nd cortisol sample (h:min)	6:12	0:36	7:33	0:51	0.008**
Time point for 3rd cortisol sample (h:min)	6:46	0:34	8:03	0:51	0.008**
Cortisol value at awakening (nmol/l)	13.9	6.8	15.3	8.1	0.859
Cortisol value 30 min after awakening (nmol/l)	26.9	10.6	22.3	8.4	0.678
Cortisol value 60 min after awakening (nmol/l)	25.9	10.1	17.5	5.7	0.051
Cortisol Awakening Response 0-30 min (nmol/l)	13.0	9.3	7.0	9.3	0.260
Cortisol Awakening Response 0-60 min (nmol/l)	12.0	14.2	2.2	10.4	0.173
AUC with respect to zero 0-60 min (nmol/l*h)	46.7	14.7	38.7	12.2	0.110
AUC with respect to increase 0-60 min (nmol/l*h)	18.9	14.5	10.3	11.2	0.214
AUC with respect to change 0-60 min (nmol/l*h)	18.9	14.6	8.1	13.8	0.214
Subjective					
subjective rating	2.0	0.8	1.2	0.4	0.034*
Actiwatch Sleep Analysis					
Actual sleep time (4 h)	3:44	0:05	3:44	0:04	0.905
Sleep efficiency (4 h, %)	93.7	2.2	93.7	1.9	0.959
Number of awakenings (4 h)	17	4	16	5	0.683
Fragmentation Index (4 h)	20.3	8.6	22.9	8.5	0.575
Actual sleep time (whole sleep)	6:34	1:05	7:55	0:41	0.005**
Sleep efficiency (whole sleep, %)	91.0	2.0	92.3	2.5	0.114
Number of awakenings (whole sleep)	30	10	37	7	0.066
Fragmentation Index (whole sleep)	21.8	7.2	25.3	8.0	0.093
FBT-PRO variables for whole sleep					
Session total time (min)	428	57	506	53	0.005**
Relaxation time (min)	269	98	325	118	0.037*
Stress time (min)	116	99	134	85	0.314
Average HR (times/min)	63	6	62	5	0.245
Relaxation percentage (%)	63	22	63	21	0.878
Stress percentage (%)	27	22	28	21	0.878
Relaxation/Stress -balance (-1 - +1)	0.4	0.5	0.4	0.4	0.767
Absolute Stress Vector	0.15	0.02	0.15	0.02	0.203
Absolute Relaxation Vector	70.0	4.1	71.1	3.8	0.169
FBT-PRO variables for 4-hour period					
Relaxation time (min)	130	65	126	65	0.799
Stress time (min)	90	71	94	68	0.726
Average HR (times/min)	64	6	64	6	0.233
Relaxation/Stress -balance (-1 - +1)	0.2	0.6	0.2	0.6	0.624
Absolute Stress Vector	0.16	0.02	0.16	0.03	0.305
Absolute Relaxation Vector	68.7	4.3	69.4	4.0	0.333
HRV variables for whole sleep					
Data ok (%)	97.2	5.2	98.2	4.3	0.128
RR interval (ms)	966	84	975	76	0.508
SDNN (ms^2)	53.6	10.1	55.4	9.6	0.646
RMSSD (ms^2)	32.9	8.7	34.7	9.8	0.333
HF (ms^2)	1002	649	1025	595	0.575
LF (ms^2)	1606	844	1677	865	0.333
LF/HF	2.2	1.0	2.1	0.7	0.575
LF+HF (ms^2)	2629	1405	2722	1411	0.508
HRV variables for 4-hour period					
Data ok (%)	97.3	6.5	98.1	4.2	0.465
RR interval (ms)	946	88	953	82	0.575
SDNN (ms^2)	49.5	7.1	49.7	7.7	0.799
RMSSD (ms^2)	31.2	8.5	32.4	9.4	0.508
HF (ms^2)	906	571	946	607	0.646
LF (ms^2)	1445	801	1476	907	0.799
LF/HF	2.4	1.2	2.1	0.7	0.285
LF+HF (ms^2)	2368	1228	2439	1468	0.721

1.22±0.44, p=0.034). However, there tended to be more awakenings and a higher fragmentation index during the whole day off sleep, but not significantly (p=0.066 and 0.093, respectively). Despite of this, sleep also tended to be more efficient during the day off (p=0.114). There were no statistically significant differences between workday 2 and day off sleeps in any HRV variable mean values over the 4-hour period or over the whole sleep. There was seen a tendency towards lower cortisol at awakening ($AC_{60\ min}$) on day off compared to workday 2 (p=0.051), but awakening responses did not differ between these sleeps.

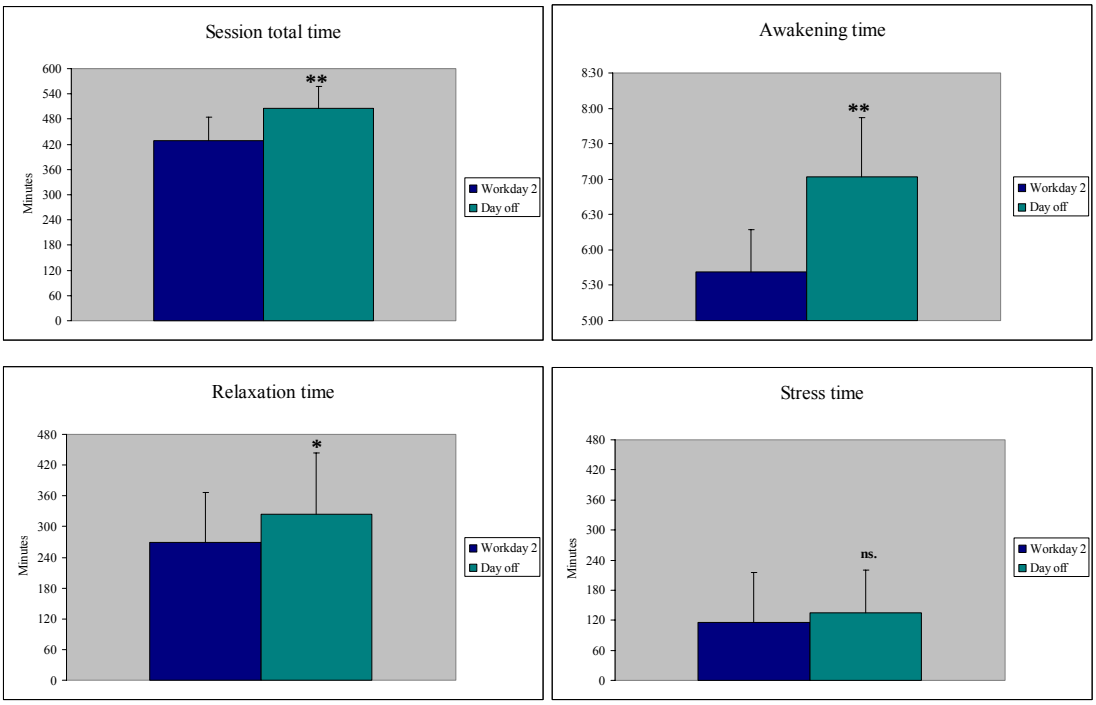

FIGURE 7. Session total time (total duration of sleep), awakening time, duration of relaxation and stress states on workday 2 and day off sleep.

6.3 Comparison of better and worse workday sleep

6.3.1 Subjective ratings of the quality of sleep

The subjective ratings of the quality of sleep (1=slept well, 2=slept moderately or 3=slept badly) were different on workday sleeps in seven (n=7) subjects (better sleep: 1.43±0.53 vs. worse: 2.43±0.53, p=0.008). However, there were no statistically

significant differences between better and worse sleep in stress and relaxation detected or in ASA variables analyzed for the 4-hour period or for the whole sleep, nor in the cortisol variables. There was significantly more LF power (p=0.028) during better than during worse sleep when the whole sleep was analyzed. Also a tendency towards greater LF+HF power was seen during better than during worse sleep (p=0.063). No differences were found in HRV variables in the 4-hour period between better and worse sleep.

6.3.2 Fragmentation of sleep

After the categorization, better and worse sleeps were not different in session total time (the total duration of sleep) but differed in the following sleep analysis variables: actual sleep time (p=0.005), sleep efficiency (p=0.003), number of awakenings (p=0.007) and fragmentation index (an index of restlessness, p=0.003) for the 4-hour period and in fragmentation index (p=0.003) for the whole sleep. The difference in fragmentation index during the 4-hour period between better and worse workday sleep at individual level was on average 18.0±14.0 (range 0.6–38.6). Therefore, some subjects had very little difference between better and worse sleep in fragmentation index, and there was some overlapping in a way that the fragmentation index in worse sleep in some persons was smaller than the fragmentation index in better sleep in some other persons. Figure 8 (page 51) illustrates these sleep quality differences measured with the sleep analysis.

There was significantly more relaxation time (better: 147±59 vs. worse: 119±69 min, p=0.033) during the less than more fragmented 4-hour period. There was also a tendency towards shorter stress time (better: 71±65 vs. worse: 99±73 min, p=0.059) in less than in more fragmented sleep during the 4-hour period. Relaxation/stress –balance differed significantly (better: 0.36±0.57 vs. worse: 0.10±0.64, p=0.047) during the 4-hour period in less and more fragmented workday sleep. In addition, there was significantly more relaxation time (better: 304±104 vs. worse: 267±91 min, p=0.026) in less than more fragmented sleep during the whole sleep. There was also seen a tendency towards shorter stress time (better: 95±89 vs. worse: 124±88 min, p=0.075) during the whole sleep in less than more fragmented sleep. Also relaxation/stress –balance tended to differ during the whole sleep (better: 0.51±0.46 vs. worse: 0.36±0.41, p=0.091)

between less and more fragmented sleep. The differences in FBT-PRO variables are presented in Figure 9.

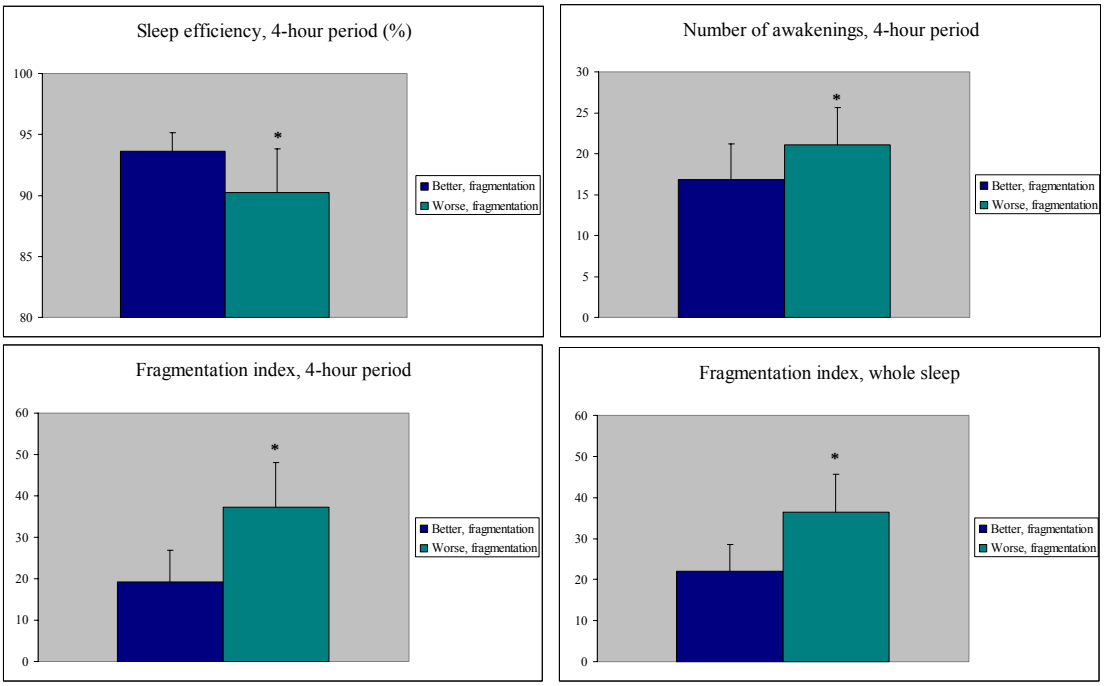

FIGURE 8. Sleep efficiency, number of awakenings, and fragmentation index during the 4-hour period, and fragmentation index for the whole sleep in more and less fragmented workday sleep.

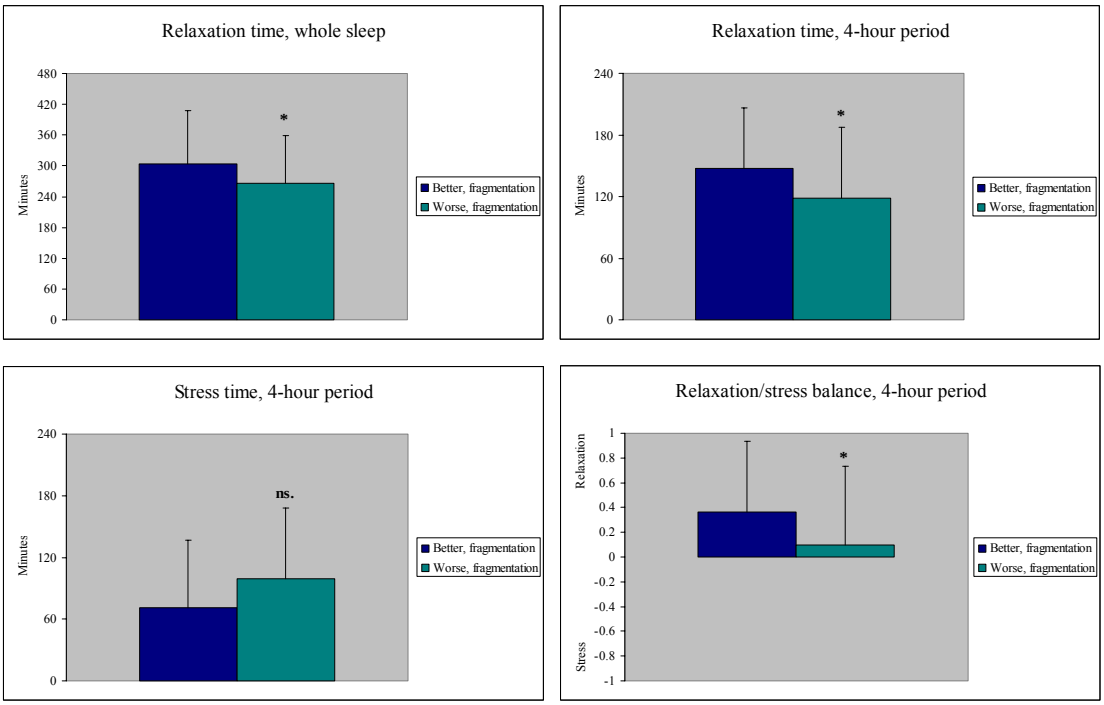

FIGURE 9. Relaxation time during the whole sleep, and relaxation and stress times, as well as relaxation/stress –balance during the 4-hour period in more and less fragmented workday sleep.

No significant differences were found in HRV variables for the whole sleep. However, there was significantly more LF power, but no other differences, in the 4-hour period during worse than better sleep (p=0.050). No differences were observed in any cortisol variables after less and more fragmented workday sleep.

6.3.3 Actual sleep time

Workday sleeps with different actual sleep time (AST) were also categorized to better and worse. The difference in actual sleep time was significant between better and worse sleep (longer AST: 7:13±0:41, shorter AST: 6:13±0:55, p=0.003). At individual level, the difference between better and worse sleep in actual sleep time was on average 0:59±0:46 h:min (range 0:07–2:38 h:min). Bed time was significantly earlier (p=0.021) and session total time (the total duration of sleep) longer (p=0.005) during the sleep with longer actual sleep time. Awakening time in the morning was not significantly different between the two sleeps (longer AST: 5:56±0:40, shorter AST: 5:48±0:35, p=0.423). There was significantly more relaxation (p=0.026) during the whole sleep with longer actual sleep time, but no differences were found in stress time (p=0.859). There weren't any significant differences in detected stress and relaxation during the 4-hour periods. The RMSSD value was significantly greater (p=0.050) during sleep with longer actual sleep time than with shorter actual sleep time, but no other differences in HRV variables between the sleeps were found. Figure 10 (page 53) illustrates the differences between the two sleeps.

Cortisol awakening responses tended to be smaller after sleep with longer actual sleep time than with shorter actual sleep time ($CAR_{0\text{-}30\ min}$: longer AST: 9.34±12.31 vs. shorter AST: 16.49±16.06, p=0.062, and $CAR_{0\text{-}60\ min}$: longer AST: 6.72±19.29, shorter AST: 15.08±15.34, p=0.075). $AUC_{i\ 0\text{-}60\ min}$ and $AUC_{c\ 0\text{-}60\ min}$ were significantly smaller (p= 0.050 for both) after sleep with longer actual sleep time. $AUC_{g\ 0\text{-}60\ min}$ did not differ between shorter and longer sleeps. Individual variation in these awakening responses seemed to be high. No significant differences were found in raw cortisol values in the morning ($AC_{0/30/60\ min}$). Figure 11 (page 53) represents the differences in cortisol awakening responses between workday sleeps with longer and shorter actual sleep time.

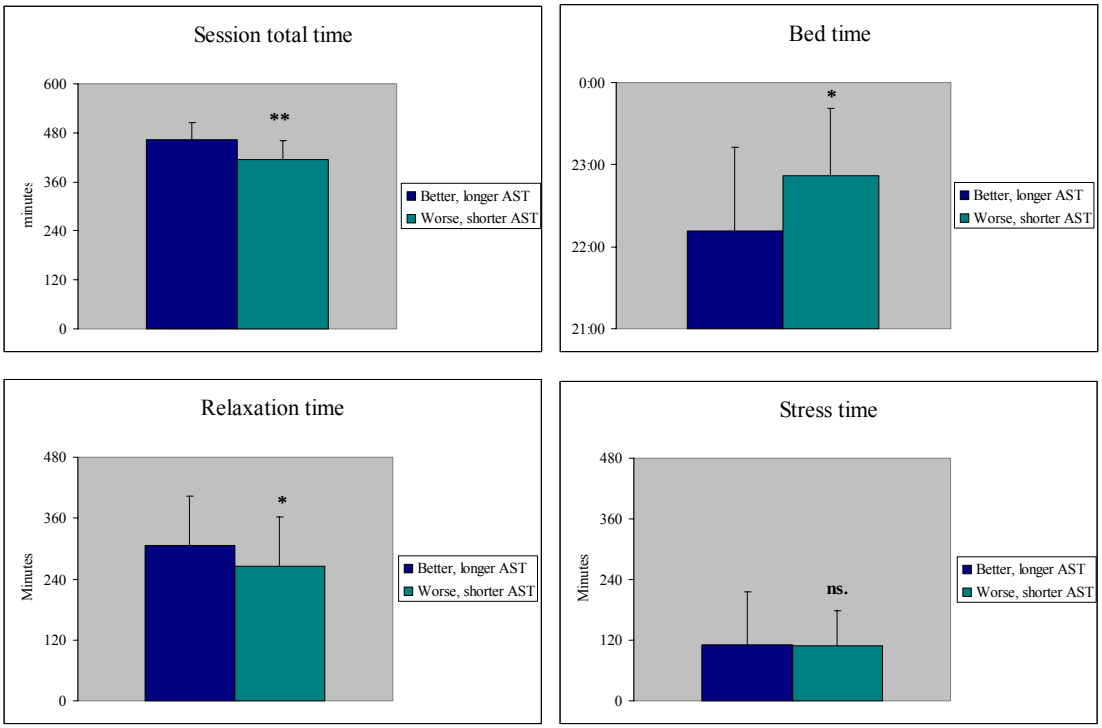

FIGURE 10. Session total time (the total duration of sleep), bed time, and duration of relaxation and stress states during workday sleep with longer and shorter actual sleep time.

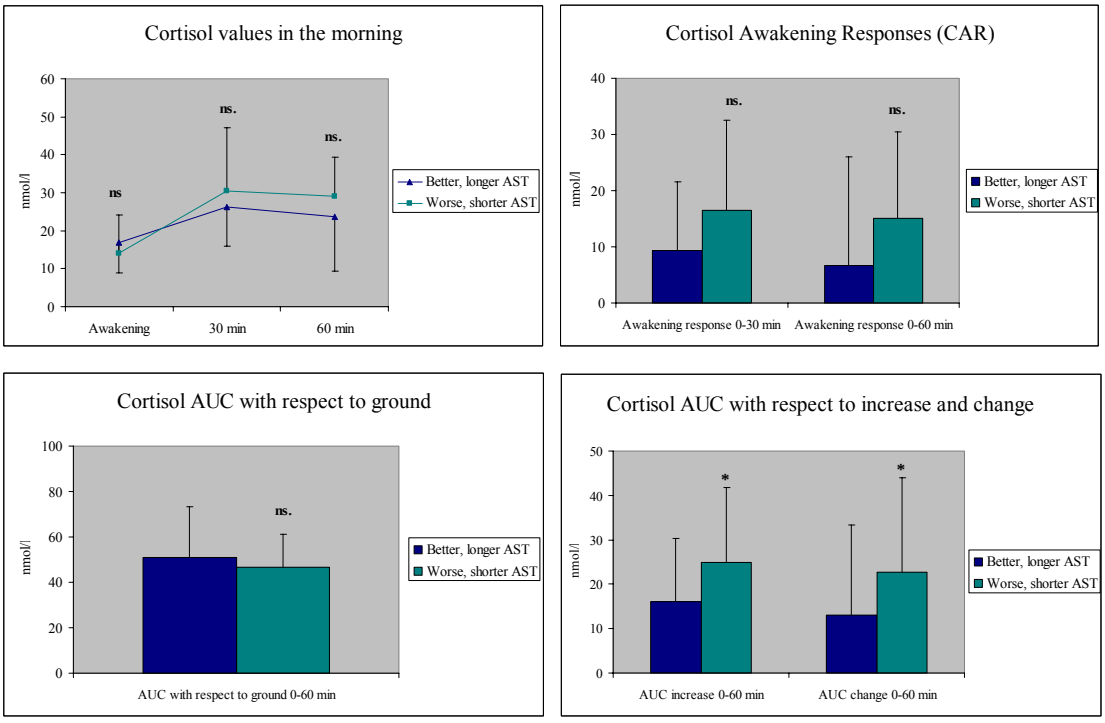

FIGURE 11. Raw cortisol values and awakening responses in absolute values as well as in areas under the curve on workday sleeps with longer and shorter actual sleep time.

6.3.4 Cortisol at awakening

The sleep with a lower cortisol value at awakening ($AC_{0\ min}$) in the morning was defined to be better and the sleep with a greater cortisol value to be worse in the categorization. Cortisol at awakening, which was the criterion variable, was significantly (p=0.003) lower in the morning after better than worse sleep (11.26±6.31 vs. 19.66±9.58 nmol/l, respectively). However, $CAR_{0\text{-}60\ min}$ was significantly (p=0.041) greater after sleep with lower than with greater $AC_{0\ min}$ (19.28±14.83 vs. 2.53±16.48 nmol/l, respectively). Cortisol curves and the magnitudes of $CAR_{0\text{-}60\ min}$ are presented in Figure 12.

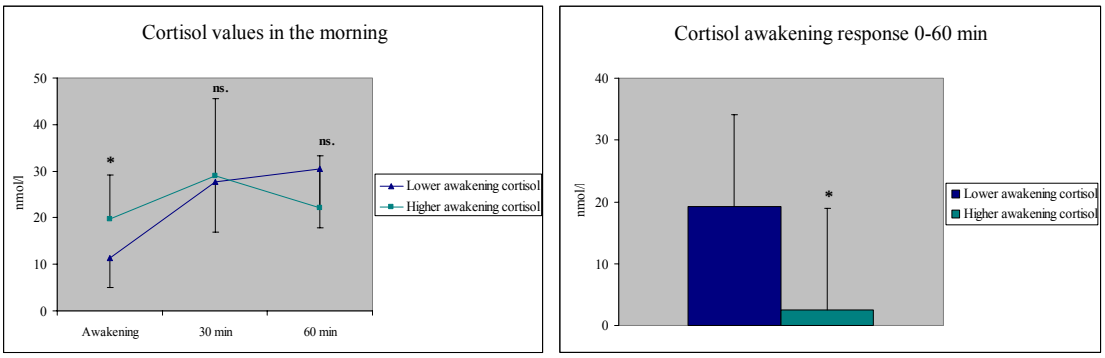

FIGURE 12. Cortisol curves and awakening responses in absolute values ($CAR_{0\text{-}60\ min}$) in workday sleeps with lower and greater cortisol at awakening ($AC_{0\ min}$).

The increase and change in cortisol values after awakening ($AUC_{i\ 0\text{-}60min}$ and $AUC_{c\ 0\text{-}60min}$) were significantly greater after sleep with lower than with higher $AC_{0\ min}$ (p=0.050 and p=0.041, respectively). Interestingly, there tended to be less stress time (p=0.075) during the whole sleep in sleep with lower $AC_{0\ min}$. The absolute stress vector had significantly (p=0.031) lower values during the 4-hour period in sleep with lower AC_{0min}. There also tended to be more HF power in the 4-hour period (p=0.075). There were no differences in relaxation time (p=0.790) or ASA variables between sleeps with different awakening cortisol ($AC_{0\ min}$).

6.3.5 Cortisol awakening response

Finally, the sleep with a smaller cortisol response ($CAR_{0\text{-}60min}$) in the morning was defined to be better and the sleep with a greater response to be worse. The results show

that cortisol awakening responses in absolute values ($CAR_{0\text{-}30\ min}$ and $CAR_{0\text{-}60\ min}$), the latter being used as a criterion, differed significantly between the sleeps (6.61±14.25 vs. 19.22±12.31 nmol/l, p=0.01, and 0.79±14.83 vs. 21.01±14.19 nmol/l, p=0.003, respectively). $AC_{0\ min}$ was significantly smaller (p=0.016) and $AC_{60\ min}$ greater (p=0.008) during the day with a greater awakening response ($CAR_{0\text{-}60min}$). $AUC_{i\ 0\text{-}60\ min}$ and $AUC_{c\ 0\text{-}60\ min}$ were significantly smaller (p=0.01 and 0.004, respectively) in sleeps with smaller $CAR_{0\text{-}60\ min}$. Actiwatch Sleep Analysis indicated that the quality of sleep was better during sleep with smaller $CAR_{0\text{-}60\ min}$ because the actual sleep time was significantly longer (7:04±1:02 vs. 6:23±0:42 h:min, p=0.050) and sleep efficiency greater (90.75±3.45% vs. 88.25±2.39%, p=0.029) during sleep with smaller than with greater $CAR_{0\text{-}60\ min}$. Figure 13 expresses cortisol curves and actual sleep times after this categorization.

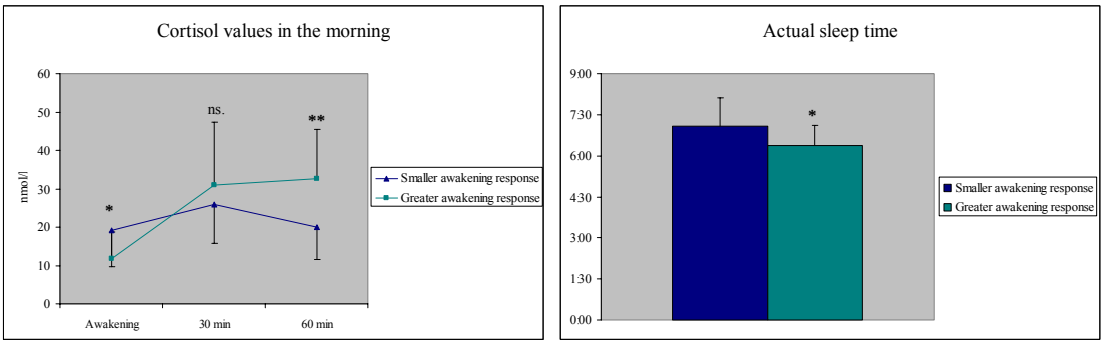

FIGURE 13. Cortisol curves and actual sleep times during workday sleep with a smaller and greater cortisol awakening response ($CAR_{0\text{-}60\ min}$).

There were not any significant differences between the sleeps in stress and relaxation times during the whole sleep or during the 4-hour period. However, SDNN was significantly smaller (p=0.010) in the 4-hour period during sleep with smaller $CAR_{0\text{-}60\ min}$.

7 DISCUSSION

7.1 Summary of the results

This chapter summarizes the most important results of the present study. The first study approach was to compare stress and relaxation indices during awake and sleep. HR and HRV -based FBT-PRO Heartbeat Analysis Software detected significantly more stress during awake-time (awake 65±20% vs. sleep 25±19%, p=0.008) and relaxation during sleep (awake 1±3% vs. sleep 65±19%, p=0.002). During sleep, HR was lower compared to awake-time (awake 84±6 vs. sleep 60±5 beats/min, p=0.002), and HRV variables RMSSD and HF power were significantly greater (p=0.003 for both), whereas LF/HF was significantly lower (p=0.002) than during awake-time.

The second study approach was to compare stress and relaxation indices during the cycle of workdays and days off. There were not any significant differences in the analyzed variables between workday 1 and 2 sleeps. However, there were some differences between workday 2 and day off sleeps. The actual sleep time was significantly longer (WD2 6:34±1:05 vs. DO 7:55±0:41 h:min, p=0.005), and there was significantly more relaxation time (WD2 4:29±1:38 vs. DO 5:25±1:58, p=0.037) during day off than during workday 2 sleep. However, there were no differences in the traditional HRV variables between workday 2 and day off sleep. The day off sleep was rated to be subjectively better (p=0.034) than workday 2 sleep.

The comparison of stress and relaxation indices during better and worse workday sleep was the third study approach. There were not any differences in stress and relaxation detected during *subjectively* different sleeps, even though there was significantly more LF power during better sleep (p=0.028). Relaxation time was longer during sleep with smaller than with greater *fragmentation index* during the 4-hour period and during the whole sleep (relaxation time for whole sleep: 5:05±2:01 vs. 4:35±1:48 h:min, p=0.026). Relaxation time was significantly longer during workday sleep with more than with less *actual sleep time* (5:06±1:37 vs. 4:25±1:37 h:min, p=0.026) and the RMSSD value was

significantly greater (p=0.050) in sleep with more actual sleep time. The increase and change in cortisol after awakening ($AUC_{i\ 0\text{-}60min}$ and $AUC_{c\ 0\text{-}60\ min}$) were significantly greater (p=0.050 for both) after sleep with less actual sleep time. Lower *awakening cortisol* ($AC_{0\ min}$) was accompanied by a greater increase and change in cortisol after awakening ($AUC_{i\ 0\text{-}60min}$ and $AUC_{c\ 0\text{-}60\ min}$, p=0.050 and 0.041, respectively). There also tended to be less stress time (p=0.075) during sleep with lower awakening cortisol ($AC_{0\ min}$). There was longer actual sleep time (p=0.050) and greater sleep efficiency (p=0.029) but no differences in detected stress or relaxation during sleep with smaller than with greater *cortisol awakening response* ($CAR_{0\text{-}60\ min}$).

7.2 HRV-based stress and recovery during awake and sleep

During awake-time HR was elevated (R-R intervals shortened), and HRV-variables expressing parasympathetic modulation (RMSSD and HF power) were decreased, and the LF/HF ratio was increased compared to sleep-time, all suggesting increased sympathetic activity during awake. Only a small amount of relaxation and a significant amount of stress was detected during awake, which agrees with decreased parasympathetic and increased sympathetic modulation during awake-time. Also the amount of physical activity (106±81 min, 11±8%) and recovery from physical activity (197±121 min, 20±12%) were high during the day. This may reveal that the nursing work is physically demanding, as reported in the literature (Trinkoff et al. 2003), including lots of hurrying, bending, twisting, and heavy lifting. Hui et al. (2001) showed that nurses in geriatric settings had a heart rate exceeding 90 beats/min in 57% and 110 beats/min in 19% of their working hours. In the present study, the average HR for awake-time was 84±6 beats/min, including the off work time also. These physical demands of nursing work may affect the detection of stress and recovery during awake-time, and increase the minutes of physical activity and recovery from physical activity.

HRV calculations indicated that parasympathetic modulation of ANS was increased during sleep compared to awake-time, as RMSSD and HF power were significantly greater during sleep than awake. Also LF/HF and HR were significantly lower during sleep compared to awake-time, which confirms the predominance of parasympathetic over sympathetic modulation during sleep. Relaxation is defined as a state when

parasympathetic modulation is dominating, and it is calculated from HR and HF power variables. Therefore, the finding that relaxation dominated sleep agrees with the present HR and HRV results if they indicate parasympathetic control. It has indeed been shown that high frequency variation (HF) is predominantly under parasympathetic control (Martinmäki et al. 2006) and that HF power highly correlates with RMSSD (Otzenberger et al. 1998). These results were in line with previous studies showing decreased heart rate and LF/HF ratio and increased HF power during night-time (e.g. Beckers et al. 2006, Bilan et al. 2005, Carrington et al. 2003, Ito et al. 2001, Ramaekers et al. 1998). However, in the LF component of HRV no increase was found during sleep in this study, which is in contrast to the findings of Ito and co-workers (2001) but in line with Bilan et al. (2005), who did not observe any day-night differences for the LF component. Ramaekers et al. (1998) reported that the relatively small day/night differences in SDNN and LF power do not always reach statistical significance in smaller groups.

Because it seems that most of the people have stress (mental or physical born increase in activity or both) during the day, more profound information about recovery can probably be obtained if recovery is measured during night-sleep, which is the natural period for recovery. It was found in this study that relaxation dominated only 65% and stress dominated 25% of the sleep time. Furthermore, there was a lot of individual variation in stress and relaxation during sleep, which indicates that there really are differences in recovery during sleep. It was also observed that there was virtually no relaxation state detected during awake-time. These reasons may make sleep an even more advisable time period for the measurement of recovery. Sleep is also a physiologically stable condition with a minimal amount of uncontrolled external factors affecting the measurements.

Vrijkotte et al. (2000) showed in their study dealing with work stress and ambulatory cardiovascular profile that heart rate and RMSSD during sleep were the best predictors of mild hypertension. It has been found by van Amelsvoort et al. (2003) that the need for recovery after work is a strong predictor of cardiovascular disease later on. In another study, van Amelsvoort and co-workers (2000) found that shift workers had decreased HRV during sleep compared to daytime workers. It is also generally known that shift work is strongly associated with increased cardiovascular disease risk. It has

been shown that sleep quality is negatively related to feelings of fatigue, and therefore, appropriate sleep is important for recovery (Rook & Zijlstra 2006). The importance of sleep for recovery has been recognized, but studies concerning recovery during sleep in relation to work stress and health are still rare. Recovery during sleep should therefore be studied more extensively. (Zijlstra & Sonnentag 2006.)

As far as athletes are concerned, a study considering autonomic modulation of overtrained athletes during sleep recently showed that there was no difference in HRV between overtrained and control athletes during sleep, but parasympathetic modulation was slightly diminished after awakening in overtrained athletes (Hynynen et al. 2006a). The authors concluded that studies of autonomic modulation in overtraining should concentrate on different challenges, such as awakening, in the future. On the other hand, Hynynen and co-workers (2006b) have also shown that in overtraining state nocturnal HR was higher and HRV diminished (LF and HF power) compared to a good recovery condition. Earlier it has also been shown that overtraining, which has similarities with burnout symptoms, is associated with increased sympathetic activity during supine rest (Uusitalo et al. 2000, Mourot et al. 2004). In addition, it has been shown by Pichot et al. (2000) that a heavy training period of three weeks decreased parasympathetic and increased sympathetic activity of the ANS during sleep measured by HRV, followed by a rebound in HRV variables during a recovery week. Furthermore, HRV seemed to reflect the training status better than e.g. resting heart rate, as resting HR changed only up to 10% during the heavy training period, whereas in HRV up to 40% changes were observed, indicating that HRV during sleep appears to be a potential tool for measuring cumulated training load in athletes.

In conclusion, the results of the present study clearly showed that it is possible to detect stress and recovery based on HR and HRV from ambulatory RR-interval recordings because, on average, there was more stress during awake and more relaxation during sleep. Therefore, the new method seems to be an appropriate tool for the measurement of stress and recovery during awake and sleep. Sleep is the most important period for physiological recovery of a person, and the finding of the present study that there are real differences in recovery during sleep further support the idea that nocturnal HRV measurement may provide a powerful tool for deeper understanding and more precise evaluation of the process of recovery in both athletes and employees.

7.3 Stress and recovery during the cycle of workdays and days off

Workday 1 vs. workday 2. The results from comparison of workday 1 and 2 sleep showed that these sleeps were very similar, as the only difference was a tendency for a lower fragmentation index for the whole sleep on workday 2. These results indicate that there was no accumulation of work stress during the working week. However, a "first night effect" may have influenced the measurement so that heart rate and movement monitors could have induced more stress on workday 1. It has also been reported that the quality of sleep is quite low on the night before the start of the working period (usually Sunday-Monday) (Rook & Ziljstra et al. 2006). This may be linked to anticipation of work demands – people start thinking about their work Sunday evening, and their worry affects the quality of their sleep. It has also been presented that the surge in blood pressure is the highest on Monday morning, which is a time point when cardiovascular events often occur (Murakami et al. 2004). Altogether, it may also be the case in this study that the combination of the "first night effect" and anticipation of the coming working period may have influenced the autonomic nervous system during the sleep preceding workday 1, which may have induced the same stress level as the effect of the working period. However, it is also possible that the working period did not increase stress or that coincidence plays some role with this small number of subjects.

Workday 2 vs. day off. The hypothesis of more relaxation during the day off sleep was supported as there was significantly more relaxation time during sleep on day off than on workday 2. There were no differences in detected stress time between these two sleeps. Subjects also slept significantly longer during day off than during workday 2. The difference was found in awakening time because bed time did not differ between these days. The subjects also reported better subjective sleep quality on the day off. We did not observe any statistically significant differences in HRV variables between workday 2 and day off, as was the case also in comparison of workday 1 and 2. This may suggest that HRV variables by themselves are not enough for measurement of recovery. The present new method seemed to succeed in combining the information from HR and HRV to make conclusions about stress and recovery during sleep.

The results showed that more recovery during day off was achieved through longer night-sleep. In actual sleep time, an average of 6:45 versus 6:42, and 6:34 versus 7:55 in comparison of workday 1 and 2, and comparison of workday 2 and day off, respectively (different number of subjects), were found in this study. The workday sleeps were relatively short compared to the average need for sleep in humans presented in the literature (7 to 9 hours) (Härmä & Sallinen 2004). Therefore, these results may indicate that insufficient sleep during the working week was compensated for by longer sleep during the day off. The results were in accordance with Edéll-Gustafsson et al. (2002) who also reported significantly longer sleep duration during the day off among Swedish female workers within the health care system. Nearly 40% of the subjects in that study complained of insufficient sleep. Rook & Zijlstra (2006) reported that sleep time increases and fatigue scores decrease during the weekend. Åkerstedt et al. (2000) also reported that early morning work is characterized by severe subjective sleepiness regardless of occupation, and that during the traditional five days work and two days off one to two days of recovery are normally needed.

In the background information questionnaire, one subject reported having had abnormal tiredness weekly during the previous month, three subjects less frequently and 10 subjects never. Two subjects reported having had abnormal insomnia weekly during the past month, seven subjects less frequently, and five never. None of the subjects reported having had these aforementioned symptoms daily. However, these results indicate that there were some sleep problems present (especially associated with insomnia), which is in accordance with numerous studies reporting a combination of stress and sleep disorders as an important occupational problem, especially among health care professionals working in shifts (Muecke 2005, Suzuki et al. 2005, Kilpatrick & Lavoie-Tremblay 2006, Ruggiero 2003). Compared to the general population of Finland, the subjects in this study did not, however, report a greater presence of insomnia. Ohayon & Partinen (2002) reported that insomnia symptoms occurring at least three times a week affected more than a third of the nearly one thousand citizens of Finland from the age groups 18 years and older who participated in the telephone interview study.

In addition, there were no differences in cortisol values in the morning, which have been used as a golden standard for the physiological measurement of work stress, between workday 2 and day off. This finding is in contrast to the findings of Kunz-Ebrecht et al.

(2004) and Schlotz et al. (2004), who found decreased cortisol responses during the weekend, when the subjects were off work. These differences were discussed to arise from higher levels of stress or anticipation of activities on the working day. Kunz-Ebrecht (2004) reported that awakening time was not associated with cortisol response, in line with e.g. Pruessner et al. (1997). On the other hand, Schlotz et al. (2004) found an influence of awakening time to cortisol responses, referring to Edwards et al. (2001) and Federenko et al. (2004) with similar findings. Thorn et al. (2006) reported a negative correlation between awakening time and cortisol secretion in the morning (increase after awakening and AUC), but also greater cortisol awakening responses on weekdays compared to weekends, which was not accounted for by awakening time. However, this finding was abolished when the subjects who showed no increase in the morning (suspected non-adherence to the instructions) were extracted from the analyses.

7.4 Comparison of better and worse workday sleep

Subjective ratings. When the workday sleeps with different subjective ratings were compared (n=7), there weren't any significant differences in detected stress and relaxation nor in sleep analysis (ASA) or cortisol variables. However, there was significantly more LF power and a tendency to more LF+HF power during the whole sleep in the subjectively better night. Therefore, HRV seemed to be slightly greater during the sleep which was also rated to be subjectively better, but no differences were found in the short term components of HRV more closely related to parasympathetic modulation. The lack of significant differences in stress and relaxation, ASA, and cortisol variables may indicate that there are also some other factors than the objective sleep quality affecting the subjective opinion of the sleep in the morning.

Rook & Zilstra (2006) reported in their study that the better the participants felt upon arising and the more positive the ratings of the sleep period were, the less likely they were to experience fatigue at the end of the working day. This illustrates the importance of subjective sleep quality in addition to objective measurements. Also Pilcher (2000) reported that subjective sleep quality was better related to sleepiness during awake-time than the quantity of sleep. In the present study, we did not observe that the subjective sleep quality in the morning would have been associated with stress or relaxation

detected during the preceding sleep. Blagrove et al. (1998) found a small correlation between actigraph-detected movements during night and subjective sleep quality, but in this study, we did not observe connections between sleep analysis variables and subjective ratings.

Fragmentation of the sleep. The workday sleeps were categorized by fragmentation index, an index of restlessness, for the 4-hour period at the early stage of the sleep. This was done because this is the phase of sleep that contains most of the slow wave sleep (SWS), which is considered to be the most restorative phase of sleep. During SWS the energy stores of the brain are fulfilled and growth hormone secretion is increased, and breathing frequency and heart rate are lowered – relaxation is therefore perfect. SWS mostly occurs during the first 4 to 5 hours of sleep, and it accounts for approximately 25% of the total sleep duration. (Härmä & Sallinen 2004, Horne 1989.) It has been hypothesized that at least four hours of sleep is needed for recovery and normal cognitive processes (Harrison & Horne 2000). There were eleven subjects (n=11) used in the statistical analysis in whom actigraphic actual sleep time, sleep efficiency, and number of awakenings during the 4-hour period, as well as fragmentation index for both the 4-hour period and the whole sleep differed significantly. FBT-PRO detected significantly longer relaxation time during the 4-hour period and during the whole sleep in less than more fragmented sleep. There was also a tendency towards less stress time during the 4-hour period and during the whole sleep in less than more fragmented sleep. In addition, relaxation/stress –balance differed significantly during the 4-hour period. However, there were no other differences than greater LF power during the 4-hour period in more than less fragmented sleep in the traditional HRV variables. No differences were seen in any cortisol values in the morning.

Therefore, these results give evidence that restlessness during sleep would be associated with reduced relaxation time and increased stress time detected with this new method. Moreover, restlessness during sleep seems to be associated with a change in the balance of relaxation and stress in favour of stress state. Sleep fragmentation has been shown to cause alterations in sleep architecture by increasing stage 1 sleep along with decreasing SWS and REM-sleep (Bonnet & Arand 2003, Spiegel et al. 2005), although some studies have provided evidence that it is the disturbance of sleep continuity rather than changes in sleep stages which produces the effects of sleep fragmentation (Bonnet &

Arand 2003). Sleep fragmentation may increase sleepiness during daytime or decrease psychomotor performance, even though total sleep time may be only minimally reduced (Bonnet & Arand 2003), or cause metabolic disturbances such as obesity or Type 2 diabetes (Spiegel et al. 2005). A possible interpretation of these findings may be that sleep fragmentation increases sympathetic nervous system activity and metabolic rate, affecting e.g. catecholamine and cortisol secretion. It is to be mentioned that sleep loss even without sleep fragmentation seems to increase sympathetic activity (Spiegel et al. 2005.) The results of the present study give evidence that sleep fragmentation may be reflected with this new method, even though no changes were observed in cortisol secretion between sleeps. Further, it was surprising that the only difference in the traditional HRV variables was the greater LF power during the 4-hour period in more than less fragmented sleep. This may indicate that the activity level of the autonomic nervous system during sleep is not necessarily linked to body movements or that body movements may increase some components of HRV. However, the results of the present study give some evidence that the combined HR and HRV information of the new FBT-PRO indices may be more sensitive than the traditional HRV variables to evaluate whether the body is recovering or not. It can be concluded that the fragmentation of sleep may have caused changes in the proportion of different phases of sleep during the 4-hour period and/or increased sympathetic activity, which were better reflected with this new method than the traditional HRV variables.

Actual Sleep Time. Longer actual sleep time was due to earlier bed time because no difference was seen in awakening time. The length of the actual sleep time differed on average an hour (7:13 versus 6:13). Longer actual sleep time was associated with greater relaxation time, but no differences were found in stress time. Also RMSSD was significantly greater during workday sleep with longer actual sleep time. These findings indicate that the duration of sleep is a very important factor for physiological recovery to occur. It is generally assumed that insomnia-related symptoms, sleepiness and fatigue follow a U-shaped distribution as a function of the duration of sleep (e.g. Kronholm et al. 2006, Grander & Kripke 2004), indicating that the mid-range duration (about 7–8 hours of sleep per night depending on a study) seems to be the most beneficial for health and well-being. Especially short night-sleep has been shown to be a risk factor for health. For example Kripke et al. (2002) showed that very short sleep duration (less than 4.5 hours) was associated with increased risk for mortality distinct of co-

morbidities in a study sample of a 1.1 million persons. Acute sleep deprivation and/or accumulated sleep debt has been linked to metabolic (e.g. overweight, obesity, and glucose intolerance) and cardiovascular diseases (e.g. increased risk for coronary events or hypertension). The reason for the finding that also excessive duration of sleep is associated with increased mortality is not clear and may reflect some other abnormalities, such as diseases or psychological problems. Insufficient sleep seems to be especially common in shift workers. (Wolk et al. 2005.) In the present study, the amount of sleep during the working period was relatively low, followed by a recovery sleep during the day off, as mentioned earlier. A short duration of workday sleep seems to have an impact on recovery, as better recovery (longer relaxation time) was achieved during longer workday sleep compared to shorter workday sleep. On the other hand, Rook & Zijlstra (2006) found that there were no differences in recovery (decrease in the feelings of fatigue) when accounting for the duration of sleep, but the amount of sleep plays a role in maintaining sleep quality, and therefore affecting recovery indirectly. Further, the results of the present study seem to suggest that the duration of sleep is a more important factor for recovery than the subjective ratings of sleep, as significant differences in stress and relaxation were found during sleeps with different actual sleep times but not during sleeps with different subjective ratings. More evidence that the objective measurements of the quality of sleep are needed was obtained from the comparison of less and more fragmented sleep, as restlessness during sleep was associated with lower relaxation and higher stress indices. However, it should be kept in mind that the number of subjects was low in the comparison of subjectively different sleeps.

We could observe that the magnitudes of cortisol awakening responses tended to be smaller after sleep with greater actual sleep time. Also the increase and change in cortisol values ($AUC_{i\ 0\text{-}60\ min}$ and $AUC_{c\ 0\text{-}60min}$) were significantly smaller after sleep with longer actual sleep time. In this comparison the bed time differed but awakening time did not, and the subjects were all women, so these variables cannot explain the differences found. Schlotz et al. (2004) reported slightly higher cortisol levels at awakening and a slightly smaller mean increase in the subjects who self-reported having slept longer. Also Wüst et al. (2000) reported a slightly smaller cortisol awakening response in subjects who reported a longer night-sleep, but it explained less than one percent of the variability in free cortisol levels after awakening. Späth-Schwalbe et al.

(1992) found in a sleep-laboratory study that during undisturbed sleep and after spontaneous awakening, cortisol levels were lower and awakening responses higher in long sleepers and vice versa in short sleepers. This may indicate that pre-stimulus levels of cortisol can have a modulatory effect on stimulated cortisol release. In the present study, the compared sleeps were both on workdays with relatively early awakening, but the effect of spontaneous versus interrupted awakening cannot be excluded.

It should be kept in mind that in this study we did not notice significant differences in cortisol values between workday and day off, even though more relaxation was detected during the day off sleep, and the difference in the duration of sleeps was greater in that comparison than in the comparison based on actual sleep time (where workday sleeps were used). However, awakening time differed in the workday 2 versus day off comparison, but in the comparison between workday 1 and 2, there were not any differences in awakening time. It is difficult to find any other reasonable explanations for the findings other than similar stress level (on average) during the day off and workday 2 mornings, resulting in similar cortisol secretion patterns despite of longer relaxation time during day off sleep, or that the awakening time had affected the cortisol results during day off.

Cortisol at awakening ($AC_{0\ min}$) and cortisol awakening response ($CAR_{0\text{-}60\ min}$). In the present study, there tended to be less stress time (91±59 min versus 128±109 min, p=0.075) during sleep with lower awakening cortisol, but no difference was found in relaxation time. It should, however, be noticed that also actual sleep time tended to be shorter (6:28±0:42 versus 6:58±1:06 h:min, p=0.168) during sleep with lower awakening cortisol, and this may explain the finding of less stress time to some extent. It was also found that there was a greater awakening response, as well as an increase and change in cortisol AUC after a sleep with lower cortisol at awakening ($AC_{0\ min}$).

It was found in the present study that the lower awakening response ($CAR_{0\text{-}60\ min}$) was associated with a smaller increase and change in cortisol AUC after awakening. Even though the actual sleep time was longer and the sleep efficiency greater during sleep with smaller than with greater cortisol awakening response, there were not any differences in stress and relaxation detected or in HRV between these sleeps.

Most of the studies regarding stress and cortisol secretion have focused on experimental laboratory stress or stress in cross-sectional studies dealing with selected groups (e.g. subjects with burnout or chronic fatigue syndrome). De Vente et al. (2003) reported that burnout subjects showed an elevated HR at rest and higher cortisol values from awakening until 30 minutes from awakening compared to healthy controls, which may be indicative of sustained activation in burnouts. Similarly, Grossi et al. (2005) showed elevated morning cortisol levels from awakening to one hour from awakening in female burnout patients compared to women reporting low levels of burnout. However, Pruessner et al. (1999) found that higher perceived stress in male and female teachers was correlated with greater increases in cortisol values during the first hour after awakening (sampling at awakening, 15, 30, and 60 minutes after), whereas burnout was associated with lower overall cortisol levels. Moreover, Roberts et al. (2004) found a lower cortisol awakening response in chronic fatigue syndrome patients. Schulz et al. (1998) reported that chronically stressed subjects had a significantly greater increase in cortisol after awakening than less stressed subjects, and that chronically stressed women showed more pronounced elevations in cortisol than men. Backhaus et al. (2004) reported that persons suffering from chronic disturbances of sleep (duration of insomnia 11.6±11.2 years) had lower awakening cortisol. The reason for this finding according to Backhaus and co-workers was that awakenings during sleep are associated with increased cortisol activation, which results in decreased HPA-axis activation after awakening. Lundberg & Hellström (2002) found significant positive correlations between workload (amount of overtime work) and morning cortisol levels from awakening to 45 minutes from awakening in more than 200 Swedish women. The results of the studies focusing on connection of acute or chronic stress and secretion of cortisol are, therefore, inconsistent. Despite of contradictory findings, it seems that persons suffering from burnout or chronic fatigue syndrome have disturbed HPA-axis function, resulting in either elevated or reduced cortisol values after awakening.

Surprisingly, data on longitudinal studies of real life stress and cortisol secretion in healthy and non-burnout population seems to be absent. To my knowledge, there has not been a single longitudinal study concerning cortisol secretion and stress. However, Dahlgren et al. (2004) found a flattened cortisol pattern but a tendency towards higher values during the day and bed time in the high versus low stress condition. Kunz-Ebrecht et al. (2004) found that salivary cortisol levels on waking did not differ by

gender or socioeconomic position, or between work and weekend days, but awakening responses were higher on work than on weekend days, which may indicate that cortisol output early in the day is sensitive to the influence of work-related stress and its anticipation. It has also been presented by Powell et al. (2002) that evening cortisol would be a promising marker of chronic stress in middle-aged women. On the other hand, according to Powell and co-workers there has been much diversity in study designs and it has been presented that potential markers of chronic stress would include also elevated 24-h cortisol, suppression of 24-h cortisol, elevated overnight cortisol, elevated morning cortisol as well as catecholamine measurements. Therefore, it seems that there does not exist a consensus for reliable use of cortisol in stress research currently, which makes it difficult to interpret the results.

However, the results of the present study give some support for the speculations that a higher baseline level of cortisol might reduce the net stress response to some degree, as presented also earlier (Kudielka et al. 2004a). The results of the present study may also indicate that the detection of stress and relaxation based on HR and HRV may be better associated with the cortisol value at awakening rather than cortisol awakening responses, but further studies are definitely needed to confirm that.

7.5 Limits of the HRV-based method to evaluate stress and recovery

There are some factors that may affect the results of the HRV-based analysis method. It has been shown that various factors affect HR and HRV, including e.g. age, gender (e.g Umetani et al. 1998), and physical fitness (e.g Gulli et al. 2003, Gallagher et a. 1992), but also heredity (e.g. Kupper et al. 2004). It has been shown in several studies that HR and HRV decrease with increasing age (e.g. Antelmi et al. 2004, Umetani et al. 1998), but better physical fitness has mostly been associated with greater HRV (e.g. Buchheit & Gindre 2006, Aubert et al. 2003). Women have been shown to have augmented parasympathetic and attenuated sympathetic modulation reflected in HRV compared to men (e.g. Huikuri et al. 1996). Therefore, if the analysis of stress and recovery is conducted for the first time, it is advisable to do an RR-interval measurement of a long period of time, which contains both sleep and awake-time to get correct information about subjects' physiological background parameters, e.g. resting heart rate. If correct

background parameters are used, as was the case in the present study, the results should be more accurate. In the present study, a maximal bicycle ergometer test for the subjects was applied to get correct information about the maximal heart rate of the subjects. Also all of the measured 24-hour periods were analyzed first, and the lowest heart rate was used as the resting heart rate when the measured periods were analyzed again for the statistical analyses. The inclusion of rest and night-sleep for the measured period is therefore very advisable.

The traditional HRV results are very individual. Therefore, it can be assumed that the new method would be the most suitable for within subject study designs, at least if the number of subjects is relatively low, as was the case in this study. In this study, subjects of different ages were studied, but on the other hand, the study was performed as a within-subject design so that the same subjects were compared at different time periods, which should eliminate the effect of differences between subjects. On the other hand, the new method takes the individual level of HRV into account, and stress and relaxation are calculated in relation to each person's individual level and range of HRV and background parameters. Therefore, it is important to use the correct individual background parameters, as was done in the present study. Finally, there are no reference levels reported in the user guide of FBT-PRO to compare the achieved values of stress and relaxation, i.e. whether there has been enough recovery during the measured period. Another problem in interpreting the results may be the difficulty to find out an individual reference or "baseline" level to compare the results e.g. from several measurements.

Physical activity during the recording can affect the reliability of the results because it is impossible to detect psychological stress reactions during exercise and recovery from exercise. Also, relaxation may not be recognized during sleep if there has been physical activity just prior to going to bed, increasing the activity of ANS during early phases of sleep. Also illnesses and pharmacological substances used by subjects may affect HR and HRV, and at least make it more difficult to interpret the achieved results. An example on the effects of sickness on the results can be given based on the data of this study. One subject caught flu during the study, and when she was sick, only 36 minutes of relaxation and 435 minutes of stress was detected during night-sleep. On the day when she was healthy again, 305 minutes of relaxation and 24 minutes of stress was

detected during night-sleep. This concretizes the meaning of illnesses on the analysis. Of course, this sick day was not used in the statistical analyses of this study.

Another important factor is the technical side of the measurement. The quality of RRI-recording, percentage of error in the data and automatic correction of the data can all affect results. An ideal situation would be perfect data with no artefacts caused by technical apparatus, but that is very difficult to achieve in ambulatory real life measurements.

7.6 Limits of the study

There are some limitations that need to be considered when interpreting the results. The number of subjects could have been higher because there were only 14 subjects who had good enough heart rate data and did not have illnesses affecting the function of ANS. In addition, in the comparison of subjectively different sleeps, there were only 7 subjects in whom the workday sleeps were subjectively different. It is also possible that recovery during the sleeps which were used in the comparison was not different enough to get more significant results with this number of subjects. Moreover, there were not enough nights to find out if the values in the subjects' sleep analysis variables were within their normal range, i.e. how representative their studied night-sleeps were compared to their normal sleep.

It should also be noted that the "first night effect" could have had an impact on the results as the subjects may have experienced more stress and qualm because of the excitement caused by the measurement, instructions to follow, and new devices to carry during workday 1. Because of the real life setting of the study, we can't be 100% sure about the self-reported bed times of the subjects. In some cases, activity monitoring had to be used to confirm when a person had gone to bed. We tried to avoid this methodological problem by examining also the 4-hour period in the early stage of sleep starting 30 minutes after the determined bed time in addition to the whole sleep. In addition, because of the real life nature of the study, the effects of increased physiological arousal caused by possible consumption of caffeinated drinks (coffee, tea)

or alcohol, or smoking during the measured period were not controlled in the statistical analysis, which may have affected the results.

For interpretation of cortisol results, it has to be remembered that the adherence to instructions for taking cortisol samples is a very important issue, especially regarding cortisol values in the morning and awakening responses. The samples should be taken with strict reference to awakening (e.g. Clow et al. 2004). It has, however, been shown that in the ambulatory measurement the adherence may not be sufficient, as 20–30% of the subjects might not follow instructions (Thorn et al. 2006, Broderick et al. 2004). In this study, the subjects were asked to follow the instructions (appendix 4), and to refrain from eating or drinking 15 minutes before taking a cortisol sample. Later on it was checked from the data that the self-reported sampling times of the first cortisol samples matched the awakening times reported by the subjects. If there was a difference in sampling times, it was mathematically taken into account in calculations of the areas under the curve. As a whole, the timing of taking the samples succeeded well, but the possibility of non-adherence to the instructions cannot be excluded in this study. It is also possible that flatter cortisol profiles during weekends, which have been reported in the literature, may not account for reduced stress (e.g. Kunz-Ebrecht et al. 2004) but for non-adherence to the instructions (Thorn et al. 2006). Similarly to Thorn et al. (2006), no differences in cortisol values between workday and day off were found in the present study, but in the present study no subjects were removed from the analyses because of not showing an increase in cortisol after awakening.

The subjects were also allowed to follow their normal habits in the morning with no other restrictions than the 15 minutes without food and drink before taking the saliva samples. Therefore, it is possible that during the 60-minute period when the samples were taken in the morning, some subjects may, for example, have cycled to work by bike. It could, theoretically, be possible that very high intensity exercise could have stimulated cortisol secretion when sympathetic nerve activity is high and catecholamines in the blood increased (Chicharro et al. 1998). However, Jacks et al. (2002) reported that only exercise with high intensity (76% of VO_{2max}) and long duration (>40 minutes) results in significant elevations in saliva cortisol. Consequently, exercise has probably not affected the results of the present study.

One shortcoming of the study may be that only the physiological variables (besides subjective ratings of sleep) related to stress and recovery were dealt with and psychological variables, such as self-reported level of different moods (relaxed, stressed, and irritated in a scale from 1 to 6, i.e. not at all – very much) in the morning were ignored. However, the purpose of the study was to compare different physiological methods to analyze stress and recovery, rather than to combine physiological and psychological variables. As far as subjective ratings of the quality of sleep are concerned, it is worth noticing that the 3-point scale used in the present study might not have been the best for its purpose. With a broader gradual or continuous scale, or using a more detailed questionnaire about the quality of sleep, the present study might have provided us deeper understanding about the issue of subjective versus objective sleep quality.

7.7 Conclusions

This study indicated that stress and recovery can be detected based on ambulatory R-R interval recordings and heart rate and heart rate variability –based analysis. Stress state dominated awake-time, and relaxation state dominated sleep. There was also some time in physical activity and recovery from physical activity states detected during awake, indicating that the nursing work is physically demanding. As sleep is the most important period for recovery of an individual, it seems to be a suitable period for the measurement of recovery. This assumption was supported by the results, as very little relaxation state was detected during awake, but on the other hand, quite a lot of time in stress state, with a lot of variation, was detected during sleep. Therefore, these results indicate that the sympathetic activity of the autonomic nervous system is dominating during awake-time, but quite often also during sleep. Because recovery occurs when the body is in a relaxed state and parasympathetic activity is dominating, sustained sympathetic activity or decreased parasympathetic modulation of the heart after bed time results in impaired recovery during sleep.

Workday 1 and 2 did not differ from each other in detected stress or relaxation, but during day off and during workday sleep with longer actual sleep time, more relaxation time (and therefore better recovery) was achieved particularly through longer night-

sleep. Increased duration of the sleep seems to be associated with greater duration of relaxation state, and increased restlessness (fragmentation index) during sleep to be associated with shorter duration of relaxation state and greater duration of stress state. These results emphasize the meaning of sleep duration and quality for recovery. Therefore, it is not only the quantity or the quality of sleep, but both which have an impact on recovery. The results of the present study show that when a person sleeps longer, the body is presumably longer in relaxation state. It seems that during a short sleep the body does not recover properly – instead a longer sleep is required for adequate physiological recovery to take place. This may be of great importance especially nowadays, when many people generally tend to sleep shorter than the recommended 7 to 9 hours a day, either voluntarily or involuntarily.

Interestingly, there were no differences on the day off in the traditional HRV variable mean values during sleep, including the average LF/HF ratio, even though the subjects were significantly longer in relaxation state. On the other hand, there was a significantly greater RMSSD-value (in addition to greater relaxation time) during workday sleep with longer than with shorter actual sleep time. Restlessness during sleep (fragmentation index) had little effect on the traditional HRV variables compared to the new stress and relaxation indices. These results, therefore, give some evidence that this new HR and HRV –based method improves the accuracy of analyzing the physiological state of the body compared to the traditional HRV variables. It is difficult to say whether the sympathetic or parasympathetic activity of the autonomic nervous system is dominating by using the traditional HRV variables, but it seems to be possible by using this new method.

Subjective differences in the quality of sleep did not seem to have an impact on stress or relaxation in the present study. This may indicate that there were some other factors influencing the subjective opinion of the sleep than the autonomic modulation of the heart, but the low number of subjects and the rough 3-point scale in subjective ratings may also have influenced the results. The lower cortisol value at awakening seemed to be preceded by sleep with less stress time compared to sleep with greater cortisol at awakening, but the cortisol awakening response was not as good indicator of stress as cortisol value at awakening in this study. Taking into account that the results of the

earlier studies of stress and cortisol secretion have yielded inconsistent information, it is difficult to interpret the interplay between recovery and secretion of cortisol.

This study was carried out on healthy working population and the aim of the study was to compare the same subjects within different study approaches to measure stress and recovery. The subjects did not report any significant amount of work-related stress and they did not show any symptoms of burnout based on psychological questionnaires. Given that, the results gave some evidence that it is possible to differentiate recovery during sleep also in ambulatory real life measurements in healthy population. Further studies are needed with a greater number of subjects and different subject groups (e.g. athletes) for stronger conclusions of the ability of this new heart rate and heart rate variability –based method to detect acute and chronic stress, and health-maintaining recovery periods during awake and sleep compared to other physiological methods.

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Tero

REFERENCES

Aardal, E. & Holm. A. C. 1995. Cortisol in saliva – reference ranges and relation to cortisol in serum. European Journal of Clinical Chemistry 33 (12): 927–933.

Aasa, U., Kalezic, N., Lyskov, E, Ängquist, K-A. & Barnekow-Bergkvist, M. 2006. Stress monitoring of ambulance personnel during work and leisure time. International Archives of Occupational and Environmental Health 80 (1): 51–59.

Achten, J. & Jeukendrup, A. E. 2003. Heart Rate Monitoring – Applications and Limitations. Sports Medicine 33 (7): 517–538. Review.

ACSM – American College of Sports Medicine. 2001. ACSM's Guidelines for Exercise Testing and Prescription. Philadelphia: Lippincott Williams & Wilkins.

Ahola, K., Honkonen, T., Isometsä, E., Kalimo, R., Nykyri, E., Koskinen, S. Aromaa, A. & Lönnqvist, J. 2006. Burnout in the general population - Results from the Finnish Health 2000 Study. Social psychiatry and psychiatric epidemiology 41: 11–17.

An American Sleep Disorders Association Report 1995. Practice Parameters for the Use of Actigraphy in the Clinical Assessment of Sleep Disorders. Sleep 18 (4): 285–287.

Ancoli-Israel, S., Cole, R., Alessi, C., Chambers, M., Moorcroft, W. & Pollak, C. P. 2003. The Role of Actigraphy in the Study of Sleep and Circadian Rhythms. Sleep 26 (3): 342–392.

Angeli, A., Minetto, M., Dovio, A. & Paccotti, P. 2004. The overtraining syndrome in athletes: a stress-related disorder. Journal of Endocrinological Investigation 27 (6): 603–612.

Antelmi, I., de Paula, R. S., Shinzato, A. R., Peres, C. A., Mansur, A. J. & Grupi, C. J. 2004. Influence of age, gender, body mass index, and functional capacity on heart rate variability in a cohort of subjects without heart disease. American Journal of Cardiology 93 (3): 381–385.

Aubert, A. E., Seps, B. & Beckers, F. 2003. Heart Rate Variability in Athletes. Review. Sports Medicine 33 (12): 889–919.

Backhaus, J., Junghanns, K. & Hohagen, F. 2004. Sleep disturbances are correlated with decreased morning awakening salivary cortisol. Psychoneuroendocrinology 29 (9):1184–1191.

Baker, F. C, Maloney, S. & Driver, H. S. 1999. A comparison of subjective estimates of sleep with objective polysomnographic data in healthy men and women. Journal of Psychosomatic Research 47: 335–341.

Bartels, M., Van den Berg, M., Sluyter, F., Boomsma, D. I. & de Geus, E. J. C. 2003. Heritability of cortisol levels: review and simultaneous analysis of twin studies. Psychoneuroendocrinology 28: 121–137.

Beckers, F., Verheyden, B. & Aubert, A. E. 2006. Aging and nonlinear heart rate control in a healthy population. American Journal of Physiology: Heart & Circulatory Physiology 290: 2560–2570.

Bernard, T., Gavarry, O., Bermon, S., Giacomoni, M., Marconnet, P. & Falgairette, G. 1997. Relationships between oxygen consumption and heart rate in transitory and steady states of exercise and during recovery: influence of type of exercise. European Journal of Applied Physiology: Occupational Physiology 75: 170–176.

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

Bigger J. T., Fleiss, J. L., Rolnitzky, L. M. & Steinman, R. C. 1993. The ability of several short-term measures of RR variability to predict mortality after myocardial infarction. Circulation 88: 927–934.

Bilan, A., Witczak, A., Palusinski, R., Myslinski, W & Hanzlik, J. 2005. Circadian rhythm of spectral indices of heart rate variability in healthy subjects. Journal of Electrocardiology 38: 239–243.

Blagrove, M., Owens, D. S., MacDonald, I., Sytnik, N., Tucker, P. & Folkard, S. 1998. Time of day effects in, and the relationship between, sleep quality and movement. Journal of Sleep Research 7: 233–239.

Bonnet, M. H. & Arand, D. L. 2003. Clinical effects of sleep fragmentation versus sleep deprivation. Clinical Review. Sleep Medicine Reviews 7 (4): 297–310.

Broderick, J. E., Arnold, D., Kudielka, B.M. & Kirschbaum, C. 2004. Salivary cortisol

sampling compliance: comparison of patients and healthy volunteers. Psychoneuroendocrinology 29 (5): 636–650.

Buchheit, M. & Gindre, C. 2006. Cardiac parasympathetic regulation: respective associations with cardiorespiratory fitness and training load. American Journal of Physiology: Heart & Circulatory Physiology. 291 (1): 451–458.

Cambridge Neurotechnology. The Actiwatch Activity Monitoring System – Instructions for Use and Software Manual. Cambridge, UK.

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

Carrington, M., Walsh, M., Stambas, T., Kleiman, J. & Trinder, J. 2003. The influence of sleep onset on the diurnal variation in cardiac activity and cardiac control. Journal of Sleep Research 12: 213–221.

Cavanaugh, M. A., Boswell, W. R., Roehling, M. V. & Boudreau, J. W. 2000. An empirical examination of self-reported work stress among U.S. managers. Journal of Applied Psychology 85: 65–74.

Cesana, G., Sega, R., Ferrario, M., Chiodini, P., Corrao, G. & Mancia, G. 2003. Job strain and blood pressure in employed men and women: a pooled analysis of four northern italian population samples. Psychosomatic Medicine 65: 558–563.

Charmandari, E., Tsigos, C. & Chrousos, G. 2005. Endocrinology of the Stress Response. Annual Reviews of Physiology 67: 259–284. Review.

Chen, K. Y & Bassett, D. R. Jr. 2005. The Technology of Accelerometry-Based Activity Monitors: Current and Future. Medicine & Science in Sports & Exercise Vol. 37, (Suppl) 490–500.

Cheng, Y., Kawachi, I., Coakley, E. H., Schwartz, J. & Colditz, G. 2000. Association between psychosocial work characteristics and health functioning in American women: prospective study. British Medical Journal 320: 1432–1436.

Chicharro, J. L., Lucia, A., Perez, M., Vaquero, A. F. & Urena, R. 1998. Saliva composition and exercise. Review. Sports Medicine 26 (1): 17-27.

Clow, A., Thorn, L., Evans, P. & Hucklebridge, F. 2004. The Awakening Cortisol Response: Methodological Issues and Significance. Stress 7 (1): 29–37.

Collins, S. M., Karasek, R. A. & Costas, K. 2005. Job Strain and Autonomic Indices of Cardiovascular Disease Risk. American Journal of Industrial Medicine 48: 182–193.

Craig, A. & Cooper, R. E. 1992. Symptoms of Acute and Chronic Fatigue. In Smith, A. P. & Jones, D. M. (Eds.) Handbook of Human Performance (Volume 3, State and Trait, pp 289–339). Academic Press, London.

Cropley, M., Dijk, D-J & Stanley, N. 2006. Job strain, work rumination, and sleep in school teachers. European Journal of Work and Organizational Psychology 15: 181–196.

Dahlgren, A., Åkerstedt, T. & Kecklund, G. 2004. Individual differences in the diurnal cortisol response to stress. Chronobiology International 21 (6): 913–922.

Dahlgren, A., Kecklund, G. & Åkerstedt, T. 2005 Different levels of work-related stress and the effects on sleep, fatigue and cortisol. Scandinavian Journal of Work, Environment and Health 31(4): 277–285.

Davies, C.T.M, Di Prampero, P.E. & Cerretelli, P. 1972. Kinetics of cardiac output and respiratory gas exchange during exercise and recovery. Journal of Applied Physiology 32: 618–625.

De Vente, W., Olff, M., Van Amsterdam, J. G. C., Kamphuis, J. H. & Emmelkamp, P. M. G. 2003. Physiological differences between burnout patients and healthy controls: blood pressure, heart rate, and cortisol responses. Occupational and Environmental Medicine 60: i54–i61.

Dekker, J. M., Schouten, E. G., Klootwijk, P., Pool, J., Swenne, C. A. & Kromhout, D. 1997. Heart rate variability from short electrocardiographic recordings predicts mortality from all causes in middle-aged and elderly men. The Zutphen Study. American Journal of Epidemiology 145: 899–908.

Dobkin, P. L. & Pihl, R. O. 1992. Measurement of Psychological and Heart Rate Reactivity to Stress in the Real World. Psychotherapy & Psychosomatics 58: 208–214.

Dronjak, S. & Gavrilovic, L. 2006. Effects of stress on catecholamine stores in central and peripheral tissues of long-term socially isolated rats. Brazilian Journal of Medical and Biological Research 39: 785–790.

Earle, T. L., Linden, W. & Weinberg, J. 1999. Differential effects of harassment on cardiovascular and salivary cortisol stress reactivity and recovery in women and men. Journal of Psychosomatic Research 46: 125–141.

Eckberg, D. L. 1997. Sympathovagal Balance: A Critical Appraisal. Circulation 96: 3224–3232.

Edéll-Gustafsson, U. M., Kritz, E. I. K. & Bogren, I. K. 2002. Self-reported sleep

quality, strain and health in relation to perceived working conditions in females. Scandinavian Journal of Caring Sciences 16: 179–187.

Edwards, S., Evans, P., Hucklebridge, F. & Clow, A. 2001. Association between time of awakening and diurnal cortisol secretory activity. Psychoneuroendocrinology 26: 613–622.

Ekstedt, M. 2005. Burnout and Sleep. Academic Dissertation. Karoliska Institutet, Stockholm, Sweden.

Eller, N. H., Netterstrom, B. & Hansen, Å. M. 2006. Psychosocial factors at home and at work and levels of salivary cortisol. Biological Psychology. Article in press.

European Foundation for the Improvement of Living and Working Conditions 2005. Work-related stress. Report. Available in electric format: http://www.eurofound.ie/ewco/reports/NL0502TR01/NL0502TR01.pdf

Federenko, I., Wüst, S., Hellhammer, D. H., Dechoux, R., Kumsta, R. & Kirschbaum, C. 2004. Free cortisol awakening responses are influenced by awakening time. Psychoneuroendocrinology 29: 174–184.

Firstbeat Technologies 2005. VO_2 Estimation Method Based on Heart Rate Measurement. Available in electric format: http://www.firstbeat.fi/files/VO2_Estimation.pdf

Firstbeat Technologies Oy. The Handbook of Wellness Analysis Software. In Finnish: Firstbeat Hyvinvointianalyysi. Käsikirja -versio 1.4.0.14.

Gaab, J., Hüster, D., Peisen, R., Engert, V., Heitz, V., Schad, T., Schürmeyer, T.H. & Ehlert, U. 2002. Hypothalamic–pituitary–adrenal axis reactivity in chronic fatigue syndrome and health under psychological, physiological, and pharmacological stimulation. Psychosomatic Medicine 64: 951–962.

Gallagher, D., Terenzi, T. & de Meersman, R. 1992. Heart rate variability in smokers, sedentary and aerobically fit individuals. Clinical Autonomic Research 2 (6): 383–387.

Garde, A. H., Laursen, B., Jorgensen, A. H. & Jensen, B. R. 2002. Effects of mental and physical demands on heart rate variability during computer work. European Journal of Applied Physiology 87: 456–461.

Geurts, S. A. E., Taris, T. W., Kompier, M. A. J., Dikkers, J. S. E., van Hooff, M. L. M. & Kinnunen, U. M. 2005. Work-home interaction from a work psychological perspective: Development and validation of a new questionnaire, the SWING. Work & Stress 19 (4): 319–339.

Gleeson, M. 2002. Biochemical and Immunological Markers of Overtraining. Review. Journal of Sports Science and Medicine 1: 31–41.

Goldberger, J. J., Challapalli, S., Tung, R., Parker, M. A. & Kadish, A. H. 2001. Relationship of heart rate variability to parasympathetic effect. Circulation 103 (15): 1977–1983.

Gottlieb, D. J., Redline, S., Nieto, J., Baldwin, C. M., Newman, A. B., Resnick, H. E. & Punjabi, N. M. 2006. Association of usual sleep duration with hypertension: The Sleep Heart Health Study. Sleep 29: 1009–1014.

Grander, M. A. & Kripke, D. F. 2004. Self-reported sleep complaints with long and short sleep: a nationally representative sample. Psychosomatic Medicine 66 (2): 239–241.

Grossi, G., Perski, A., Ekstedt, M., Johansson, T., Lindström, M. & Holm, K. 2005. The morning salivary cortisol response in burnout. Journal of Psychosomatic Research 59: 103–111.

Guasti, L., Simoni, C., Mainardi, L., Crespi, C., Cimpanelli, M., Klersy, C., Gaudio, G., Grandi, A. M., Cerutti, S. & Venco, A. 2005. Global link between heart rate and blood pressure oscillations at rest and during mental arousal in normotensive and hypertensive subjects. Autonomic Neuroscience: Basic and Clinical 120: 80–87.

Gulli, G., Cevese, A., Cappelletto, P., Gasparini, G. & Schena, F. 2003. Moderate aerobic training improves autonomic cardiovascular control in older women. Clinical Autonomic Research 13 (3):196–202.

Guyton, A. C. & Hall, J. E. 2000. Textbook of medical physiology. W. B. Saunders Company, Philadelphia. Tenth edition.

Hall, M., Vasko, R., Buysse, D., Ombao, H., Chen, Q., Cashmere, J. D., Kupfer, D. & Thayer, J. F. 2004. Acute Stress Affects Heart Rate Variability During Sleep. Psychosomatic Medicine 66: 56–62.

Harrison, Y. & Horne, J. A. 2000. The Impact of Sleep Deprivation on Decision Making: A Review. Journal of Experimental Psychology: Applied 6 (3): 236–249.

Hautala, A. 2004. Effect of physical exercise on autonomic regulation of heart rate. Academic dissertation. Department of Internal Medicine, University of Oulu.

Hjortskov, N. Rissén, D, Blangsted, A. K., Fallentin, N., Lundberg, U. & Sogaard, K.

2004. The effect of mental stress on heart rate variability and blood pressure during computer work. European Journal of Applied Physiology 92: 84–89.

Horne, J. 1989. Why we sleep: the functions of sleep in humans and other mammals. 1st paperback ed. Oxford University Press, New York. 319 p.

Hui, L., Ng, G. Y. F., Yeung, S. S. M. & Hui-Chan, C. W. Y. 2001. Evaluation of physiological work demands and low back neuromuscular fatigue on nurses working in geriatric wards. Applied Ergonomics 32 (5): 479–483.

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

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

Hynynen, E., Nummela, A., Rusko, H., Hämäläinen, I. & Jylhä, R. 2006b. Effects of training on cardiac autonomic modulation during night sleep in cross country skiers. Abstract. International Congress on Science and Nordic Skiing 2006, Vuokatti, Finland.

Härmä, M. & Sallinen, M. 2004. Hyvä uni – hyvä työ. Työterveyslaitos, Helsinki. 1.-2. painos, Vammalan kirjapaino Oy.

Ice, G. H., Katz-Stein, A., Himes, J. & Kane, R. L. 2004. Diurnal cycles of salivary cortisol in older adults. Psychoneuroendocrinology 29 (3): 355–370.

Ito, H., Nozaki, M., Maruyama, T, Kaji, Y. & Tsuda, Y. 2001. Shift work modifies the circadian patterns of heart rate variability in nurses. International Journal of Cardiology 79: 231–236.

Isowa, T., Ohira, H. & Murashima, S. 2006. Immune, endocrine and cardiovascular responses to controllable and uncontrollable acute stress. Biological Psychology 71: 202–213.

Jacks, D. E., Sowash, J., Anning, J., McGloughlin, T. & Andres, F. 2002. Effect of exercise at three exercise intensities on salivary cortisol. Journal of Strength and Conditioning Research 16 (2): 286–289.

Jackson, A., Blair, S., Mahar, M., Weir, L., Ross, R. & Stuteville, J. 1990. Prediction of functional aerobic capacity without exercise testing. Medicine and Science in Sports and Exercise 22 (6): 863–870.

Jean-Louis, G., Kripke, D. F. & Ancoli-Israel, S. 2000. Sleep and Quality of Well-Being. Sleep 23: 1–7.

Josten, E. J. C., Ng-A-Tham, J. E. E. & Thierry, H. 2003. The effects of extended workdays on fatigue, health, performance and satisfaction in nursing. Journal of Advanced Nursing 44 (6): 643–652.

Karasek, R. A. 1979. Job Demands, Job Decision Latitude, and Mental Strain: Implications for Job Redesign. Administrative Science Quarterly 24 (2): 285–308.

Kario, K., Schwartz, J. E. & Pickering, T. G. 1999. Ambulatory physical activity as a determinant of diurnal blood pressure variation. Hypertension 34: 685–691.

Kawakami, N., Haratani, T. & Araki, S. 1998. Job strain and arterial blood pressure, serum cholesterol, and smoking as risk factors for coronary heart disease in Japan. International Archives of Occupational and Environmental Health 71:429–432.

Kelly, L. A., Reilly, J. J., Fairweather, S. C., Barrie, S., Grant, S & Paton, J. Y. 2004. Comparison of two Accelerometers for Assessment of Physical Activity in Preschool Children. Pediatric Exercise Science 16: 324–333.

Kettunen, J. & Saalasti, S. 2004. Procedure for detection of stress by segmentation and analyzing heart beat signal. United States Patent Application 20050256414, WO 2004/016172 A1.

Kettunen, J. & Saalasti, S. 2005. Procedure for deriving reliable information on respiratory activity from heart period measurement. United States Patent Application 20050209521.

Kilpatrick, K. & Lavoie-Tremblay, M. 2006. Shiftwork: what health care managers need to know. The Health Care Manager 2006. 25:160–166.

King, S. L. & Hegadoren, K. M. 2002. Stress Hormones: How Do They Measure Up? Biological Research for Nursing 4 (2): 92–103.

Kirschbaum, C. & Hellhammer, D. H. 1994. Salivary cortisol in psychoneuroendocrine research: recent developments and applications. Psychoneuroendocrinology 19(4): 313–333.

Kivimäki, M., Head, J., Ferrie, J. E., Brunner, E., Marmot, M. G., Vahtera, J. & Shipley, M. J. 2006. Why is evidence on job strain and coronary heart disease mixed? An illustration of measurement challenges in the Whitehall II study. Psychosomatic Medicine 68: 398–401.

Kripke, D. F, Garfinkel, L., Wingard, D. L., Klauber, M. R. & Marler, M. R. 2002. Mortality associated with sleep duration and insomnia. Archives of General Psychiatry 59: 131–136.

Kristal-Boneh, E., Raifel, M., Froom, P. & Ribak, J. 1995. Heart rate variability in health and disease. Scandinavian Journal of Work, Environment & Health 21: 85–95.

Kronholm, E., Härmä, M., Hublin, C., Aro, A. R. & Partonen, T. 2006. Self-reported sleep duration in Finnish general population. Journal of Sleep Research 15: 276–290.

Kryger, M. H., Roth, T. & Dement, W. C. 1989. Principles and Practice of Sleep Medicine. W. P. Saunders Company, Philadelphia.

Kudielka, B. M., Schommer, N. C., Hellhammer & D. H., Kirschbaum, C. 2004a. Acute HPA axis responses, heart rate, and mood changes to psychosocial stress (TSST) in humans at different times of day. Psychoneuroendocrinology 29: 983–992.

Kudielka, B. M., von Känel, R., Gander, M-L & Fischer, J. E. 2004b. Effort-reward imbalance, overcommitment and sleep in a working population. Work & Stress 18 (2): 167–178.

Kudielka, B. M., von Kanel, R., Preckel, D., Zgraggen, L., Mischler, K. & Fischer, J. E. 2006. Exhaustion is associated with reduced habituation of free cortisol responses to repeated acute psychosocial stress. Biological Psychology 72: 147–153.

Kunz-Ebrecht, S. R., Kirschbaum, C., Marmot, M. & Steptoe, A. 2004. Differences in cortisol awakening response on work days and weekends in women and men from the Whitehall II cohort. Psychoneuroendocrinology 29: 516–528.

Kuper, H. & Marmot, M. 2003. Job strain, job demands, decision latitude, and risk of coronary heart disease within the Whitehall II study. Journal of Epidemiology and Community Health 57: 147–153.

Kupper, N. H. M., Willemsen, G., van den Berg, M., de Boer, D., Posthuma, D., Boomsma, D. I. & de Geus, E. J. C. 2004. Heritability of Ambulatory Heart Rate Variability. Circulation 110: 2792–2796.

Kushida, C. A., Chang, A., Gadkary, C., Guilleminault, C., Carrillo, O. & Dement, W.

C. 2001. Comparison of actigraphic, polysomnographic, and subjective assessment of sleep parameters in sleep-disordered patients. Sleep Medicine 2. 389–396.

Landsbergis, P. A., Schnall, P. L., Belkic, K. L., Baker, D., Schwartz, J. & Pickering, T. G. 2001. Work stressors and cardiovascular disease. Work 17:191–208. Review.

Landsbergis, P. A., Schnall, P. L., Warren, K., Pickering, T. G. & Schwartz, J. E. 1994. Association between ambulatory blood pressure and alternative formulations of job strain. Scandinavian Journal of Work, Environment & Health 20: 349–363.

Lazarus, R. S. 1966. Psychological Stress and the Coping Process. New York: McGraw-Hill.

Lee, S., Colditz, G., Berkman, L. & Kawachi, I. 2002. A prospective study of job strain and coronary heart disease in US women. International Journal of Epidemiology 31: 1147–1153.

Leproult, R., Colecchia, E. F., L'Hermite-Balériaux, M. & Van Cauter, E. 2001. Transitions from dim to bright light in the morning induces an immediate elevation of cortisol levels. Journal of Clinical Endocrinology & Metabolism 86: 151–157.

Lewis, J. G., Bagley, C. J., Elder, P. A., Bachmann, A. W. & Torpy, D. J. 2005. Plasma free cortisol fraction reflects levels of functioning corticosteroid-binding globulin. Clinica Chimica Acta 359: 189–194.

Littner, M., Kushida, C. A., McDowell Anderson, W., Bailey, D., Berry, R. B., Davila, D. G., Hirshkowitz, M., Kapen, S., Kramer, M., Loube, D., Wise, M. & Johnson, S. F. 2003. Practice Parameters for the Role of Actigraphy in the Study of Sleep and Circadian Rhythms: An Update for 2002. Sleep 26: 337–341.

Lockley, S. W., Skene, D. J. & Arendt, J. 1999. Comparison between subjective and actigraphic measurement of sleep and sleep rhythms. Journal of Sleep Research 8 (3): 175–183.

Lovallo, W. R. 2005. Cardiovascular reactivity: Mechanisms and pathways to cardiovascular disease. International Journal of Psychophysiology 58: 119–132.

Lucini, D., Norbiato, G., Clerici, M. & Pagani, M. 2002. Hemodynamic and Autonomic

Adjustments to Real Life Stress Conditions in Humans. Hypertension 39: 184–188.

Lucini, D., Di Fede, G., Parati, G. & Pagani, M. 2005. Impact of chronic psychosocial stress on autonomic cardiovascular regulation in otherwise healthy subjects. Hypertension 46 (5): 1201–1206.

Lundberg, U. 2005. Stress hormones in health and illness: The roles of work and gender. Review. Psychoneuroendocrinology 30 (10): 1017–1021.

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

Malliani, A. 2005. Heart rate variability: from bench to bedside. Review. European Journal of Internal Medicine 16: 12–20.

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

Martinmäki, K., Rusko, H., Kooistra, L., Kettunen, J. & Saalasti, S. 2006. Intraindividual validation of heart rate variability indexes to measure vagal effects on hearts. American Journal of Physiology: Heart and Circulatory Physiology 290: H640–647.

McEwen, B. 1998. Protective and damaging effects of stress mediators. The New England Journal of Medicine 338: 171–179.

Meijman, T. F. & Mulder, G. 1998. Psychological aspects of workload. In Handbook of Work and Organizational Psychology (eds. Drenth, P. J. D., Thierry, H., de Wolff, C. I.). 2nd ed. Psychology Press, Hove, 5–33.

Mourot, L., Bouhaddi, M., Perrey, S., Cappelle, S., Henriet, M. T., Wolf, J. P., Rouillon, J. D. & Regnard, J. 2004. Decrease in heart rate variability with overtraining: assessment by the Poincare plot analysis. Clinical Physiology and Functional Imaging 24: 10–18.

Muecke, S. 2005. Effects of rotating night shifts: literature review. Journal of Advanced Nursing 50: 433–439.

Murakami, S., Otsuka, K., Kubo, Y., Shinagawa, M., Yamanaka, T., Ohkawa, S. & Kitaura, Y. 2004. Repeated ambulatory monitoring reveals a Monday morning surge in blood pressure in a community-dwelling population. American Journal of Hypertension 17: 1179–1183.

Ohayon, M. M. & Partinen, M. 2002. Insomnia and global sleep dissatisfaction in Finland. Journal of Sleep Research 11: 339–346.

Oppenheim, A.V. & Schafer, R.W. 1989. Discrete-Time Signal Processing. Prentice Hall Inc., Englewood Cliffs.

Otzenberger, H., Gronfier, C., Simon, C., Charloux, A., Ehrhart, J., Piquard, F. & Brandenberger, G. 1998. Dynamic heart rate variability: a tool for exploring sympathovagal balance continuously during sleep in men. American Journal of Physiology 275: H946–950.

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

Perini, R. & Veicsteinas, A. 2003. Heart rate variability and autonomic activity at rest and during exercise in various physiological conditions. European Journal of Applied Physiology 90 (3-4): 317–325.

Perrewé, P. L. & Zellars, K. L. 1999. An examination of attributions and emotions in the transactional approach to the organizational stress process. Journal of Organizational Behavior 20: 739–752.

Petrovsky, N. & Harrison, L. C. 1997. Diurnal rhythmicity of human cytokine production – A dynamic disequilibrium in T helper cell type 1/T helper cell type 2 balance? Journal of Immunology 158: 5163–5168.

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

Pichot, V., Bourin, E., Roche, F., Garet, M., Gaspoz, J-M., Duverney, D., Antoniadis, A., Lacour, J-R. & Barthelemy, J-C. 2002. Quantification of cumulated physical fatigue at the workplace. European Journal of Physiology 445: 267–272.

Pignatelli, D., Magalhaes, M. M. & Magalhaes, M. C. 1998. Direct effects of stress on adrenocortical function. Hormone and metabolic research 30:464–474.

Pilcher, J. J. 2000. Self-report Sleep Habits as Predictors of Subjective Sleepiness. Behavioural Medicine 25: 161–168.

Pilcher, J. J., Ginter, D. R. & Sadowsky, B. 1997. Sleep quality versus sleep quantity: Relationships between sleep and measures of health, well-being and sleepiness in college students. Journal of Psychosomatic Research 42: 583–596.

Porges, S. W. 1995. Cardiac Vagal Tone: A Physiological Index of Stress. Neuroscience

and Biobehavioral Reviews 19 (2): 225–233.

Powell, L. H., Lovallo, W. R., Matthews, K. A., Meyer, P., Midgley, A. R., Baum, A., Stone, A. A., Underwood, L., McCann, J. J., Janikula Herro, K. & Ory, M. G. 2002. Physiologic markers of chronic stress in premenopausal, middle-aged women. Psychosomatic Medicine 64 (3): 502–509.

Pruessner, J. C., Wolf, O. T., Hellhammer, D. H., Buske-Kirschbaum, A., von Auer, K., Jobst, S., Kaspers, F. & Kirschbaum, C. 1997. Free cortisol levels after awakening: A reliable biological marker fort he assessment of adrenocortical activity. Life Sciences 61: 2539–2549.

Pruessner, J. C., Hellhammer, D. H. & Kirschbaum, C. 1999. Burnout, Perceived Stress, and Cortisol Responses to Awakening. Psychosomatic Medicine 61: 197–204.

Pruessner, J. C., Kirschbaum, C., Meinlschid, G. & Hellhammer, D. H. 2003a. Two formulas for computation of the area under the curve represent measures of total hormone concentration versus time-dependent change. Psychoneuroendocrinology 28: 916–931.

Pruessner, M., Hellhammer, D. H., Pruessner, J. C. & Lupien, S. J. 2003b. Self-reported depressive symptoms and stress levels in healthy young men: associations with the cortisol response to awakening. Psychosomatic Medicine 65: 92-99.

Pulkkinen, A. 2003. The accuracy of new heart rate based methods to estimate oxygen consumption. Master's thesis, University of Jyväskylä, Department of Biology of Physical Activity. (In Finnish: Uusien sykkeeseen perustuvien hapenkulutuksen arviointimenetelmien tarkkuus).

Pulkkinen, A., Kettunen, J., Martinmäki, K., Saalasti, S., & Rusko, H. K. 2004. On- and off- dynamics and respiration rate enhance the accuracy of heart rate based VO2 estimation. ACSM Congress, Indianapolis, June 2-5, 2004. Abstract: Medicine and Science in Sports and Exercise 36(5): Supplement: 253.

Putignano, P., Dubini, A., Toja, P., Invitti, C., Bonfanti, S., Redaelli, G., Zappulli, D. & Cavagnini, E. 2001. Salivary cortisol measurement in normal-weight, obese and anorexic women: comparison with plasma cortisol. European Journal of Endocrinology 145: 165–171.

Ramaekers, D., Ector, H., Aubert, A. E., Rubens, A. & Van de Werf, F. 1998. Heart rate variability and heart rate in healthy volunteers – Is the female autonomic nervous system cardioprotective? European Heart Journal 19: 1334–1341.

Rau, R. 2006. The association between blood pressure and work stress: The importance

of measuring isolated systolic hypertension. Work & Stress 20: 84–97.

Riese, H., Van Doornen, L. J., Houtman, I. L. & De Geus, E. J. 2004. Job strain in relation to ambulatory blood pressure, heart rate, and heart rate variability among female nurses. Scandinavian Journal of Work, Environment & Health 30: 477–485.

Roberts, A. D., Wessely, S., Chalder, T., Papadopoulos, A. & Cleare, A. J. 2004. Salivary cortisol response to awakening in chronic fatigue syndrome. British Journal of Psychiatry 184: 136–141.

Rook, J. W. & Zijlstra, F. R. H. 2006. The contribution of various types of activities to recovery. European Journal of Work and Organizational Psychology 15: 218–240.

Rosmond, R. 2003. Stress induced disturbances of the HPA axis: a pathway to Type 2 diabetes? Review. Medical Science Monitor: International Medical Journal of Experimental and Clinical Research 9: RA35–39.

Ross, R. M. & Jackson, A. S. 1990. Exercise concepts, calculations & computer applications. Publisher, Benchmark Press, Inc. Indiana. USA. ISBN: 0-936157-51-8.

Ruggiero, J. S. 2003. Correlates of fatigue in critical care nurses. Research in Nursing & Health 26: 434–444.

Rusko, H. K. 2006. Presentation on the course “Applied exercise physiology”. University of Jyväskylä, Department of Biology of Physical Activity.

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

Sallinen, M., Härmä, M., Kalimo, R. & Hakanen, J. 2000. The prevalence of sleep debt and its association with fatigue, performance and accidents in the modern society. People and work, Research reports 33: 140–143.

Schlotz, W., Hellhammer, J., Schulz, P. & Stone, A. A. 2004. Perceived Work Overload and Chronic Worrying Predict Weekend-Weekday Differences in the Cortisol Awakening Response. Psychosomatic Medicine 66: 207-214.

Schnall, P. L., Schwartz, J. E., Landsbergis, P. A., Warren, K. & Pickering, T. G. 1998. A longitudinal study of job strain and ambulatory blood pressure: results from a three-year follow-up. Psychosomatic Medicine 60: 697–706.

Schulz, P., Kirschbaum, C., Pruessner, J. & Hellhammer D. 1998. Increased free cortisol secretion after awakening in chronically stressed individuals due to work overload. Stress and Health 14 (2): 91–97.

Selye, H. 1946. The general adaptation syndrome and the diseases of adaptation. Journal of Clinical Endocrinology 6: 117–230.

Shapiro, P. A., Sloan, R. P., Bagiella, E., Kuhl, J. P., Anjilvel, S. & Mann, J. J. 2000. Cerebral activation, hostility and cardiovascular control during mental stress. Journal of Psychosomatic Research 48: 485–491.

Siegrist, J., Starke, D., Chandola, T., Godin, I., Marmot, M., Niedhammer, I. & Peter, R. 2004. The measurement of effort-reward imbalance at work: European comparisons. Social Science & Medicine 58: 1483–1499.

Signal, T. L., Gale, J. & Gander, P. H. 2005. Sleep Measurement in Flight Crew: Comparing Actigraphic and Subjective Estimates to Polysomnography. Aviation, Space, and Environmental Medicine 76 (11): 1058–1063.

Singh, R. B., Cornelissen, G., Weydahl, A., Schwartzkopff, O., Katinas, G., Otsuka, K., Watanabe, Y., Yano, S., Mori, H., Ichimaru, Y., Mitsutake, G., Pella, D., Fanghong, L., Zhao, Z., Rao, R. S., Gvozdjakova, A. & Halberg, F. 2003. Circadian heart rate and blood pressure variability considered for research and patient care. International Journal of Cardiology 87 (1): 9–28; discussion 29–30.

Sluiter, J. K., van der Beek, A. J. & Frings-Dresen, M. H. 1999. The influence of work characteristics on the need for recovery and experienced health: a study on coach drivers. Ergonomics 42: 573–583.

Sluiter, J. K., Frings-Dresen, M. H., van der Beek, A. J. & Meijman, T. F. 2001. The relation between work-induced neuroendocrine reactivity and recovery, subjective need for recovery, and health status. Journal of Psychosomatic Research 50: 29–37.

Sonnentag, S. & Kruel, U. 2006. Psychological detachment from work during off-job time: The role of job stressors, job involvement, and recovery-related self-efficacy. European Journal of Work and Organizational Psychology 15 (2): 197–217.

Sparks, K., Faragher, B. & Cooper, C. L. 2001. Well-being and occupational health in the 21st century workplace. Journal of Occupational & Organizational Psychology 74: 489–509.

Spiegel, K., Knutson, K., Leproult, R., Tasali, E. & Van Cauter, E. 2005. Sleep loss: a novel risk factor for insulin resistance and Type 2 diabetes. Invited review. Journal of Applied Physiology 99: 2008–2019.

Späth-Schwalbe, E., Gofferje, M., Kern, W., Born, J. & Fehm, H. L. 1991. Sleep disruption alters nocturnal ACTH and cortisol secretory patterns. Biological Psychiatry 29 (6): 575–584.

Späth-Schwalbe, E., Scholler, T., Kern, W., Fehm, H. L. & Born J. 1992. Nocturnal adrenocorticotropin and cortisol secretion depends on sleep duration and decreases in association with spontaneous awakening in the morning. Journal of Clinical Endocrinology & Metabolism 75: 1431–1435.

Stone, A. A., Schwattz, J. E., Smyth, J., Kirschbaum, C., Cohen, S., Hellhammer, D. & Grossman, S. 2001. Individual differences in the diurnal cycle of salivary free cortisol: a replication of flattened cycles for some individuals. Psychoneuroendocrinology 26 (3): 295–306.

Suzuki, K., Ohida, T., Kaneita, Y., Yokoyama, E. & Uchiyama M. 2005. Daytime sleepiness, sleep habits and occupational accidents among hospital nurses. Journal of Advanced Nursing 52: 445–453.

Taris, T. W., Beckers, D. G. J., Verhoeven, L. C., Geurts, S. A. E., Kompier, M. A. J. & van der Linden, D. 2006. Recovery opportunities, work – home interference, and well-being among managers. European Journal of Work and Organizational Psychology 15 (2): 139–157.

Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology 1996. Heart Rate Variability: Standards of Measurement, Physiological Interpretation, and Clinical Use. Circulation 93: 1043–1065.

Tennant, C. 2001. Work-related stress and depressive disorders. Journal of Psychosomatic Research 51: 697–704.

Thorn, L., Hucklebridge, F., Evans, P. & Clow, A. 2006. Suspected non-adherence and weekend versus week day differences in the awakening cortisol response. Psychoneuroendocrinology 31: 1009–1018.

Trinkoff, A. M., Lipscomb, J. A., Geiger-Brown, J., Storr, C. L & Brady, B. A. 2003. Perceived physical demands and reported musculoskeletal problems in registered nurses. American Journal of Preventive Medicine 24 (3): 270–275.

Tryon, W. W. 2004. Issues of Validity in Actigraphic Sleep Assessment. Sleep 27 (1):

158–165.

Tsuji, H., Larson, M. G., Venditti, F. J. Jr, Manders, E. S, Evans, J. C., Feldman, C. L. & Levy, D. 1996. Impact of reduced heart rate variability on risk for cardiac events. The Framingham Heart Study. Circulation 94: 2850–2855.

Tsutsumi, A., Kayaba, K., Tsutsumi, K. & Igarashi, M. 2001. Association between job strain and prevalence of hypertension: a cross sectional analysis in a Japanese working population with a wide range of occupations: the Jichi Medical School cohort study. Occupational and Environmental Medicine 58: 367–373.

Tulppo, M. P., Mäkikallio, T. H., Takala, T. E. S., Seppänen, T. & Huikuri, H.V. 1996. Quantitative beat-to-beat analysis of heart rate dynamics during exercise. American Journal of Physiology 271: H244–252.

Tworoger, S. S., Davis, S., Vitiello, M. V., Lentz, M. J. & McTiernan, A. 2005. Factors associated with objective (actigraphic) and subjective sleep quality in young adult women. Journal of Psychosomatic Research 59: 11–19.

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

Uusitalo, A. L., Uusitalo, A. J. & Rusko, H. K. 2000. Heart rate and blood pressure variability during heavy training and overtraining in the female athlete. International Journal of Sports Medicine 21: 45–53.

Valdez, P., Ramirez, C. & Garcia, A. 1996. Delaying and extending sleep during weekends: Sleep recovery or circadian effect? Chronobiology International 13 (3): 191–198.

Van Amelsvoort, L. G. P. M., Schouten, E. G., Maan, A. C., Swenne, C. A. & Kok, F. J. 2000. Occupational determinants of heart rate variability. International Archives of Occupational and Environmental Health 73: 255–262.

Van Amelsvoort, L. G. P. M., Kant, I. J., Bultmann, U. & Swaen, G. M. H. 2003. Need for recovery after work and the subsequent risk of cardiovascular disease in a working population. Occupational & Environmental Medicine 60 (Suppl I): i83-i87.

Van Reeth, O., Weibel, L., Spiegel, K., Leproult, R. Dugovic, C. & Maccari, S. 2000. Interactions between stress and sleep: from basic research to clinical situations. Sleep Medicine Reviews 4 (2): 201–219.

Vitiello, M. V., Larsen, L. H., Drolet, G., Madar, E. L. & Moe, K. E. 2002. Gender

differences in subjective–objective sleep relationships in non-complaining healthy older adults. Sleep 25S: A61.

Vitiello, M. V., Moe, K. E., Larsen, L. H. & Prinz, P. N. 1997. Age-related sleep change: relationships of objective and subjective measures of sleep in healthy older men and women. Sleep Research 26: 220.

Vrijkotte, T. G., van Doornen, L. J. & de Geus, E. J. 2000. Effects of work stress on ambulatory blood pressure, heart rate, and heart rate variability. Hypertension 35 (4): 880–886.

Vuksanovic, V. & Gal, V. 2006. Heart rate variability in mental stress aloud. Medical Engineering & Physics. Article in press.

Walsh, J. K. & Lindblom, S. S. 2000. Psychophysiology of sleep deprivation and disruption. In M. R. Pressman & W. C. Orr (Eds.), Understanding sleep: The evaluation and treatment of sleep disorders (pp. 73–110). Washington D. C.

Walsleben, J. A., Kapur, V. K., Newman, A. B., Shahar, E., Bootzin, R. R., Rosenberg, C. E., O'Connor, G. & Nieto, J. 2004. Sleep and Reported Daytime Sleepiness in Normal Subjects: the Sleep Heart Health Study. Sleep 27: 293–298.

Wennerblom, B., Lurje, L., Karlsson, T., Tygesen, H., Vahisalo, R. & Hjalmarson, Å. 2001. Circadian variation of heart rate variability and the rate of autonomic change in the morning hours in healthy subjects and angina patients. International Journal of Cardiology 79: 61–69.

Wetherell, M. A., Crown, A. L., Lightman, S. L., Miles, J. N. V., Kaye, J. & Vedhara, K. 2006. The four-dimensional stress test: Psychological, sympathetic–adrenal–medullary, parasympathetic and hypothalamic–pituitary–adrenal responses following inhalation of 35% CO_2. Psychoneuroendocrinology 31: 736–747.

Williams, E., Magid, K. & Steptoe, A. 2005. The impact of time of waking and concurrent subjective stress on the cortisol response to awakening. Psychoneuroendocrinology 30 (2): 139–148.

Wolk, R., Gami, A. S., Garcia-Touchard, A. & Somers, V. K. 2005. Sleep and Cardiovascular Disease. Review. Current Problems in Cardiology 30 (12): 625–662.

Wüst, S., Wolf, J., Hellhammer, D. H., Federenko, I., Schommer, N. & Kirschbaum, C. 2000. The cortisol awakening response – normal values and confounds. Noise & Health 7: 77–85.

Zarkovic, M. Stefanova, E., Ciric, J., Penezic, Z., Kostic, V., Sumarac-Dumanovic, M., Macut, D., Ivovic, M. S., & Gligorovic, P. V. 2003. Prolonged psychological stress suppresses cortisol secretion. Clinical Endocrinology 59: 811–816.

Zijlstra, F. R. H. & Sonnentag, S. 2006. After work is done: Psychological perspectives on recovery from work. European Journal of Work and Organizational Psychology 15: 129–138.

Åkerstedt, T., Kecklund, G., Gillberg, M., Lowden, A. & Axelsson, J. 2000. Sleepiness and days of recovery. Transportation Research Part F 3, 251–261.

Åkerstedt, T., Knutsson, A., Westerholm, P., Theorell, T., Alfredsson, L. & Kecklund, G. 2002. Sleep disturbances, work stress and work hours - A cross-sectional study. Journal of Psychosomatic Research 53 (3): 741–748.

Åkerstedt, T., Nilsson, P. M. 2003. Sleep as restitution: an introduction. Journal of Internal Medicine 254: 6–12.

APPENDIXES

Appendix 1. Written information about the study

TYÖN KUORMITUSTEKIJÖIDEN JA TYÖNTEKIJÄN KUORMITTUNEISUUDEN OBJEKTIIVISEN MITTAUSMENETELMÄN KEHITTÄMINEN JA KÄYTTÖÖNOTTO
- sairaalainterventio

Tutkimuksen lähtökohta

Työikäisten uupuminen työssään, liikalihavuuden mukanaan tuomat ongelmat ja sairaudet sekä fyysisen kunnon yleinen heikkeneminen ovat romuttamassa ihmisten hyvinvointia ja elämänhallintaa. Ongelmien taustalla ovat ihmisten elämäntavoissa tapahtuneet muutokset: työelämä on muuttunut kiireisemmäksi ja psyykkisesti kuormittavammaksi samalla, kun elvyttävän ja palauttavan vapaa-ajan sekä liikunnan määrä on yleisesti vähentynyt. Tunnistetuista ongelmista huolimatta käytännön toimijoilta puuttuvat objektiiviset menetelmät, joilla he voisivat seurata sekä antaa palautetta työn ja vapaa-ajan terveysvaikutuksista. Käytännön toimijat tarvitsevat helppokäyttöisiä työkaluja, jotka soveltuvat sekä yleisen tason ryhmäseulontoihin, erilaisten tilanteiden vaikutusten tutkimiseen ja yksilötason seurantaan.

Käynnistyvä projekti on monen tahon yhteistyöyritys luoda yllämainittuja työkaluja esim. työterveyshuoltoon kuormituksen objektiiviseksi mittaamiseksi. Projektista päävastuussa on Jyväskylän yliopiston liikuntabiologian laitos ja muina yhteistyötahoina ovat Jyväskylän yliopiston psykologian laitos, Firstbeat Technologies Oy, Suunto Oy, Kilpa- ja huippu-urheilun tutkimuskeskus, Kuntoutuskeskus Peurunka ja Jyväskylän Seudun Työterveys.

Tutkimuksen tavoitteena on saada työntekijöiden kuormittuneisuuden ja voimavarojen palautumisen kartoittamiseen käyttömalleja, joita voidaan soveltaa käytäntöön muun muassa työterveyshuollossa sekä hyvinvointipalveluja tarjoavissa yrityksissä seuraavissa tilanteissa:

- työssään ylikuormittuneiden työntekijöiden seulonta
- työn sisältöön ja työyhteisöihin kohdistuvien muutostilanteiden seuranta
- yksilöllisen kuormittumisen ja palautumisen tarkka seuranta ja hallinta
- motivointi ja opettaminen/oppiminen oman kuormittuneisuuden ja palautumisen itseseuranta
- syke- ja liikuntamittausten yhdistäminen erilaisissa kuormittumista ja palautumista sisältävissä tilanteissa, kuten kuntoilu tai erilaiset työsuoritukset

Tutkimuksen käytännön toteutus

Käytännössä tutkimukseen osallistuminen johtaa pieneen lisähäiriöön työpäivän aikana. Tutkimuksia tehdään kahden työpäivän aikana, jotka valitaan tutkittavan toiveen mukaan työjakson alusta ja lopusta (esim. viikon alku- ja loppupäivä), ja yhden vapaapäivän aikana. Tutkittava kantaa kyseisinä kolmena vuorokautena (24 h) mukanaan Suunnon jatkuvasti sykettä ja sen vaihtelua mittaavaa mittaria sekä liikettä mittaavaa 'kelloa'. Molemmat laitteet ovat hyvin kevyitä, eivätkä aiheuta fyysistä lisärasitetta tutkittavalle. Tämän lisäksi tutkittava joutuu vastaamaan muutamiin tuntemuksia kuvaaviin kysymyksiin työpäivän aikana tiettyinä ajankohtina. Myös tutkittavan työtä tarkkaillaan työpäivän aikana kahtena etukäteen määrättynä lyhyenä ajankohtana. Tutkittava pitää myös päiväkirjaa päivänsä kulusta ja täyttää illalla päiväkohtaista kuormittuneisuutta kuvaavan kyselyn.

Tutkimuspäivien jälkeen tutkittava täyttää 2 kyselylomaketta, joissa tutkittavaa pyydetään arvioimaan työstressiään ja 3 kyselylomaketta, jossa tutkittava arvioi perusominaisuuksiaan (persoonallisuus, masentuneisuus, vuorovaikutustaidot) yhdessä haastattelijan kanssa. Tutkittavalle tehdään maksimaalinen fyysistä suorituskykyä kuvaava testi tarvittaessa lääkärin valvonnassa, sekä lyhyt lääkärin tarkastus ja haastattelu. Tutkittavalta otetaan sylkinäytteitä stressihormonitasojen tutkimiseksi erikseen määrättyinä ajankohtina tutkimuspäivien aikana.

Tutkittavan oikeudet ja tutkijoiden velvollisuudet

Tutkittavalla on oikeus kieltäytyä mistä tahansa tutkimuksen osa-alueesta tai keskeyttää tutkimus milloin hän haluaa ilman erillisiä perusteluja.

Tutkijat ovat velvollisia ilmoittamaan tutkittaville heitä koskevat mittauksissa esille tulleet toimenpiteitä vaativat tulokset. Lisäksi tutkittavien on mahdollisuus saada henkilökohtaista palautetta itsestään, kuormitustilanteestaan ja voimavarojen palautumisestaan. Tällaista tietoa voi hyödyntää itseseurannassa ja se voi tarjota myös lähtökohtia työn kehittämiseen.

Tutkimustietoja käsitellään luottamuksellisesti ja nimettöminä. Tutkijat ovat luonnollisesti vaitiolovelvollisia yksilötulosten suhteen.

Jos teillä on kysyttävää, saatte lisätietoja seuraavilta henkilöiltä:

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Appendix 2. Written informed consent

TYÖN KUORMITUSTEKIJÖIDEN JA TYÖNTEKIJÄN KUORMITTUNEISUUDEN OBJEKTIIVISEN MITTAUSMENETELMÄN KEHITTÄMINEN JA KÄYTTÖÖNOTTO

Olen saanut tietoa
- tutkimuksen tarkoituksesta
- tutkimuksen kulusta
- tutkittavaan kohdistuvista toimenpiteistä

Olen tietoinen
- tutkittavan oikeuksista ja velvollisuuksista

Suostun
- maksimaaliseen polkupyöräergometriatestiin
- 24-tunnin sykeseurantaan ja liikeanturien pitoon
- haastatteluihin ja kyselykaavakkeiden täyttöön
- työpäivän aikana tapahtuvaan lyhyeen seurantaan ja mielialakysymyksiin
- syljen kortisolinäytteiden ottoon

Osallistun yllä mainittuun tutkimukseen tutkittavana vapaaehtoisesti.

Nimi (tekstaten selkeästi)_____

Osoite
_____

Puhelinnumero
_____

Sähköpostiosoite
_____

Aika ja paikka
_____

Allekirjoitus
_____

Appendix 3. Study diary.

SYKETUTKIMUKSEN AIKAISTEN TOIMINTOJEN KIRJAAMINEN

Täytä päiväkirjaan sykemittauksen aikaisesta toiminnastasi sykkeeseen vaikuttava <u>toiminta</u> sekä sen <u>alku- ja loppukellonaika,</u> mielellään minuutin tarkkuudella.

Seuraavassa listassa on lueteltu tärkeimpiä syketasoon vaikuttavia toimintoja:

- työvuoron alku ja loppu
- tauot työvuoron aikana
- työtehtävän kuormittavuuden vaihtuminen
- voimakkaat kielteiset tunnetilat
- äkilliset yllättävät tapahtumat
- nukkumaan meno ja herääminen
- liikkuminen autolla, pyörällä, jalan
- makuulla, istuallaan tai seisaallaan olo pitemmän aikaa
- saunominen, suihku
- ruokailu, kahvin, teen, tupakan ja alkoholinkäyttö
- liikunta
- rentoutumishetken alku ja loppuaika

TÄYTÄ NÄMÄ TIEDOT:
Syntymäaika: _____
Pituus: _____
Paino: _____
Tupakoitko? kyllä en
Aktiivisuusluokka: _____

Nukuin ensimmäisen yön:
hyvin kohtalaisesti huonosti

Nukuin toisen yön:
hyvin kohtalaisesti huonosti

Nimi: _____Päivämäärä: _____ Otos:
Kh: _____ Otin mittarin pois seuraavana aamuna klo:_____

Alkoi	**Loppui**	**Tekeminen**

Appendix 4. Instructions for the measurement day.

Jyväskylän yliopiston SYTY-projekti/ KSSHP
Hei _____

Tässä sinulle ohjeet ensimmäiseen mittauspäivään, joka sisältää monenlaisia osioita. Mittaukset alkavat siitä, kun sinulle on nyt asennettu kolme mittaria (rrRecorder ja liikemittarit) tallentamaan muistiin kehosi fysiologisia reaktioita ja liikkeidesi määrää. Tämän ohjeen viimeisellä sivulla on lisää tietoa mittareista kohdassa ”sykkeen vuorokausimittaus”, lue ne tässä yhteydessä jo läpi.

Tänä ensimmäisenä iltana sinun ei tarvitse tehdä muuta, kuin totutella mittareiden olemassa oloon ja tehdä hieman valmisteluja huomista varten. Huomiseen päivään sinulla kuuluu näiden mittareiden pitämisen lisäksi sylkinäytteiden ottaminen itseltäsi, päiväkirjan kirjoittaminen, työvuoron jälkeen sinisen lomakenipun täyttäminen ja itsenäinen rentoutushetki.

Tähän ohjeeseen olemme aikajärjestyksessä koonneet huomisen päiväsi kulun, jotta sinun olisi helpompi hahmottaa millaisia osioita missäkin vaiheessa päivää toivoisimme sinun toteuttavan.

Lue jo tänä iltana ohjeita läpi, niin huomisen kulku hahmottuu sinulle paremmin.

Mikäli sinulla ilmenee mittausten aikana jotain hankaluuksia, voit ottaa meihin yhteyttä

Yhteistyöterveisin, Terhi Rönkä puh. xxx-xxxxxxx
Henna Hämäläinen puh. xxx-xxxxxxx
Tero Myllymäki puh. xxx-xxxxxxx
Jaana Mähönen puh. xxx-xxxxxxx

ILLALLA TEHTÄVIÄ VALMISTELUJA

Valmistelut koskevat sylkinäytteiden ottamista. Huomisen aikana tulet ottamaan itseltäsi kuusi sylkinäytettä. Näytteiden ottamiselle on määritelty tarkat ajankohdat, joista sinun tulisi pitää kiinni. Ajankohdat ovat seuraavat:

1) välittömästi herättyäsi
2) puoli tuntia heräämisestäsi
3) tunti heräämisestäsi
4) kolme tuntia heräämisestäsi
5) illan rentoutumishetken jälkeen
6) nukkumaan mennessäsi

Ajankohtia vastaavat numerot on näyteputkissa. Tästä numerojärjestyksestä sinun on tärkeä pitää kiinni. Tutustu jo nyt illalla alla oleviin sylkinäytteen otto ohjeisiin ja valmistele aamun ensimmäisen näytteen otto varaamalla putki 1 sänkysi viereen.

SYLKINÄYTTEEN OTTO-OHJE

1. Avaa putken päässä oleva korkki ja ota putken yläosassa olevan hylsyn sisältä vanutuppo suuhusi.

2. Pidä vanua suussasi noin minuutin ajan, jolloin sylki kastelee tupon. Voit pureskella ja pyöritellä sitä suussasi, jolloin syljen eritys kiihtyy.

3. Kun tuppo on märkä, sylje se takaisin hylsyyn ja aseta korkki paikoilleen.

HUOM! Pyri olemaan syömättä ja juomatta 15 min ennen näytteen otto ja huuhtele suu muutaman kerran tavallisella vedellä heti syönnin jälkeen, jotta suusta vanutuppoon tuleva neste on sylkeä.

HUOM! Muista merkitä kellonaika putkessa olevaan tarraan.

HUOMIO! Kun otat yhden putkista käteesi, huomaat, että siihen on teipattu paperiluiska, jossa kolme tunnetilaa. Aina kun huomenna on sylkinäytteenottohetki, rastita juuri sitä ennen kultakin janalta kohta, joka vastaa juuri sen hetkistä tunnetilaasi.

Suuri kiitos sinulle vaivannäöstäsi jo tässä vaiheessa.

MITTAUSPÄIVÄN SISÄLTÖ

VÄLITTÖMÄSTI HERÄTTYÄSI
Paina tallentimessa olevaa nappia (jota eilen painoit yhdessä tutkijan kanssa).
Ota sylkinäyteputki numero 1 yöpöydältäsi. Rastita putkeen teipatulta janalta kohta, joka kuvaa juuri tämän hetkistä tunnetilaasi. Rastituksen jälkeen ota sylkinäyte ohjeen mukaisesti, joka edellisellä sivulla. Muista merkitä putkeen tarkka kellonaika juuri tällä hetkellä.
HUOM! On erittäin tärkeää, että otat näytteen jo heti sängyssä, etkä ole ennen näytteen ottoa esim. juonut tai syönyt mitään tai muutoinkaan ollut valveilla. Otettuasi näytteen laita putki takaisin pussiin.

KOKO PÄIVÄN AJAN
Täytä päiväkirjaa tekemisistäsi niin tarkkaan kuin sinun on mahdollista.

30 MINUUTTIA HERÄÄMISESTÄSI
Ota pussista putki 2. Täytä putkeen teipattu lappu ja ota sylkinäyte aikaisemman tavoin.

60 MINUUTTIA HERÄÄMISESTÄSI
Ota pussista putki 3 ja toimi aikaisemman kaltaisesti. Muista merkitä jälleen putkeen tarkka kellonaika ja rastittaa tämän hetkiset tunteesi.

3 TUNTIA HERÄÄMISESTÄSI
Ota pussista putki 4 ja toimi aikaisemman kaltaisesti. Mikäli olet aamuvuorossa, tämän näytteen ottoaika sijoittuu työpäivääsi.

ILLALLA TYÖPÄIVÄSI JÄLKEEN

RENTOUTUSHETKI
Illan aikana (tai aamupäivällä, mikäli työvuorosi alkaa puolenpäivän jälkeen) sinun tulisi löytää 15 minuutin hetki, jolloin pystyt rentoutumaan. Istu rentoutuksen ajan mukavasti ja pyri välttämään puhumista ja fyysistä aktiivisuutta. Ota tämä hetki itsellesi rauhoittumiseen ja rentoutumiseen. Aloittaessasi rentoutumista paina tallentimessa olevaa nappia, kuten teit herättyäsikin. **HUOM!** Välittömästi rentoutumisen jälkeen ota pussista putki 5 ja toimi samoin kuin aikaisempienkin näytteiden kanssa.

SINIKANTINEN LOMAKENIPPU
Täytä lomakkeet sinulle parhaiten sopivana ajankohtana työpäiväsi jälkeen. Täytettyäsi laita lomakenippu takaisin pussiin.

NUKKUMAAN MENNESSÄSI
Ota pussista putki 6 ja toimi samoin kuin päivän muidenkin näytteiden oton kanssa. Merkitse nukkumaan menoaikasi myös päiväkirjaan. Nuku hyvin ja seuraavana aamuna voit sitten ottaa mittarit pois.

Sykkeen vuorokausimittaus

Polar RR-recorder on sydämen sykettä mittaava laite, johon kuuluu rinnan ympärille asennettava panta sekä siihen johdoilla kytketty tallennin. Laite tallentaa jokaisen sydämen lyöntisi, ja näet punaisen valon vilkkuvan laitteen kyljessä sydämenlyöntiesi tahdissa. Laitteen tulee olla ylläsi n. puolitoista vuorokautta siitä lähtien kun tutkija asettaa sen käyntiin. Laite ei todennäköisesti häiritse lainkaan normaaleja päivittäisiä toimintojasi tai nukkumistasi. Sinun tulee pitää laitetta ylläsi koko sovitun mittausjakson ajan **lukuun ottamatta suihkussa / saunassa käyntiä.** Laite ei ole vesitiivis, joten ota se pois ennen suihkuun menoa niin, että irrotat vain pannan ympäriltäsi. Takaisin laittaessasi sinun ei tarvitse tehdä muuta kuin huolehtia, että panta on oikein päin ylläsi (johdot lähtevät alaspäin) ja oikeassa kohdassa rintakehää. Mikäli lopetat mittauksen ennen kuin annat mittarin tutkijalle, sinun ei tarvitse tehdä muuta kuin irrottaa panta ympäriltäsi.

HUOM!

Paina kerran tallentimessa olevaa nappia (vihreä valo alkaa vilkkua):

- heti herättyäsi (= ennen kuin nouset sängystä ylös)
- aloittaessasi rentoutuksen illalla

Liikemittaus

Ranteeseesi ja nilkkaasi asennetut laitteet mittaavat liikettä. Pidä niitä yhtä kauan kuin RR-recorderiakin. Nämä mittarit eivät ole vesitiiviitä, joten ota myös ne pois suihkuun / saunaan mennessäsi. Mikäli mittareista kuuluu piippaava ääni, älä välitä siitä, se loppuu itsestään (n. 20 sek. kuluttua).

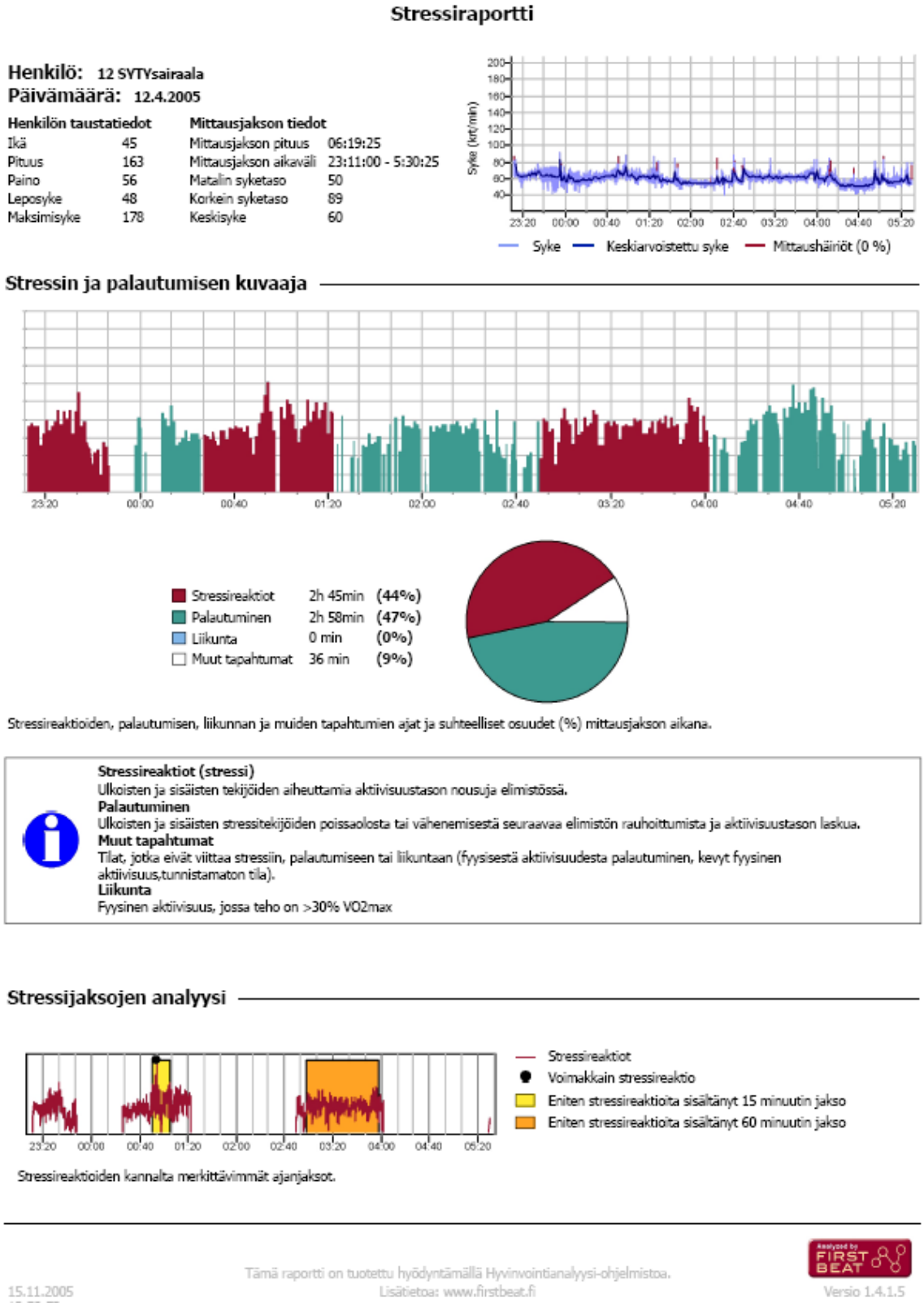
Appendix 5. Content of Data export –function of Wellness Analysis Software (www.firstbeat.fi).

PERSON PROFILE		
Identifier	Unit	Explanation
Person name		Name
Date of birth	dd.mm.yyyy	Date of birth
Gender		Gender
Smoking	TRUE|FALSE	Does the person smoke more than 10 cigarettes per day?
Length	cm	Height
Weight	kg	Weight
Activity class	(0 -> 10)	Activity class 0-7 according to Jackson et al. (1990), 7.5, 8, 8.5, 9, 9.5 ja 10 according to Firstbeat. Activity classes 7,5 – 10 are not in use at Firstbeat Pro version 1.3.2 or earlier.
Heart beat max	times/min	Maximal heart rate
Heart beat min	times/min	Resting heart rate
MET max	MET	Maximal performance METs (MET max = VO_{2max} (ml/kg/min) / 3,5 ml/kg/min)
Respiration rate max	times/min	Maximal respiration rate at maximal exercise
Ventilation max	l/min	Maximal ventilation at maximal exercise
Vital capacity	l	Lung volume
SCALAR VARIABLES		
Identifier	Unit	Explanation
Description	text	Value of the Description field in Firstbeat Pro software.
SessionStartDate	dd.mm.yyyy	Session start date
SessionStartTime	hh:min:sec	Session start time
SessionTotalTime	min	Session total time
RelaxationTime	min	Total time of relaxation
StressTime	min	Total time of stress
TimeBelow20pMETMax	min	Total time in minutes of periods where intensity was less than 20% of METmax.
Time20pTo30pMETMax	min	Total time in minutes of periods where intensity was between 20% and 30% of METmax.
TimeOver30pMETMax	min	Total time in minutes of periods where intensity was over 30% of METmax.
Time4MinPeriods30pTo40pMETMax	min	Total time in minutes of over 4 minute periods where intensity was between 30% and 40% of METmax.
Time4MinPeriodsOver40pMETMax	min	Total time in minutes of over 4 minute periods where intensity was over 40% of METmax.
AverageHR	times/min	Session average heart rate
AverageRespiration	times/min	Session average respiration rate
AverageVentilation	l/min	Session average ventilation
AverageVO2	ml/kg/min	Session average oxygen consumption
AveragePercentageVO2	%	Session average relative oxygen consumption
MaxHR	times/min	Session highest 10 second average heart rate
MaxRespiration	times/min	Session peak respiration rate
MaxVentilation	l/min	Session peak ventilation
MaxVO2	ml/kg/min	Session peak oxygen consumption
MaxPercentageVO2	%	Session peak relative oxygen consumption

MinHR	times/min	Session lowest 10 second average heart rate
EETotal	kcal	Session total energy expenditure
EnergyConsumedCH	kcal	Session total energy expenditure from carbon hydrates
EnergyConsumedFat	kcal	Session total energy expenditure from fat
EEPredictRest	kcal	An estimate of energy expenditure for 24 hours based on measurement. For the unmeasured period there is assumed that energy expenditure is in resting level.
EEBelow20pMETMax	kcal	Total energy expenditure in periods where intensity was less than 20% of METmax.
EE20pTo30pMETMax	kcal	Total energy expenditure in periods where intensity was between 20% and 30% of METmax.
EEOver30pMETMax	kcal	Total energy expenditure in periods where intensity was over 30% of METmax.
TrainingEffect	(1.0 -> 5.0)	Training Effect revealing the effect of session exercise on performance level
EpocPeak	ml/kg	Session peak EPOC value
FitnessIndex	(0 -> 99)	Fitness index revealing whether session physical activity had fitness enhancing effect
HealthIndex	(0 -> 99)	Health index revealing whether session physical activity had an health enhancing effects
AbsoluteStressIndex	Index	Absolute index of stress as the average absolute stress level of the session during periods when stress state is detected
AbsoluteRelaxationIndex	Index	Absolute index of relaxation as the average absolute relaxation level of the session during periods when stress state is detected
RelaxationPercentage	%	Proportion of relaxation from session total time
StressPercentage	%	Proportion of stress from session total time
StressRelaxationBalance	(-1.00 -> 1.00)	Index revealing the balance between stress and relaxation. Negative values indicate that the balance is towards stress and positive values positive values that the balance is towards relaxation
BeatByBeatRMSSD	ms	Heart rate variability variable RMSSD ("The square root of the mean of the sum of the squares of differences between adjacent RR intervals") describing the variation in consecutive rr-intervals.
BeatByBeatSD	ms	Heart rate variability variable, standard deviation of successive heart beat intervals of the whole session
HFAverage	ms^2	Mean of the high frequency (0.15-0.4 HZ) heart rate variability power during the session
LFAverage	ms^2	Mean of the low frequency (0.04-0.15 HZ) heart rate variability power during the session
HF2Average	ms^2	Mean of the high frequency (0.15-1.0 HZ) heart rate variability power during the session
LFHFRatio	Ratio	Mean ratio between the low frequency (0.04-0.15 HZ) and the high frequency (0.15-0.4 HZ) heart rate variability power (LF/HF) during the session
LFHF2Ratio	Ratio	Mean ratio between the low frequency (0.04-0.15 HZ) the high frequency (0.15-1.0 HZ) heart rate variability power (LF/HF2) during the session
VECTORS		
Identifier	Unit	Explanation
CumulativeSecondVector	hh:min:sec	Cumulative time starting from the beginning of the session

RealTimeVector	hh:min:sec	Real time
StateVector	(1 -> 10)	Statevector indicating which Firstbeat determined state is currently on at each respective moment. State indexes: 1) physical exercise >95%VO_{2max}, 2) 95-75%VO_{2max}, 3) 75-50%VO_{2max}, 4) 50-30%VO_{2max}, 5) light physical activity, 6) relaxation, 7) recovery from physical exercise, 9) stress, 10) unrecognized state.
ArtifactCorrectedHRVector	beats/min	Artefact corrected heart rate vector
pVO2MaxVector	%	Proprotional intensity vector: oxygen consumption vector scaled according to maximal oxygen consumption
VO2Vector	ml/kg/min	Oxygen consumption vector
EPOCVector	ml/kg	EPOC vector
RespRVector	breaths/min	Respiration rate vector
VentilationVector	liters/min	Ventilation vector
EEVector	kcal/min	Total Energy expenditure vector
EEpFatVector	kcal/min	Energy expenditure from fats vector
ResourceVector	Index	Resources vector revealing the accumulation or reduction of body resources during the session
AbsoluteStressVector	Index	Absolute vector of stress, revealing the momentary absolute level of stress during the session
AbsoluteRelaxationVector	Index	Absolute vector of relaxation, revealing the momentary absolute level of relaxation during the session
ScaledStressVector	(0.00 -> 1.00)	Relative vector of stress, revealing the momentary proportional level of stress proportioned by the maximal stress value during the session
ScaledRelaxationVector	(0.00 -> 1.00)	Relative vector of relaxation, revealing the momentary proportional level of relaxation proportioned by the maximal relaxation value during the session
VLFVector	ms^2	Vector of very low frequency (0.03-0.04 Hz) heart rate variability power, determining the momentary level of heart rate variability at this spectral band during the session
LFVector	ms^2	Vector of low frequency (0.04-0.15 Hz) heart rate variability, determining the momentary level of heart rate variability at this spectral band during the session
HFVector	ms^2	Vector of high frequency (0.15-0.4 Hz) heart rate variability power, determining the momentary level of heart rate variability at this spectral band during the session
HF2Vector	ms^2	Vector of very low frequency (0.15-1.0 Hz) heart rate variability power, determining the momentary level of heart rate variability at this spectral band during the session
RSAAmplitudeVector	ms	Vector of RSA (respiratory sinus arrhythmia) amplitude, which is determined as the mean heart rate variability power at the heart beat derived respiration frequency (+0.01- -0.01). This reveals the effect of respiration on heart rate variability power.

Appendix 6. An example of a stress report from the night-sleep period.



Stressiraportti

Henkilö: 12 SYTYsairaala
Päivämäärä: 12.4.2005

Henkilön taustatiedot		Mittausjakson tiedot	
Ikä	45	Mittausjakson pituus	06:19:25
Pituus	163	Mittausjakson aikaväli	23:11:00 - 5:30:25
Paino	56	Matalin syketaso	50
Leposyke	48	Korkein syketaso	89
Maksimisyke	178	Keskisyke	60

Stressin ja palautumisen kuvaaja

Stressireaktiot	2h 45min	(44%)
Palautuminen	2h 58min	(47%)
Liikunta	0 min	(0%)
Muut tapahtumat	36 min	(9%)

Stressireaktioiden, palautumisen, liikunnan ja muiden tapahtumien ajat ja suhteelliset osuudet (%) mittausjakson aikana.

Stressireaktiot (stressi)
Ulkoisten ja sisäisten tekijöiden aiheuttamia aktiivisuustason nousuja elimistössä.
Palautuminen
Ulkoisten ja sisäisten stressitekijöiden poissaolosta tai vähenemisestä seuraavaa elimistön rauhoittumista ja aktiivisuustason laskua.
Muut tapahtumat
Tilat, jotka eivät viittaa stressiin, palautumiseen tai liikuntaan (fyysisestä aktiivisuudesta palautuminen, kevyt fyysinen aktiivisuus,tunnistamaton tila).
Liikunta
Fyysinen aktiivisuus, jossa teho on >30% VO2max

Stressijaksojen analyysi

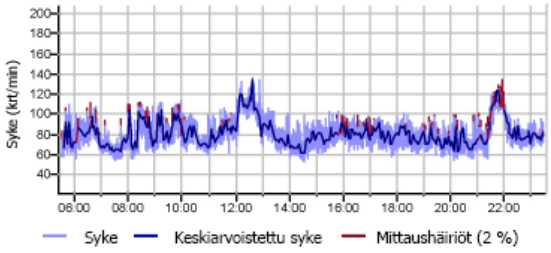
Stressireaktioiden kannalta merkittävimmät ajanjaksot.

Appendix 7. An example of a stress report from awake-time.

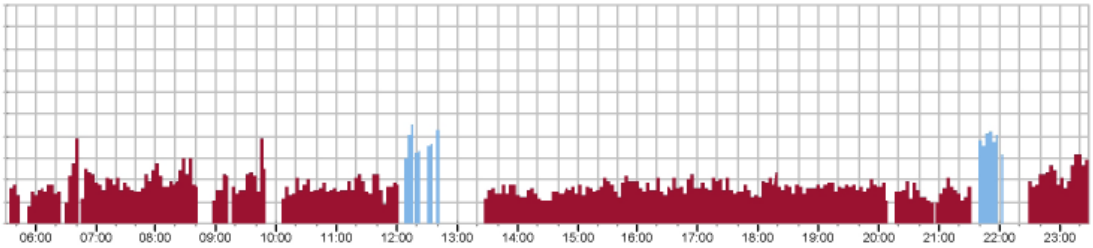
Stressiraportti

Henkilö: 12 SYTYsairaala
Päivämäärä: 13.4.2005

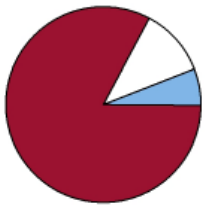
Henkilön taustatiedot		Mittausjakson tiedot	
Ikä	45	Mittausjakson pituus	17:58:39
Pituus	163	Mittausjakson aikaväli	5:30:25 - 23:29:04
Paino	56	Matalin syketaso	59
Leposyke	48	Korkein syketaso	136
Maksimisyke	178	Keskisyke	81



Stressin ja palautumisen kuvaaja



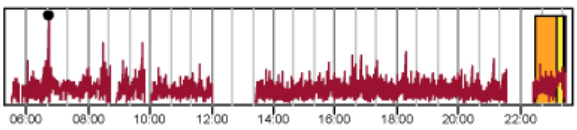
Stressireaktiot	14h 52min	**(83%)**
Palautuminen	0 min	**(0%)**
Liikunta	1h 5min	**(6%)**
Muut tapahtumat	2h 2min	**(11%)**



Stressireaktioiden, palautumisen, liikunnan ja muiden tapahtumien ajat ja suhteelliset osuudet (%) mittausjakson aikana.

Stressireaktiot (stressi)
Ulkoisten ja sisäisten tekijöiden aiheuttamia aktiivisuustason nousuja elimistössä.
Palautuminen
Ulkoisten ja sisäisten stressitekijöiden poissaolosta tai vähenemisestä seuraavaa elimistön rauhoittumista ja aktiivisuustason laskua.
Muut tapahtumat
Tilat, jotka eivät viittaa stressiin, palautumiseen tai liikuntaan (fyysisestä aktiivisuudesta palautuminen, kevyt fyysinen aktiivisuus,tunnistamaton tila).
Liikunta
Fyysinen aktiivisuus, jossa teho on >30% VO2max

Stressijaksojen analyysi



Stressireaktiot
Voimakkain stressireaktio
Eniten stressireaktioita sisältänyt 15 minuutin jakso
Eniten stressireaktioita sisältänyt 60 minuutin jakso

Stressireaktioiden kannalta merkittävimmät ajanjaksot.