NOCTURNAL HEART RATE VARIABILITY RESPONSES TO ENDURANCE TRAINING IN GOOD-TO-ELITE LEVEL ENDURANCE ATHLETES

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ABSTRACT

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The goal of endurance training is to enhance endurance capacity. The enhancements in this capacity are based on the adaptations in the oxygen delivery system and neuromuscular system. To achieve these adaptations an athlete must train to disturb homeostasis and have adequate rest between trainings to recover. The balance between stress and recovery is vital to avoid overstress symptoms with maladapted sports performance. Sport scientists are constantly developing new solutions to follow this balance. So far one of the most promising solutions is heart rate variability-based stress-recovery-follow up. Several different measurement technologies have been developed using either electrical activity-based or pulse-based heart rate variability (HRV). The application of these technologies to real-world settings as well as the interpretation of the HRV data to adjust athlete´s training are still challenges when utilizing HRV in elite sports. Some studies have been done with elite athletes, but most of the studies focusing on the effects on training have been done with either recreational of non-elite level athletes. The value of these studies in elite sports remain questionable and thus it is important to actively seek for the practical and reliable measurement technologies and appropriate HRV analyzes suitable for elite athletes.

32 endurance athletes (29 orienteers, 3 triathletes), 8 women (age 22,1 ± 3,2 yr, VO_2max 52,5 ± 3,6 ml/kg/min) and 24 men (age 22,2 ± 5,2 yr, VO_2max 65,2 ± 5,0 ml/kg/min) participated the study. 24 of these athletes were included in the final analysis. Training was not standardized over the measurement period. Athletes were asked to report their endurance training and subjective ratings of stress and recovery. Endurance training was reported in 3 categories: 1= low-intensity >90min, 2= moderate-to-high intensity <45min, 3= moderate-to-high intensity >45min. Nocturnal HRV was collected during the 6 months measurement period with EMFIT Qs sleep tracker. Collected HRV measures were analyzed in relation to: 1) subjective ratings of recovery and 2) reported endurance trainings. Additionally, long-term HRV trends were analyzed with monthly average values.

HRV parameters and subjective ratings of recovery had low, yet significant correlations (LFnu r= 0.85; p<0.001 and RMSSD 90min avg r=0.33; p= 0.41) on group level. On the individual level, subjectively reported recovery correlated with at least one HRV parameter in 17/ of 19 athletes, but the results varied a lot depending on the individual. When compared to training type 1, training types 2 and 3 led to significantly different nocturnal HRV levels when analyzed on group level. On the individual level, only few athletes had significantly altered HRV levels after the followed trainings and the HRV trends were inconsistent. Long-term analysis revealed significant decrease in HRV on monthly basis.

Subjective and objective measures of recovery were shown to correlate weakly with each other. Endurance training intensity seems to play greater role in determining acute nocturnal HRV response to training, compared to training duration. Due to limitations in data, these results are not conclusive, but give important sights to recovery monitoring in good-to-elite level endurance athletes. The decrease of the HRV levels over the measurement period question the training and recovery balance of these athletes when approaching to the competitive season and could be a potential interest for further studies.

Keywords: Heart rate variability, athlete, endurance training, intensity, duration, recovery, subjective feeling.

TIIVISTELMÄ

Koskinen, R. (2019). Nocturnal heart rate variability responses to different endurance trainings in good-to-elite level endurance athletes. Liikuntatieteellinen tiedekunta, Jyväskylän yliopisto. Liikuntafysiologian Pro Gradu-tutkielma. 69 s. 1 liite.

Kestävyysharjoittelun tavoite on kehittää kestävyyssuorituskykyä. Tämä perustuu hapenkuljetus- ja hermo-lihasjärjestelmän adaptaatioihin, jotka syntyvät urheilijan harjoittelun järkyttäessä elimistön homeostaasia ja palautumisen harjoitusten välissä ollessa riittävää. Tasapaino kuormittumisen ja palautumisen välillä on elintärkeä pyrittäessä välttämään ylikuormitustiloja ja niistä johtuvaa suorituskyvyn alenemista. Tutkijat kehittelevät jatkuvasti uusia sovellutuksia tämän tasapainotilan seurantaan ja tähän mennessä yksi lupaavimmista menetelmistä on sykevälivaihteluun perustuva seuranta. Lukuisia sydämen elektroniseen aktiivisuuteen ja verenkierron pulssiaaltoihin perustuvia mittareita on kehitelty viime vuosina, mutta tämän teknologian soveltaminen urheilijan päivittäiseen elämään ja sykevälivaihteludatan tulkinta harjoituskuorman optimoimiseksi on edelleen haastavaa huippu-urheilijoiden kanssa toimittaessa. Muutamissa tutkimuksissa on tutkittu huipputason urheilijoita, mutta suurin osa alan tutkimuksista on tehty joko kuntoilijoilla tai alemman tason kilpaurheilijoilla. Näiden tutkimusten sovellettavuus huippu-urheiluun on kyseenalaista ja siksi onkin erittäin tärkeää etsiä aktiivisesti käytännöllisiä ja luotettavia mittareita ja huippu-urheiluun sopivia analyysityökaluja.

32 kestävyysurheilijaa (29 suunnistajaa, 3 triathlonistia) 8 naista (ikä 22,1 ± 3,2 vuotta, VO_2max 52,5 ± 3,6 ml/kg/min) ja 24 miestä (ikä 22,2 ± 5,2 vuotta, VO_2max 65,2 ± 5,0 ml/kg/min) osallistuivat tutkimukseen ja 24 näistä urheilijoista oli mukana lopullisissa analyyseissa. Harjoittelua ei vakioitu mittausjaksolla, vaan urheilijoita pyydettiin raportoimaan kestävyysharjoitteluaan ja subjektiivisesti arvioitua palautuneisuuttaan. Kestävyysharjoittelu raportoitiin kolmessa kategoriassa: 1= matalatehoinen >90min, 2= keski- ja kovatehoinen <45min, 3= keski- ja kovatehoinen >45min. Yöllinen sykevälivaihtelu kerättiin kuuden kuukauden mittausjaksolla EMFIT Qs- unisensorilla (Emfit Oy, Vaajakoski, Suomi). Sykevälivaihtelua verrattiin 1) subjektiiviseen tuntemukseen ja 2) raportoituun kestävyysharjoitteluun. Lisäksi sykevälivaihtelun pitkäaikaistrendejä analysoitiin kuukausi- ja viikkotasolla.

Sykevälivaihtelun ja subjektiivisten tuntemusten yhteydet olivat ryhmätasolla tilastollisesti merkitseviä, mutta käytännössä hyvin heikkoja (LFnu r= 0.85; p<0.001 and RMSSD 90min avg r=0.33; p= 0.41). Yksilötasolla subjektiivinen palautuneisuuden tunne korreloi vähintään yhden sykevälivaihtelumuuttujan kanssa 17 / 19 tutkittavalla, mutta tulokset vaihtelivat suuresti riippuen yksilöstä. Ryhmätasolla tyypin 1 kestävyysharjoitus johti merkittävästi korkeampaan yöllisen sykevälivaihteluun verrattuna tyypin 2 ja 3 kovatehoisempiin harjoituksiin. Yksilötasolla vain muutamien urheilijoiden sykevälivaihtelu erosi tilastollisesti merkitsevästi eri harjoitustyyppien jälkeisinä öinä ja sykevälivaihtelun trendeissä oli suurta vaihtelua yksilöiden välillä. Pitkän aikavälin analyysissä havaittiin merkittävästi laskeva trendi sykevälivaihtelun kuukausikeskiarvoissa mittausjakson aikana sekä yksilö että ryhmätasolla.

Tässä tutkimuksessa palautumisen subjektiiviset ja objektiiviset mittarit korreloivat heikosti. Kestävyysharjoittelun intensiteetti vaikuttaisi olevan kestoa merkittävämpi tekijä akuutin yöllisen sykevälivaihteluvasteen synnyssä. Johtuen tutkimuksen rajoitteista tuloksista ei voi tehdä pitäviä johtopäätöksiä, mutta ne voivat antaa tärkeitä näkökulmia sykeseurannan toteuttamiseen hyvän ja huipputason kestävyysurheilijoilla. Sykevälivaihtelun laskeva trendi mittausjakson aikana asettaa kyseenalaiseksi tutkittujen urheilijoiden kuormittumisen ja palautumisen tasapainon kilpailukautta lähestyttäessä ja tämä voisi olla kiinnostava jatkotutkimusaihe.

Avainsanat: Sykevälivaihtelu, urheilija, kestävyysharjoittelu, intensiteetti, kesto, palautuminen, subjektiivinen tuntemus.

ABBREVIATIONS

ANS	Autonomic Nervous System
BCG	Ballistocardiogram
BP	Blood Pressure
BPV	Blood pressure variability
ECG	Electrocardiogram
EEG	Electroencephalogram
HF	High Frequency Power
HR	Heart Rate
HRV	Heart Rate Variability
IBI	Interbeat interval
LF	Low Frequency Power
LF/HF	Ratio of low and high frequency power
PNS	Parasympathetic Nervous System
PPG	Photoplethysmogram
PRV	Pulse rate variability
RMSSD	Root mean square of the successive differences
SNS	Sympathetic Nervous System
SWS	Slow wave sleep
TP	Total Power of power spectral density
ULF	Ultra-Low frequency Power
VLF	Very Low Frequency Power

CONTENT

1 INTRODUCTION

The goal of endurance training is to enhance endurance capacity. The enhancements in this capacity are based on the adaptations in oxygen delivery system and neuromuscular system. To achieve these adaptations athlete must train to disturb homeostasis, the equilibrium state of the body. The training stimulus must be strong enough to disturb homeostasis but at the same time it must be small enough so that the body has ability to recover from it. In case the training and recovery are not in balance, the result may be overstress state. To avoid this, sport scientists and coaches are constantly trying to develop new solutions to follow the stress-recovery state of an athlete. These solutions aim to quantify stress-recovery state objectively but when coaching humans, it is extremely important to take also the mental site into account. Thus, the optimal way for training stress follow-up will most likely be an approach somewhat combining both objective and subjective measurements.

Heart rate variability (HRV) is relatively old variable in exercise science, but its use has blown up during past 5-10 years. Nowadays it seems to be one of the most promising ways to measure the state of autonomic nervous system and this way to get an idea of body´s rest-recovery state. Heart rate variability has been used to estimate the training readiness before trainings, the training load during trainings as well as the recovery from trainings. Different companies have used its parameters to develop precise and easy-to-use follow-up tools for athletes. Some of these tools are based on short, some-minute measurements while the others are providing data from long time periods (days, nights). New measurement technologies are developed constantly and the original way of measuring RR-intervals from bi-electrode- ECG has got competitors from different pulse-based measurements.

This study aims to quantify the nocturnal HRV responses following different endurance trainings in high-level endurance athletes. The HRV is measured by using new pulse-based bed-sensor EMFIT QS. The nocturnal HRV responses are compared to the training data and the subjective feelings, collected from athletes. Also, the long-term changes in nocturnal HRV are identified. The idea of the study is to find out whether the nocturnal HRV is appropriate and effective way to measure the athlete´s training load and training readiness. Furthermore, this study aims to link the mental and biofeedback scores to give coaches some practical tools to evaluate athlete´s overall stress.

2 PHYSIOLOGY OF THE CARDIOVASCULAR SYSTEM

2.1 Overall structure and functions of cardiovascular system

The cardiovascular system includes heart, vasculature, and blood. The heart is the pump of the circulatory system. The alternate contraction of atria and ventricles ensures the blood to be distributed to the various organs (figure 1.). (Smith & Fernhall 2011, 5.)

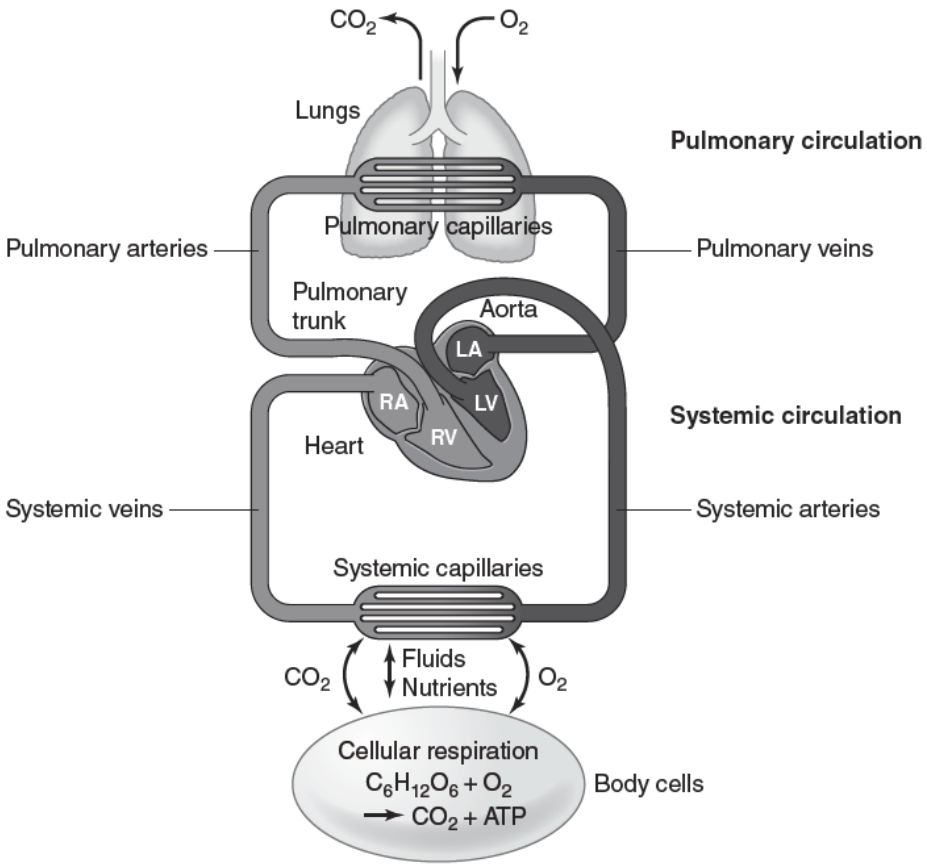

FIGURE 1. The basic anatomy and the main functions of cardiovascular system (Smith & Fernhall 2011, 4). CO_2 = carbon dioxide; O_2 = oxygen; RA = right atria; RV = right ventricle; LA = left atria; LV = left ventricle; $C_6H_{12}O_6$ = Glucose; ATP = adenosine triphosphate.

The cardiovascular system plays three main roles in helping the body to adapt to the physiological demands of the environment: (1) *Transport and delivery*. Respiratory gases, nutrients, waste products, as well as hormones and chemical messengers are transported all over the body within the blood. (2) *Hemostatic regulation.* The maintenance of the physiological equilibrium by regulation of the fluid balance among different fluid compartments, pH, thermal balance and

the blood pressure (BP). (3) *Protection.* Prevention of blood loss through hemostatic mechanisms *and* infection through the white blood cells and the lymphatic tissue. These functions are vital for maintaining homeostasis: Adequate BP helps to provide blood flow for the body tissues and supply optimal levels of oxygen. pH balance is crucial for the enzyme functions and thermoregulation via sweat formation and increased cutaneous blood flow ensures the right operating temperature for bodily functions. Metabolic regulation and transportation of glucose provide cells the energy for their functions. (Smith & Fernhall 2011, 3-5.) In this thesis, the regulatory systems behind the heart rate variability are discussed in more detail.

2.2 Anatomy of the heart

The heart has three major types of cardiac muscle: atrial muscle, ventricular muscle, and special excitatory and conductive muscle fiber. The two former types act in quite similar way compared to skeletal muscle, but the latter type contracts only weakly and its function is rather to conduct the action potentials further along the walls of the heart. (Guyton & Hall 2011, 103.)

The atria are the first chambers to receive the blood as they are filled by the flow from the superior and inferior vena cava (figure 2). After the atria, blood flows to the ventricles. Right ventricle pumps deoxygenated blood to the pulmonary circulation, while left ventricle pumps oxygen rich blood to the systemic circulation. (Smith & Fernhall 2011, 5.)

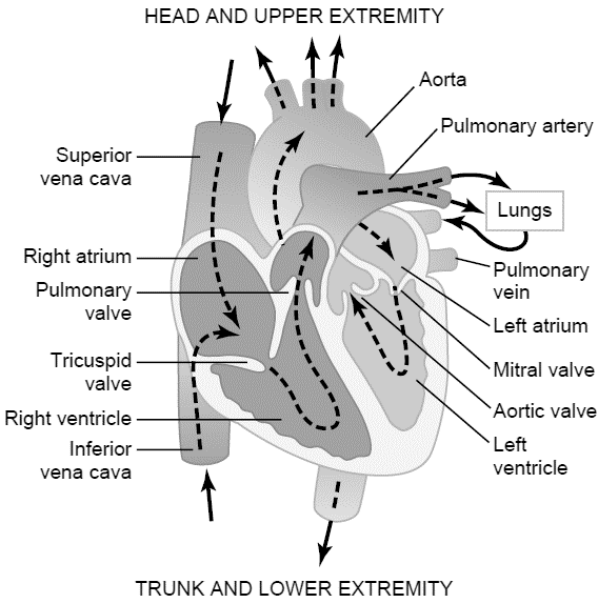

FIGURE 2. Anatomy of the heart and the directions the blood flows during the cardiac cycle. Oxygen rich blood is indicated with the light gray color (Guyton & Hall 2011, 104).

2.3 Electrical conduction in heart

The heart has a special conduction system (figure 4.) that has important role in the timing of cardiac cycle (figure 3.). There are also differences in the conduction of the action potential and as well in muscle contraction in cardiac muscle compared to skeletal muscle. The heart muscle has prominently longer contraction time compared to the skeletal muscle. Ventricular depolarization lasts about 0.2 seconds after the initial spike of depolarization. This causes also the muscle contraction to last as much as 15 times longer than in the skeletal muscle. This phenomenon is due to (1) the special slow calcium channels and (2) the increase in potassium permeability in cardiac muscle cells. The prolonged contraction time of the cardiac muscle cells is also nearly equal to the refractory period of ventricles (0.25-0.30 s), but the refractory period of atria is much shorter (about 0.15 s). (Guyton & Hall 2011, 104-105.)

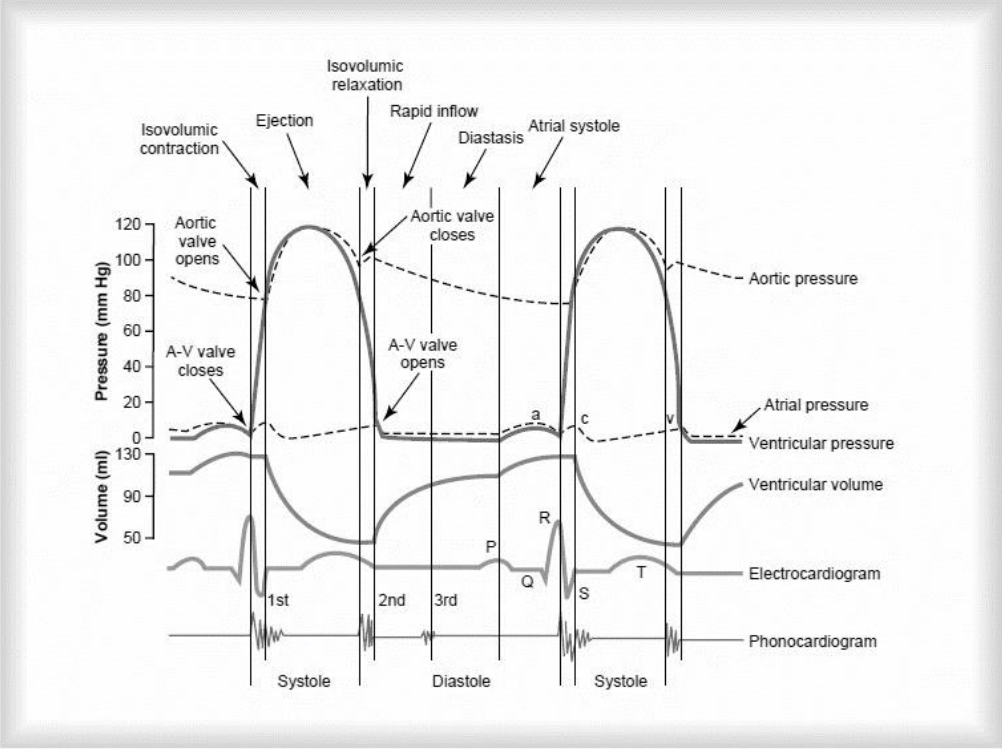

FIGURE 3. Events of the cardiac cycle for left ventricular function, showing changes in left atrial pressure, left ventricular pressure, aortic pressure, ventricular volume, the electrocardiogram, and the phonocardiogram (Guyton & Hall 2011, 107).

The conduction velocity of excitatory action potential signal along both atrial and ventricular muscle fibers is about 0.3 to 0.5 m/s, which equals to 1/10 of the conduction velocity in skeletal muscle. Thus, the conduction in normal cardiac muscle cells is relatively slow. However, the

velocity in the specialized heart conductive system (in the Purkinje fibers) is as great as 4 m/s in most parts of the system. These characteristics of the heart are important to understand when looking at the phases of the cardiac cycle. (Guyton & Hall 2011, 104-105.)

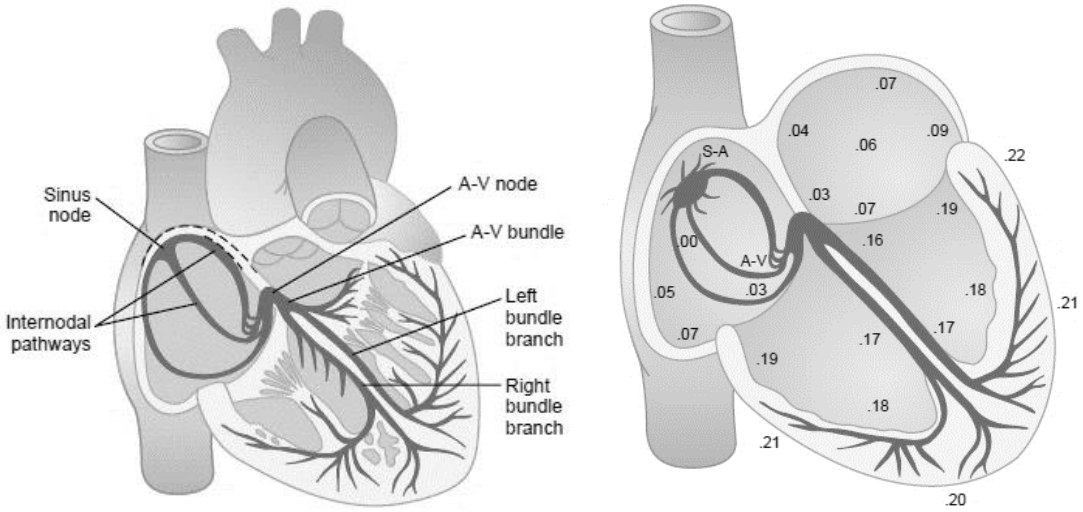

FIGURE 4. The conduction system of the heart and the times of appearance of the action potential after the initial depolarization of the sinus node. (Hall 2011, 117, 120). A-V node = atrio-ventricular node; A-V bundle = atrio-ventricular bundle.

Each *cardiac cycle* includes the cardiac events between the initiation of action potential in sinoatrial (S-A)-node and depolarization of the ventricles. The A-V-node is located in the superior lateral wall of the right atrium close to the opening of the superior vena cava. From there the action potential is conducted rapidly through both atria and then through the A-V bundle into the ventricles. Importantly, there is a delay of more than 0.1 second during the passage of the cardiac impulse from the atria into the ventricles, which allows atria contract slightly before ventricles. This timing of the atrial and ventricular contractions is vital, because it ensures the proper filling of the ventricles and again the pumping efficiency of the heart. (Guyton & Hall 2011, 106.)

2.4 The pacemakers of the heart

The normal intrinsic excitation rate of the S-A-node is around 70-80 bpm. In case S-A-node fails to excite the stimulus, there are also other alternative pacemakers with different intrinsic basal rhythms: The A-V nodal fibers, when not stimulated from some outside source, discharge at an intrinsic rhythmical rate of 40 to 60 times per minute, and the Purkinje fibers discharge at

a rate somewhere between 15 and 40 times per minute. As can be noticed, the excitation rates of the alternative pacemakers are lower than the S-A-node´s intrinsic rate. This is again an important back up mechanism ensuring the discharge in at least some part of the heart in case the superior pacemaker fails to discharge for some reason. (Guyton & Hall 2011, 120.)

The S-A-node has the highest discharge rate and the other pacemakers meet their discharge thresholds only when it fails to produce an action potential. In some cases, it is possible that some other part of the heart than S-A-node starts to act as the primary pacemaker. These abnormal pacemakers are called ectopic pacemakers and they cause also an abnormal sequence of contraction of the different parts of the heart and can cause significant debility of heart pumping. (Guyton & Hall 2011, 120.)

3 CARDIOVASCULAR ADAPTATIONS TO ENDURANCE TRAINING

The best predictor of endurance capacity is the power/velocity at the lactate threshold (anaerobic threshold or the second lactate turn point (LTP 2)) (Coyle 1999). To maximize this power, athlete must follow well-organized, progressive training program for several years. Chronic endurance training leads to several adaptations in cardiovascular system. (McArdle 2010, 460-467.) Most commonly endurance training is associated with increased VO_2max (Achten & Jeukendrup 2003). This may be a simple digit mirroring one´s capacity to perform endurance requiring tasks, but the mechanisms behind the number are much more complicated. Also, while speaking about the endurance capacity, the role of the neuromuscular system must be always considered as well.

3.1 Acute cardiovascular dynamics in endurance training

During steady-rate endurance exercise, the vasodilation happens in active muscles to increase the blood supply for them. At the same time, heart rate and stroke volume increase resulting to increased cardiac output (figure 5.).

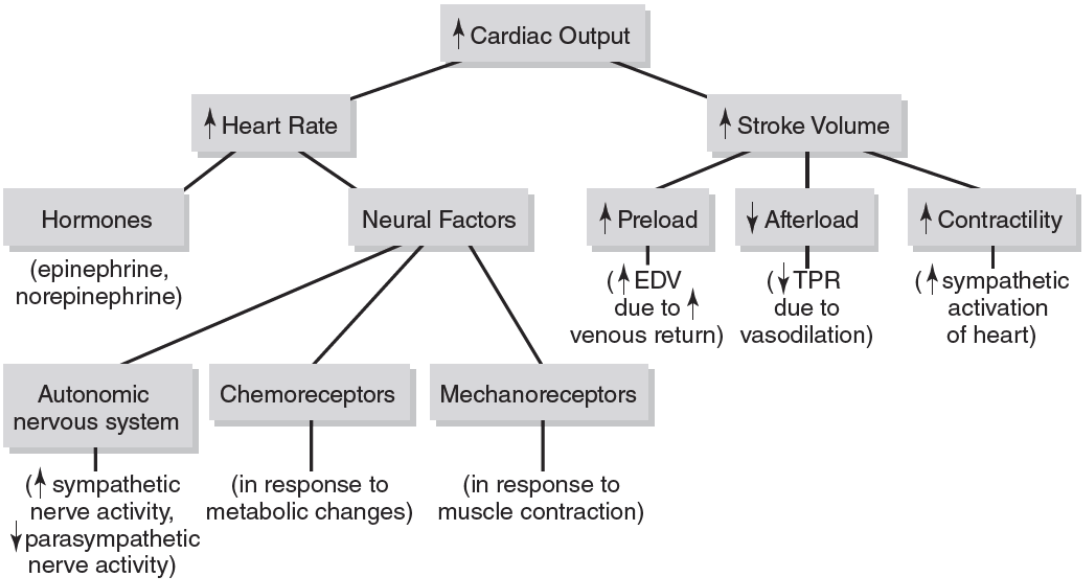

FIGURE 5. Cardiovascular adjustments enabling the increased cardiac output to meet the aerobic demands of exercise (Smith & Fernhall 2011, 140). EDV = end-diastolic volume; TPR = total peripheral resistance.

Regardless of decreased peripheral resistance, increased blood flow ramps up the systolic blood pressure during the first minutes of exercise. After this HR becomes steady but capillaries in

the muscle continue to dilate and thus lowered resistance causes a slight decrease in systolic blood pressure. After the exercise, the capillaries remain dilated for some time and resting systolic BP is lower than the pre-exercise level. This hypotensive response can last up to 12 hours. Diastolic blood pressure remains unchanged during the exercise. In graded exercise, systolic blood pressure continues rising because the constantly increasing cardiac output coincides the effect of vasodilation. During maximal exercise, the systolic BP can be 200 mmHg or higher in healthy athletes, while in the submaximal exercise about 150 mmHg is normal level in healthy population. Diastolic BP remains stable or decreases a bit in maximal exercise. (McArdle 2010, 318-319.)

3.2 Chronic cardiovascular adaptations to endurance training

Chronic endurance training causes linked adaptations in periphery, heart muscle and blood: In periphery the number, size and activity of the muscle mitochondria are increased as a result of mitochondrial biogenesis, which leads to increased a-v O_2- difference. Also, the capillary density of the muscle is increased, which increases the area of O_2-diffusion (figure 6.). (Basset & Howley 2000.)

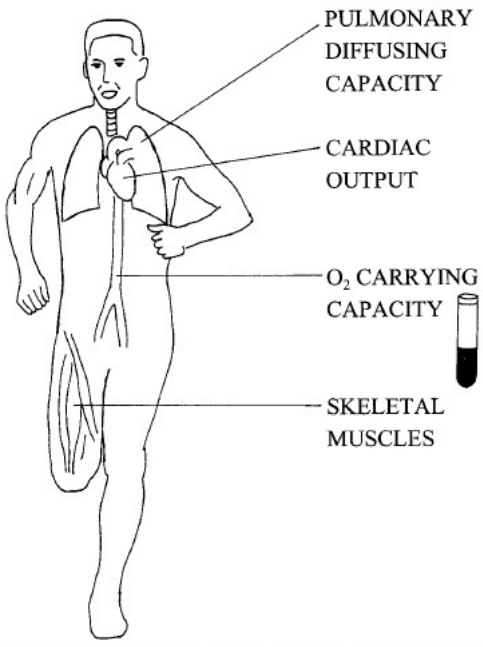

FIGURE 6. Factors contributing to aerobic capacity (Basset & Howley 2000).

The blood plasma volume is also increased following endurance training and increased blood volume increases also venous return, which helps heart´s ventricles stretch more and fill better. Chronic stretching of the ventricles leads to increased size of the left ventricular cavity (eccentric hypertrophy) and increased workload of the heart muscle also leads to left ventricular wall thickening (concentric hypertrophy). Thus, the heart becomes larger and stronger, which again leads to higher end-diastolic volume and increased ejection fraction. These two adaptations affect the maximum stroke volume and again increase the maximum cardiac output. In addition to the greater pumping capacity of the heart and the better O_2-affinity in periphery, endurance athletes have been shown to have higher hemoglobin mass compared to untrained counterparts. This means their blood can bind and deliver more oxygen from the lungs to the working muscles. (McArdle 2010, 458-465)

Changes in the heart are often called with the term “athlete´s heart” (Fagard 1997). Spirito and coworkers (1994) had 947 elite athletes from 27 different disciplines in their study, which concluded that anatomical changes of the heart greatly depend on the sports and gender, as the male endurance athlete had the most prominent concentric and eccentric hypertrophies. This finding has been confirmed by several other studies. The anatomical changes also have functional consequences as the afferent feedback (BP maintained with lower HR than previously, due to the increased stroke volume) from the system seems to lead to increased vagal input to the heart triggering sinus bradycardia. Endurance training has also been found to modify the electrophysiological characteristics of the S-A-node as such, which has an independent contribution to the sinus bradycardia (Stein et al. 2002).

4 AUTONOMIC NERVOUS SYSTEM

4.1 Function of autonomic nervous system

Autonomic nervous system (ANS) controls most of the human visceral functions (Porges 1992). It has important role in modulations of arterial pressure, gastrointestinal motility, urinary bladder emptying, sweating, body temperature, and many other functions. The special characteristic of the ANS is its ability to rapidly alter the visceral functions. The prominent changes in heart rate, arterial pressure or sweating can happen in some seconds after the change in ANS activity. ANS is activated mostly by the centers located in the spinal cord, brain stem and hypothalamus. Also, some parts of the cerebral cortex can empower the activation by excitating the lower centers. Furthermore, ANS is often activated by visceral reflexes that cause subconscious reflex responses in visceral organs. (Guyton & Hall 2011, 748)

ANS has two major portions: sympathetic nervous system (SNS) and parasympathetic nervous system (PNS). Sympathetic and parasympathetic nerve fibers secrete neurotransmitter substances called norepinephrine and acetylcholine. The fibers secreting norepinephrine are called adrenergic and the fibers secreting acetylcholine are called cholinergic. All preganglionic neurons are cholinergic in both systems while almost all the postganglionic neurons of the parasympathetic system are cholinergic and most of the sympathetic neurons are adrenergic. An exception for this is the postganglionic sympathetic neurons innervating sweat glands, piloerector muscle and very few blood vessels that secrete acetylcholine. Acetylcholine, however, is named as parasympathetic transmitter and norepinephrine respectively as sympathetic transmitter. The both sympathetic and parasympathetic stimulations have either inhibitory or excitatory effects in organs. Often these effects act reciprocally but some organs are dominantly controlled by just one of these systems. It is also important to notice the role of the receptors in effector organs; the activated receptor determines the function and the functions of the similar receptors can be pretty much opposite in two different tissues. Also, it cannot be generalized whether sympathetic or parasympathetic activation causes excitation or inhibition in an organ. (Hainsworth 1998). Sympathetic and parasympathetic systems are both normally active at the same time and basal rates of activity are called sympathetic and parasympathetic tone respectively. The continuous tone allows both systems to either increase or decrease the activity in stimulated organ. (Guyton & Hall 2011, 756.)

4.2 Autonomic control of the heart

According to the Frank-Starling mechanism, the force of contraction of heart muscle is dependent on the extent the heart has stretched during the filling. In other words, the heart will contract more powerfully if it is filled to greater extent. The pumping effectiveness of the heart is also controlled by the sympathetic and parasympathetic (vagus) nerves (figure 7.). The cardiac output can be more than doubled by sympathetic activation and minimized by the vagal stimulation, respectively. (Guyton & Hall 2011, 111-112.)

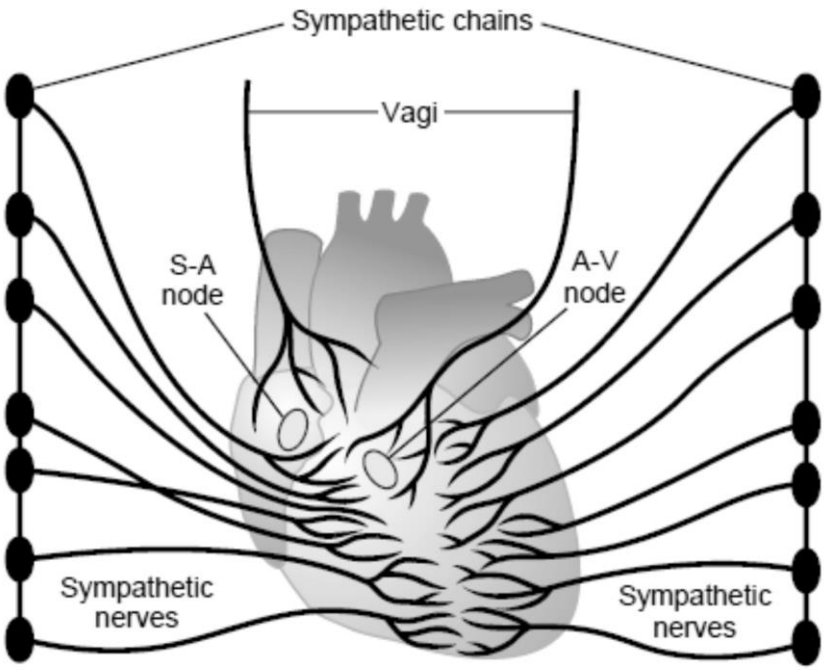

FIGURE 7. Cardiac sympathetic and parasympathetic nerves (Guyton & Hall 2011, 113). S-A node = sinoatrial node; A-V node = atrioventricular node.

Cardiac impulses are controlled by the sinoatrial node (S-A-node), the primary pacemaker of the heart. The intrinsic resting heart rate of a human varies between 110-120 bpm. This heart rate is the natural excitation-rhythm of the S-A-node and without ANS nerve supply to the heart it would be quite constant in all resting conditions. However, the normal resting heart rate varies around 60-80 bpm due to the vagal predominance and is finetuned by the sympathetic and vagal excitation. In general, the increases in sympathetic and vagal activity have opposite effects on the heart: while sympathetic stimulus increases the overall activity of the heart, vagal stimulus decreases the activity. Sympathetic nerves supply the whole heart muscle including the

ventricular muscles, but vagal nerves stimulate the heart mostly via S-A-node and atrioventricular node (A-V-node). A prominent difference between the two systems is found in their latency times: For vagal stimulus, the latency between excitation and maximal cardiac response is only 400 ms, while maximum sympathetic response happens in 20 – 30 s. Thus, only vagal tone has ability to interfere heart rate on a beat-to-beat basis. (Hainsworth 1998.)

Cardiac autonomic nervous activity is controlled by atrial and ventricular receptors, but also baroreceptors and chemoreceptors: Baroreceptors are located in the walls of arteries and they have an important role in controlling arterial pressure. These receptors are pressure-sensitive, and their stimulation increases vagal activity and inhibits sympathetic activity. Baroreflex controls the aortic pressure in rest and exercise. Under resting conditions an increase in blood pressure leads to decrease in heart rate but during exercise, increase in heart rate and blood pressure happen simultaneously. This seems to be due to the baroreflex resetting, which underlines their stabilizing role. Baroreflex is also connected to respiration, as the baroreflex function is almost completely inhibited during early inspiration. Physical exercise and mental arousal also affect the baroreflex control of the heart rate, but not prominently the control of the blood pressure. (Hainsworth 1998.)

Chemoreceptors are can be divided to peripheral and central receptors. The peripheral receptors are located near carotid sinuses and in the aortic bodies of the arch of aorta. Central receptors in turn, are located in the floor of medulla. The primary role of chemoreceptors is to control respiration. They are sensitive to oxygen and carbon dioxide (pressure) changes in arteries. The stimulation of central chemoreceptors is related to cardiovascular system, because it increases sympathetic activity in the heart and blood vessels. Carotid and aortic chemoreceptors have similar functions, but their stimulation can also induce primary reflex bradycardia along with increase in sympathetic activity. (Hainsworth 1998.)

4.3 Functions of ANS in acute exercise

The ANS regulates cardiovascular adjustments during acute exercise to fulfil the physiological demands of the working muscles and cutaneous blood flow, while also maintaining blood pressure and perfusion rates of other organs. At the initiative phase of the exercise, the rapid HR increase happens along with the resetting of arterial baroreflex, which is driven by higher brain centers that stimulate the cardiovascular center in medulla. This adjustment happens mostly due to the parasympathetic withdrawal. The feedback from muscle mechanoreceptors (muscle spindels) fortifies the initial PNS withdrawal and cardiopulmonary baroreceptors are also eventually activated by increased venous return and enhanced pumping efficiency. PNS and SNS both regulate cardiovascular functions during the whole exercise as SNS is setting the tone and PNS is responsible for rapid responses. Autonomic balance is generally shifting from PNS predominance of resting state and low intensities to SNS predominance of maximal exertion. The increasing intensity causes again progressive baroreflex resetting and the feedback from muscle metaboreceptors increases the parasympathetic withdrawal and sympathetic activation. At intensities from moderate to exhaustive, PNS activation is further amplified by sympatho-adrenal activation. (Scott et al. 2017.)

As the loading ends, these processes take place in reverse order: removed CNS drive and extinct afferent feedback from muscle spindels resets baroreflex again to the lower level and initiate the HR decrease. In this fast recovery phase (1 min), HR recovery is mainly due to the parasympathetic reactivation (Perini et al. 1989; Imai et al. 1994; Cole et al. 1999; Coote 2010; Pecanha et al. 2014), but SNS involvement has been suggested as well (Nandi & Spodick, 1977; Kannankeril et al. 2004; Pichon et al. 2004). These fast adjustments are followed by slow recovery phase of HR, which probably happens because of the progressive PNS reactivation and SNS withdrawal. The slower autonomic recovery phase is likely activated mostly by the gradual metabolite clearance and the decreased levels of circulating catecholamines, but also thermoregulatory may have a contribution. (Scott et al. 2017.)

4.4 Adaptations of ANS to endurance training

As described in earlier, continuous endurance training causes both anatomical and physiological adaptations in the cardiovascular system. Chronic endurance type loading decreases resting

HR partly via changes in ANS, but partly this happens independently of ANS (Stein et al. 2002). Increased vagal tone is generally associated with chronic endurance training, but some studies also reported minor changes in SNS input (Smith et al. 1989). On the other hand, the absence of physical activity in bed rest studies has been shown to significantly reduce parasympathetic activity (Arai et al. 1989). Some possible mechanisms leading to sinus bradycardia have been proposed: 1) myocardial stretch reflex to increase HR may be attenuated, 2) BRS might be lowered and 3) peripheral adaptations may also have a contribution to bradycardia. Carter et al. (2003) stated bradycardia is probably a result of central, reflex and peripheral adaptations. (Carter et al. 2003.)

5 HEART RATE VARIABILITY

The first evidence of the heart rate variability, respiratory sinus arrhythmia (RSA) was found already in 1733 (in horse) by Stephen Hales. In 1847 Carl Ludwig was probably the first researcher to find an acceleration in HR and BP during the inhale and deceleration during the exhale (in dog), but the interest in the variation of RR-intervals has grown progressively only since 1950´s when the first prominent approaches to use HRV for human clinical settings was made by Hon and Lee (1963) and Wolf (1967). Both Wolf (1967) and Hon and Lee (1963) emphasized the relationship between heart rate variability and nervous system status (Berntson et al. 1997.). During the last 30 years also several HRV-based solutions for sports have been invented and nowadays HRV seems to be a hot topic in the field of applied exercise physiology.

5.1 Physiological basis of HRV

In the normal human bodily function, the heartbeat and BP intervals are irregular and constantly affected by several regulatory systems that work in different timescales. Since the early examinations of RSA in 18th century, it has been known that the variation in heartbeat interval is closely linked to respiration, but nowadays we know that it also reflects the regulatory mechanisms of autonomic balance, blood pressure (BP), gas exchange, gut, heart, and vascular tone. The oscillations in healthy heart are complex and nonlinear. The variability of non-linear systems provides the flexibility to rapidly cope with an uncertain and changing environment. (Shaffer & Ginsberg 2017.) Recently, *also the heart-brain interaction has been emphasized* and it has been proposed that heart intrinsic neural system has prominent effects on the upper parts of the central nervous system and again for example on the behavior and cognitive performance (Shaffer et al. 2014). Figure 8 is a simplified presentation of the neural divisions contributing to HRV. Conventionally HRV was thought to be produced purely by ANS, but the growing body of HRV research is pointing out, that also brain cortex has interactions with cardiac intrinsic and extrinsic nerve networks (Fatisson et al. 2016). The following introduces theories about heart-brain interaction and complexity of adaptive system.

Polyvagal theory. This theory, introduced by Porges (2001), has an evolutionary approach to describe the heart-brain interaction and its consequences. It divides neural regulation of ANS to three different stages: unmyelinated vagus, sympathetic nervous system and myelinated

vagus. Unmyelinated vagus triggers digestion and responds to thread by suppressing metabolic functions, but also is behaviorally associated to immobilization. SNS, as conventionally described, is responsible for fight-or-flight- behaviors (mobilization), by increasing metabolic output and inhibiting vagal activity. Myelinated vagus, which is unique for mammals, can regulate cardiac output to foster engagement or disengagement with environment. Mammalian vagus is linked to cranial nerves and has ability to regulate social behaviors via facial expressions and vocalization. By detailing these associations, polyvagal theory links biological regulatory systems to social behavior. (Porges 2001.)

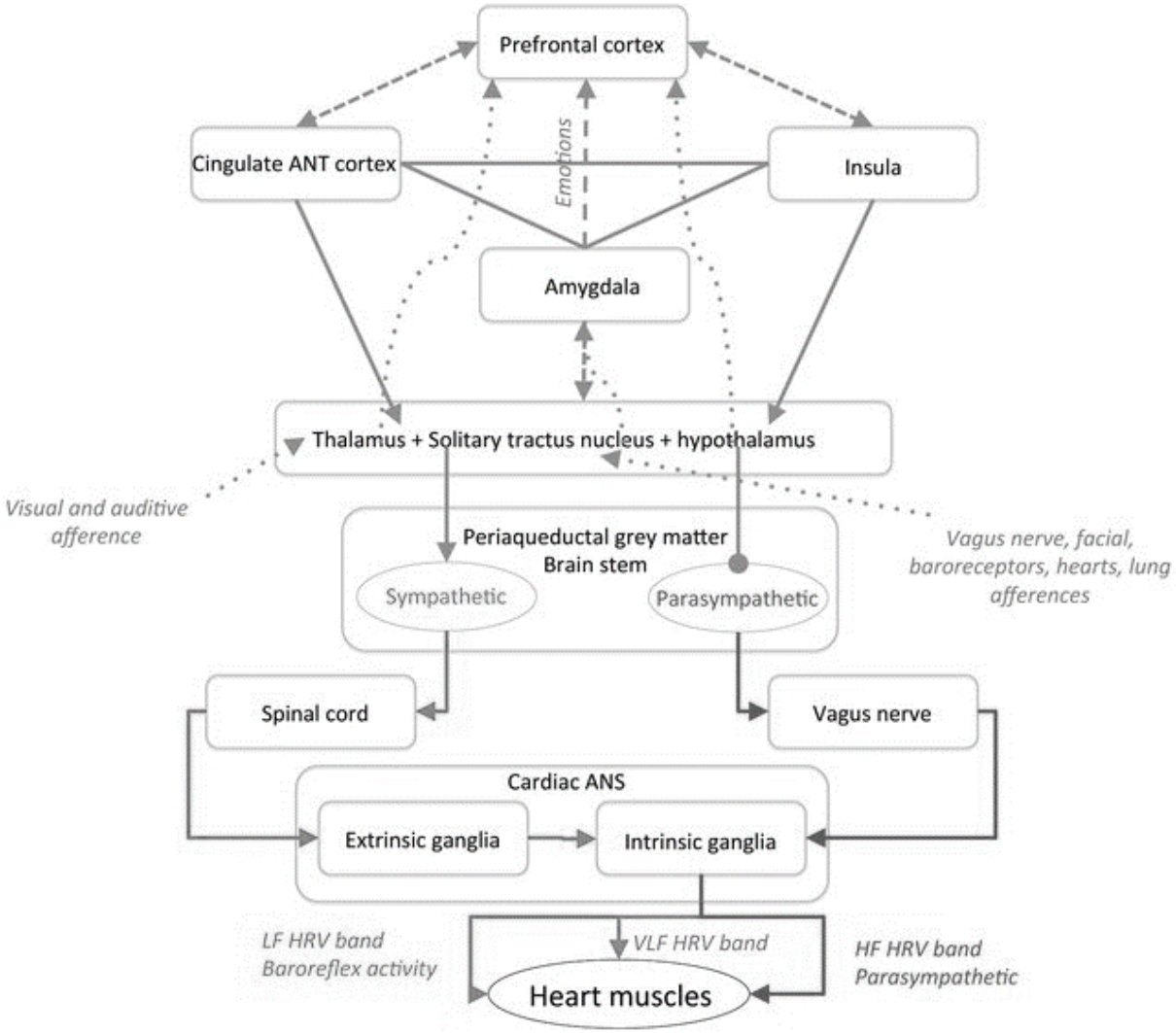

FIGURE 8. Overall nervous network, including efferent and afferent pathways contributing to HRV (Fatisson et al. 2016). LF = low frequency; HF = high frequency; VLF = very low frequency HRV.

Neurovisceral integration model. This model introduced by Thayer and colleagues (Smith et al. 2017) insists that HRV is reflecting adaptivity and flexibility of brain-body system. In case of reduced flexibility, problems occur due to the dysregulation of the system and this can hinder

e.g. the self-regulation. Central autonomic network (CAN) is a component of the internal regulation system that regulates visceromotor, neuroendocrine and behavioral functions. It is considered as the primary nervous base for self-regulatory capacity and it consists of parts of the prefrontal cortex and brain stem that are integrated to nucleus of the solitary tract (NST) and again coupled via efferent vagus with the organs outside of the brain. The coupling between NST and CAN is bidirectional, which means that changes in periphery also lead in changes in CAN. This applies to especially PNS activity and thus HF is a potential marker of this interaction. The recent expanding of this model described eight levels of vagal control ranging from the cardiac intrinsic control to the higher levels of vagal modulation in prefrontal cortex. In addition to these two models, the connections between respiratory and cardiovascular processes to metabolism and behavior have been studied by some research groups. McCraty and Childre`s (2010) Cardiac coherence model is based on slow paced breathing and its beneficial effect on vagal tone and again to physical and mental health. (Ernst 2017.)

5.2 Factors affecting HRV

While planning HRV measurement settings for either practical coaching or for scientific purposes, it is important to understand that the HRV baseline is very individual and affected by several factors. Fatisson et al. (2016) built an influence diagram of physiological and environmental factors affecting HRV. They divided factors in five groups: physiological and pathological, neuropsychological, environmental, lifestyle and non-modifiable factors (Figure 9.).

Non-modifiable factors include individual´s age, gender and genotype:

Age. Studies with children (Kazuma et al. 2002) have shown, that HRV seems to increase with biological growth but in the adult-age, it turns to decrease by age (Byrne et al. 1996; Fukusaki et al. 2000; Moodithaya & Avadhany 2012; Voss et al. 2015). This decrease is probably related to the stiffening of the aortic walls which again decreases the sensitivity of aortic baroreceptors. This increases the blood pressure and finally decreases heart rate variability.

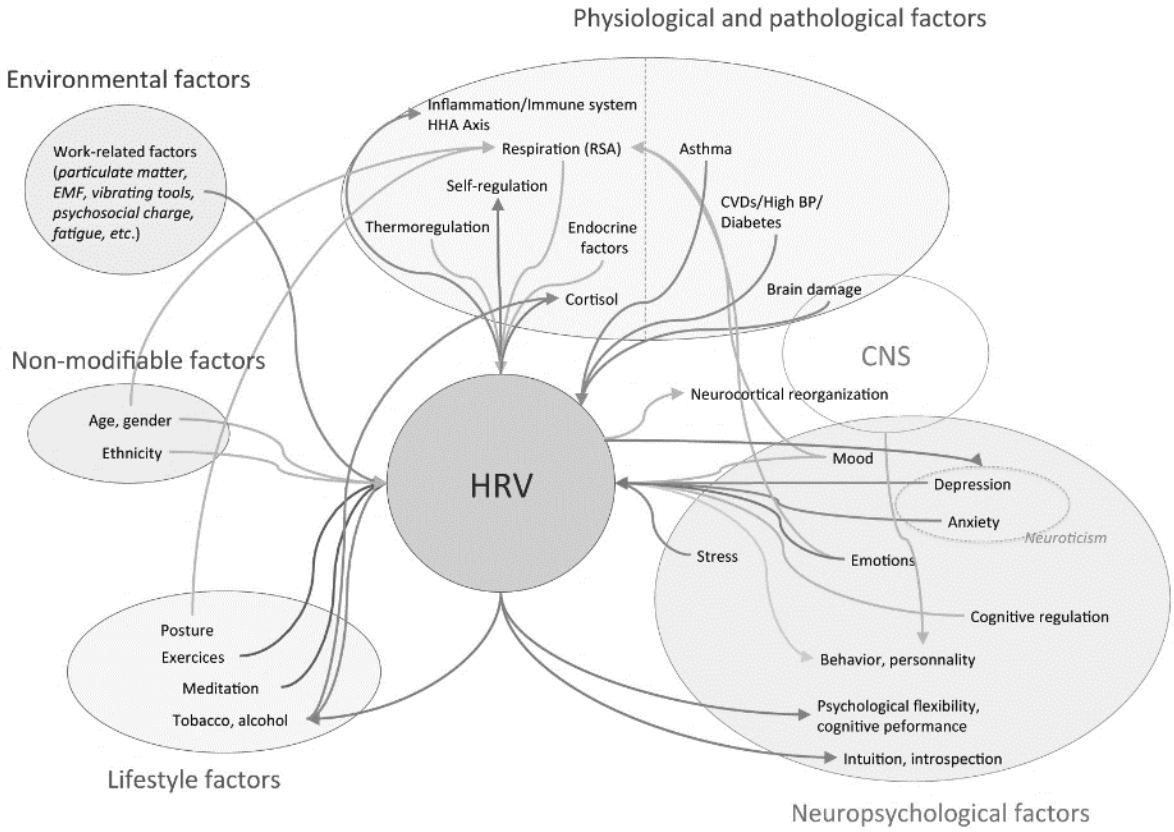

FIGURE 9. Influence diagram of factors affecting heart rate variability (Fatisson et al. 2016).
EMF = electromagnetic fields; CVD = cardiovascular disease; HHA axis = hypothalamic hypophyseal adrenal axis; CNS = central nervous system.

Gender. The meta-analysis of König and Thayer (2016) came out with women generally having lower total power (TP) of the power spectral analysis, but interestingly the vagal contribution (HF) of total power is greater in women compared to men. Women seem to be able to maintain their vagal activity better than men, since Moodithaya and Avadhany (2012) observed faster reductions of HF/LF-ratio with ageing in men compared to women. The difference also remained when the subjects were grouped according to endurance training hours. These findings were proposed to reflect the cardioprotective quality of female gender. (Furholz et al. 2013.)

Genetic contribution. The systematic review of Hill and coworkers (2015) concluded that African Americans have higher resting vagally-mediated HRV compared to European Americans, even when individual´s age, gender, health status and medication use were normalized. The heritability of HRV has been studied in several twin and family studies with different monitoring lengths and respiratory patterns (Kupper et al. 2004; Singh et al. 1999; Uusitalo et al. 2007)

and 13-54 % genetic contribution of HRV has been found. The genetic contributions are not consistent among all the HRV indices.

Physiological factors include hormonal and respiratory factors:

Hormonal factors. Thyroid hormones affect the heart directly by increasing its contractility and indirectly by modifying the SNS response (Polikar et al. 1993). Estrogen levels seem to positively correlate with HRV, which supports the cardioprotective role of female sexual hormones. (Leicht et al. 2003). Also, testosterone and estradiol have been shown to have parasympathetic effect on cardiac modulation (Wranicz et al. 2004; Dogru et al. 2010). Decreased salivary cortisol levels after self-emotional management training were correlated with the increased HRV (McCraty et al.1998). Female gender seems to be more parasympathetically responsive, while men are more sympathetically responsive (Dart et al. 2002). However, the mechanisms between the sexual hormones and HRV are complex and need to be studied further.

Respiratory factors. Thoracic respiration affects the HRV on every respiratory cycle as discussed earlier in this chapter. The physiological basis for respiratory sinus arrhythmia (RSA) has been studied widely, but there is still controversy about certain mechanisms. Berntson et al. (1993) proposed that at least cardiorespiratory rhythm generators, tonic and phasic baroreceptor and chemoreceptor reflexes, pulmonary and cardiac stretch reflexes and local mechanical and metabolic factors have contributions on RSA. Since that different models linking RSA and cardiorespiratory regulatory circuits have been proposed and respiratory-related HRV changes are known to be related to emotional and cognitive functions. (McCraty et al. 2009; McCraty & Childre 2010) The linkage of RSA and vagal tone is generally agreed after it was proven to disappear almost completely with vagal blockade. (Akselrod et al. 1981; Akselrod et al. 1985, Cacioppo et al. 1994; Katona & Jih 1975; Pagani et al. 1986; Pomeranz et al. 1985) Considering these findings, respiratory pathologies are likely to decrease HRV. For instance, in asthmatic children, lower HRV levels have been found (Kazuma et al. 1997).

In addition to the non-modifiable and physiological factors, individual´s lifestyle, environment and neuropsychological qualities are affecting HRV:

Lifestyle factors. Physical activity has clear beneficial effects on HRV. These changes are reviewed in the following chapters. Other lifestyle factors include alcohol, tobacco and drug consumption and meditation practices. Chronic smoking and alcohol consumption decrease HRV, but the impact seems to be reversible in case of drinking alcohol and smoking is stopped. High alcohol consumption is speculated to result in SNS activation and PNS inhibition, which reduces HRV, but also elevated cortisol levels indicating possible role of HPA-axis in this mechanism. Different types of medications also alter the HRV, but depending on the medication and population, the results can be very different. On the other hand, different meditation practices have been shown to have positive effect on HRV (Kim et al. 2014; Delgado-Pastor et al. 2013), which is likely related to their specific respiratory patterns.

Neuropsychological factors. Mental stress, depression and negative emotions have influence on HRV (Kemp et al. 2010; Kemp et al. 2012). Psychological stress elevates resting HR and reduces HRV but also correlates with higher risk of cardiovascular disease (Thayer et al. 2010). Lowered HRV levels are associated also with negative emotions and neuroticism (Di Simplicio et al 2012) yet the effect seems to be reversible with practicing cardiac coherence therapy or positive thinking interventions (McCraty et al. 2009; Fredrickson & Levenson 1998)

Environmental factors the systematic review of Togo and Takahashi (2009) listed occupational factors that may compromise individual´s health. Work stress, working time (shift work), exposure to particulate matter or chemical substances were systematically associated with lowered HRV indices. Fatisson et al. (2016) also mentioned vibrating tools and electromagnetic fields that may have harmful effect that can be observed on HRV. Especially the latter one is an emerging factor also in the leisure time and needs to be studied in more detail.

5.3 HRV monitoring

The effects of autonomic modulation of the heart can be simply described by monitoring the electrical activity of the SA-node, in other words by measuring the HR. The term heart rate variability has become the generally accepted term to describe the variation of both transitory HR and RR intervals. However, it is good to notice that as the HRV is based on the RRI detection from the R-peaks (ventricular depolarization) instead of P-P-intervals (atrial depolarization). Thus, the R-R time interval also contains the fluctuations in atrioventricular conduction

(P-Q interval. However, as the result of complex function of cardiac autonomic regulation, HRV assessed from RR-intervals during sinus rhythm very accurately reflects the variability of the rhythm of the S-A node (Hartikainen et al. 1998, in Malik et al. 1998, 150).

Heart rate variability was originally detected by clinical ECG-tracings. Norman Jeff Holter invented the first portable ECG-device to measure 24hr ECG in 1960´s. (Billman 2011.) Nowadays conventional HR-monitors can be used to collect the HRV-data and sophisticated commercial HRV-analysis tools have been developed over the past ten years. During the last five years several new devices measuring the RRI from the arterial pulse have been released. This *photoplethysmography (PPG)* method is based on the transillumination of the blood vessels and it is validated method to measure pulse rate variability (PRV) that very well corresponds HRV (Gil et al. 2010; Plews et al. 2017). PPG was originally used as a clinical tool, but nowadays even mobile apps using phone cameras can measure PRV from the fingertip and the same technology is used within the wrist HR-monitors. These easy-to-use devices open new opportunities to utilize HRV in daily life situations, but the accuracy of the peak detection still remains slightly better in ECG (Schäfer & Vagedes 2013), even though better algorithms are developed constantly. Recently, also a third solution for HRV detection was introduced: *ballistocardiograph* (BCG) (Kortelainen et al. 2012; Bruser et al. 2013) uses pulse-wave induced pressure changes for non-invasive peak detection. This method is relatively new and still needs more scientific evidence but seems to match well with ECG recordings (figure 10.), when the monitoring procedure is well standardized (Vesterinen et al. 2019).

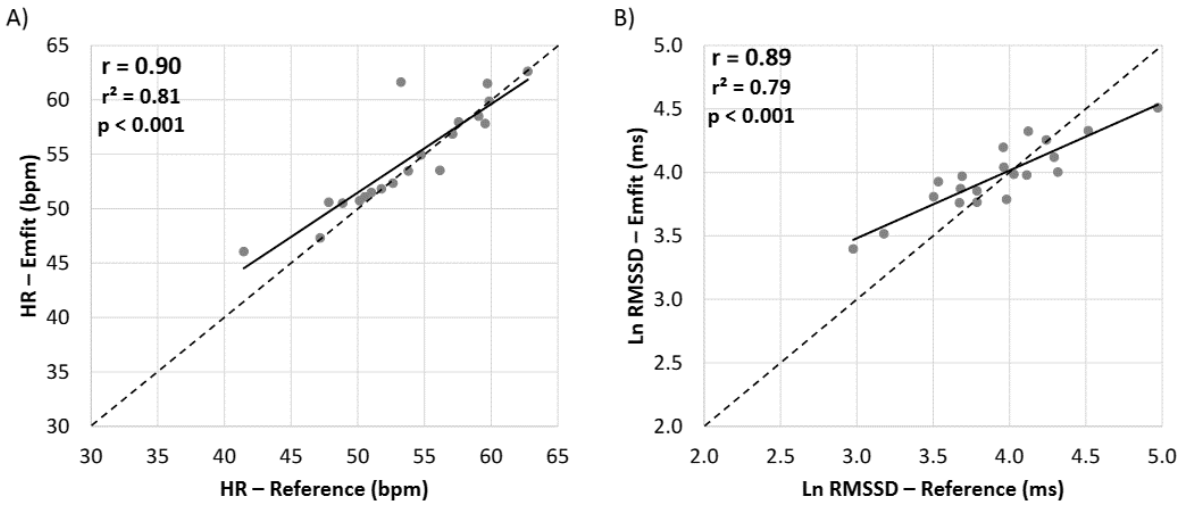

FIGURE 10. Correlation of A) HR and B) Ln RMSSD between the reference ECG and Emfit QS. Reference was 12-lead ECG. (Vesterinen et al. 2019, unpublished).

Considerations related to monitoring. When planning HRV measurement, first thing to consider is the monitoring situation, which often determines the used device. In ambulatory settings, ECG-based solutions (bi-electrode HR-monitor, Holter-device or ambulatory 12-lead ECG) may still be the most accurate method, but wearable PPG-solutions can be more practical for daily measurements – at least in non-science settings. BCG-measurements basically work only in (relatively) static resting situations (bed or chair). HRV measured in supine, standing and sitting posture also give significantly different values (Martinmäki et al. 2006) but sitting or standing measurements may importantly solve the problems related to the parasympathetic saturation in supine rest in highly trained endurance athletes (Buchheit 2014). Different HR levels, depending on the posture and exercising level, also affect the total HRV via the cycle length dependence phenomenon (shorter IBI´s, less chance for IBI variation) (Shaffer & Ginsberg 2017). Second factor to consider is monitoring length. The parameters used in the study may determine the measurement length: When trying to detect HRV changes that relate to the slow-acting regulatory processes, the parameters require measurement lengths of 24-48 hours. (Shaffer & Ginsberg 2017). Thirdly, the monitoring time also affects HRV, since HRV has certain circadian patterns in healthy population (Sammito et al. 2010). During the nocturnal HRV measurements, it is also important to acknowledge the effect of different sleep stages on HRV (Murali et al. 2003). Furthermore, the breathing pattern (controlled or uncontrolled) has to be taken into account and it may partly determine the choice of used parameters (Saboul et al. 2013). Finally, the ectopic beat elimination must be performed. Depending on the setting this may be done either manually (small sample) or with automatized algorithm (bigger samples) (Acar et al. 2000).

5.3 HRV parameters

Long-term (24 h), short-term (ST, ~5 min) or brief, and ultra- short-term (UST, <5 min) HRV can be analyzed by using time-domain, frequency-domain, and non-linear measurements. (Shaffer & Ginsberg 2017.)

Time-domain parameters. Time-domain indices of HRV (Table 1.) quantify the amount of variability in measurements of the interbeat interval (IBI). (Shaffer & Ginsberg 2017.) They can assess the variation generally by standard deviation (SDNN, SDRR, SDANN, SDNNI) or comparing the adjacent interval lengths (pNN50, RMSSD). Also, other measures are used, but

SDNN (clinical 24h HRV) and RMSSD (short-term and ultra-short-term recordings) have gained the highest popularity among the time domain indices. (Shaffer et al. 2014)

TABLE 1. Time domain parameters of HRV (Shaffer & Ginsberg 2017).

Parameter	Unit	Description
SDNN	ms	Standard deviation of NN intervals
SDRR	ms	Standard deviation of RR intervals
SDANN	ms	Standard deviation of the average NN intervals for each 5 min segment of a 24 h HRV recording
SDNN index (SDNNI)	ms	Mean of the standard deviations of all the NN intervals for each 5 min segment of a 24 h HRV Recording
pNN50	%	Percentage of successive RR intervals that differ by more than 50 ms
HR Max − HR Min	bpm	Average difference between the highest and lowest heart rates during each respiratory cycle
RMSSD	ms	Root mean square of successive RR interval differences
HRV triangular index		Integral of the density of the RR interval histogram divided by its height
TINN	ms	Baseline width of the RR interval histogram

RMSSD is the root mean square of successive differences of between normal heartbeats. It is the primary time domain measure used to estimate the vagally-mediated changes in HRV. RMSSD is correlated with high frequency (HF) power and seems to be less dependent of the respiratory rate, which makes it valuable parameter to be used in setting with non-controlled breathing. (Shaffer et al. 2014)

Frequency-domain parameters (table 2.). Frequency-domain measurements estimate the distribution of absolute or relative power into four frequency bands (figure 11.). The Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology (1996) divided heart rate (HR) oscillations into ultra-low-frequency (ULF), very-low-frequency (VLF), low-frequency (LF), and high-frequency (HF) bands. The Task Force report also stated that the analysis should be done in 5 min segments. Yet, other monitoring periods are often used and in these cases, it is important to report the used period, since it has large impact to both time and frequency domain measures. (Shaffer & Ginsberg 2017.)

TABLE 2. Frequency domain parameters of HRV (Shaffer & Ginsberg 2017).

Parameter	Unit	Description
ULF power	ms^2	Absolute power of the ultra-low-frequency band (≤0.003 Hz)
VLF power	ms^2	Absolute power of the very-low-frequency band (0.0033–0.04 Hz)
LF peak	Hz	Peak frequency of the low-frequency band (0.04–0.15 Hz)
LF power	ms^2	Absolute power of the low-frequency band (0.04–0.15 Hz)
LF power	nu	Relative power of the low-frequency band (0.04–0.15 Hz) in normal units
LF power	%	Relative power of the low-frequency band (0.04–0.15 Hz)
HF peak	Hz	Peak frequency of the high-frequency band (0.15–0.4 Hz)
HF power	ms^2	Absolute power of the high-frequency band (0.15–0.4 Hz)
HF power	nu	Relative power of the high-frequency band (0.15–0.4 Hz) in normal units
HF power	%	Relative power of the high-frequency band (0.15–0.4 Hz)
LF/HF	%	Ratio of LF-to-HF power

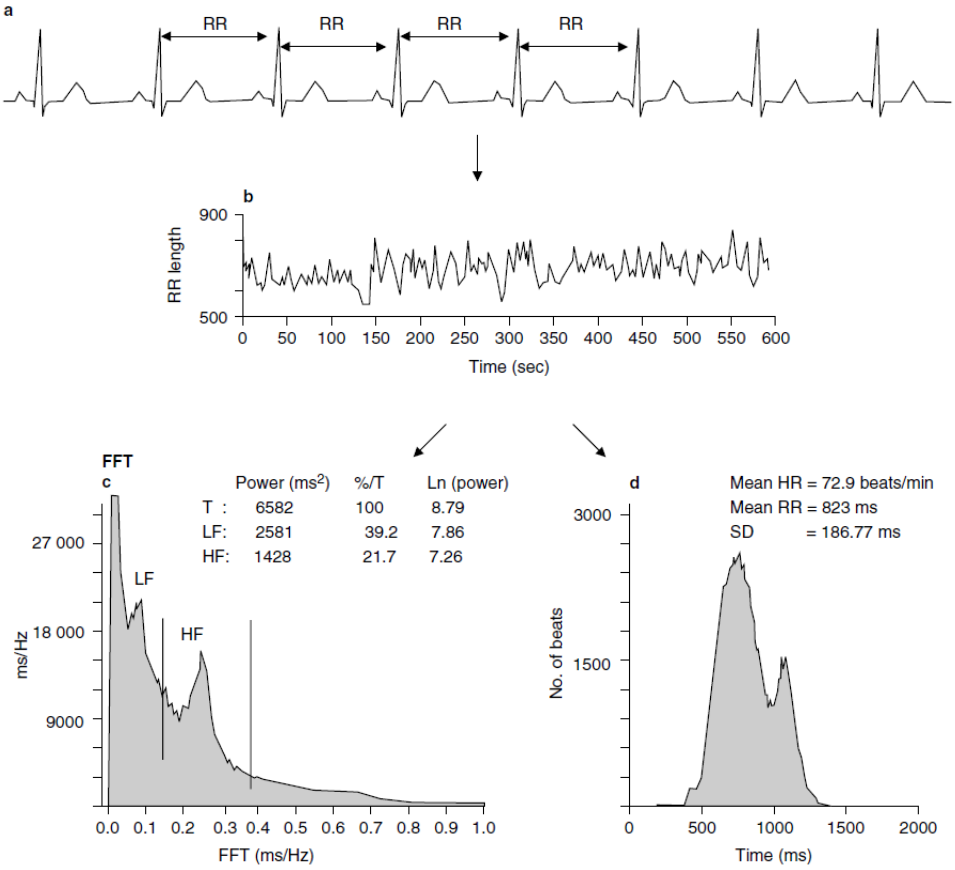

FIGURE 11. Spectral analysis of HRV: from interbeat intervals to tachogram and finally to the power spectrum (Aubert et al 2003). FFT = Fast fourier transformation; T = total power; LF = low frequency power; HF = high frequency power.

HF band is between 0.15-0.4 Hz. It is generally considered as an indicator of vagal tone and seems to be almost entirely affected by the cardiac parasympathetic activity. The oscillations in this frequency band are affected by the respiratory rate and HF band is also called *respiratory band*. HR changes related to respiratory cycle are also called as respiratory sinus arrhythmia. HR is accelerated in the inhale due to the blocked vagal activity and decelerated in the exhale as the vagal tone is restored. Vagal medical blockade has been shown to eliminate HF oscillations in many studies, but it has also reduced LF power. (Shaffer et al. 2014.)

LF band ranges between 0.04-0.15 Hz. It is called as baroreceptor range or midfrequency range because it mainly reflects changes in baroreceptor function at rest. Baroreceptors are stretch-sensitive mechanoreceptors. They measure BP changes in heart chambers, aortic arch, superior vena cava and carotid sinuses. The rise in BP increases the firing rate of baroreceptors and through the vagal afferents stimulus is conducted to the NST in medulla, where the inhibition of vasomotor center and activation of vagal nuclei happens. The PNS activation and SNS inhibition takes place and thus HR and contractility are lowered, and peripheral resistance reduced, which causes BP to be lowered. (Shaffer et al. 2014.)

In ambulatory 24-h HRV recordings, it has been suggested that the LF band also reflects sympathetic activity and the LF/HF ratio has been reported as an assessment of the balance between sympathetic and parasympathetic activity. However, this relationship has been questioned by several researchers who argue that LF rather reflects baroreceptor activity than SNS innervation of heart in resting conditions. In long-term recordings LF seems to estimate sympathetic activity when it is present, but this cannot be translated to short-term recordings and it should be noted that LF from long-term ambulatory recording cannot be replaced with LF from short-term rest measures. (Shaffer et al. 2014.) As mentioned, the role of LF in the estimation of PNS activity is controversial and it has been proposed that systolic time intervals (STI) could be better measures of cardiac sympathetic activity and should be measured in parallel with HRV monitoring to complete the overall measurement of ANS activity (Scott et al. 2017). Already in 1996, Uusitalo and coworkers showed in their double blockade study that all the HRV parameters are mainly determined by the PNS activity (Uusitalo et al. 1996).

VLF band extends from 0.0033 to 0.04 Hz. While low HRV values are shown to indicate numerous health risks, VLF power has stronger associations to all-cause mortality than HF and LF power. Low VLF has been linked to arrhythmic death, post-traumatic stress disorder (PTSD) as well as high inflammation and low testosterone levels. VLF has not yet been studied as widely as HF and LF bands. Some researchers have proposed that long-term regulation mechanisms and ANS activity related to thermoregulation, the renin-angiotensin system, and other hormonal factors can be assessed via VLF power (Akselrod et al. 1981; Cerutti et al. 1995; Claydon & Krassioukov 2008). Sympathetic blockade does not affect VLF power and VLF activity can be found even in tetraplegics, whose SNS innervation to the heart is disrupted. VLF seems to be produced mainly by the heart itself (Task Force 1996; Berntson et al. 1997). The studies of Armour et (2003) and Kember et al (2000) support the statement that the VLF rhythm is generated by the stimulation of afferent sensory neurons in the heart, which in turn activate various levels of the feedback and feedforward loops in the heart's intrinsic cardiac nervous system, as well as between the heart, the extrinsic cardiac ganglia, and spinal column. The lowered cardiac intrinsic rhythm (VLF power) may indicate the presence of some severe disease and high risk of death. In healthy population VLF power seems to increase over the night´s sleep and peak just before waking up. This increase may be associated with the morning cortisol peak. (Shaffer et al. 2014.)

ULF band takes place below 0.0033 Hz, which means that it is measuring the oscillations with time periods of 5 minutes or longer. ULF can only be assessed with 24h or longer recordings and is mainly produced by the circadian oscillations. However, other long-term regulatory systems such as core temperature regulation, metabolism and renin-angiotensin system contribute to the ULF power. Some psychiatric disorders have been detected to change 24h circadian patterns of HR and thus can be observed also as changes in ULF power. (Shaffer et al. 2014.)

The study of ultra-low frequency oscillations hasn´t been very active due to the Task Force report (1996), in which the HRV analyses were advised to be done with 5 min averaged segments. The use of 5 min segments eliminates oscillations with time periods longer than 5 min and thus the entire ULF band. Lately, the spectral analysis has been applied also for the entire 24 h period and several lower frequency rhythms has been found. (Shaffer et al. 2014.)

Correlations between time and frequency domain parameters. The correlations between time and frequency domain have been studied widely. The table 3. from the Task Force 1996 includes approximate frequency domain correlates for time domain indices.

TABLE 3. The approximate frequency domain correlates for time domain parameters (modified from Task Force 1996).

Time domain variable	Approximate frequency domain correlate
SDNN	TP
TINN	TP
SDANN	ULF
SDNN index	TP (mean 5 min total power)
RMSSD	HF
SDSD	HF
NN50 count	HF
pNN50 %	HF

5.4 Practical considerations in training stress follow-up settings

Buchheit (2014) published a comprehensive review about the training status monitoring in athletes, by using HR-derived parameters. He highlighted the importance of standardization of HRV measurements to dissociate the training-induced changes in HRV. In this light, the nocturnal HRV measurements were proposed to have the best possible standardization in real-life occasions. However, as mentioned, sleep stages and also sleep quality affect the nocturnal HRV and thus monitoring HR during slow-wave sleep stages seems to be the most potential way to overcome the challenges in data interpretation. This requires the detection of the sleep stages, but it can be accomplished by using the certain shape of Poincaré plot that occurs during this stage. Nevertheless, nocturnal measurements in practicum are noisy and difficult to implement in daily life. They are also affected by the acute training load of the previous day and may therefore mostly reflect the acute physiological stress of training day rather than accumulated training load. (Buchheit 2014.)

As a result of the aforementioned factors, Buchheit (2014) recommended the use of resting HR and time domain HRV indices in the morning upon wakening. He stated that 5-10 min short-

term measurements of RMSSD or SD1 (of Poincaré plot) are the most useful HRV monitorings among the athletes. Four reasons for this were listed as follows: 1) they are easy to collect even in really short time (less than minute), 2) the impact of respiration on these indices is very low compared to frequency domain measures, 3) time domain parameters can be calculated in Excel and do not require any sophisticated analyzing tools and 4) the day-to-day variation of these measures is likely lower than the variation of spectral indices and especially their ratios (LF/HF). (Buchheit 2014.)

Buchheit (2014) also recommended the use of the natural logarithm (ln) of RMSSD/R-R-ratio to distinguish the effects of parasympathetic saturation from sympathetic overactivity. In parasympathetic saturation vagally mediated HRV is decreased following the saturation of acetylcholine receptors in myocyte level. This phenomenon mostly occurs only in highly trained endurance athletes with very low resting HR levels. In case of saturation, Ln RMSSD/R-R-ratio is decreased prominently, because the additional acetylcholine release does not increase parasympathetic tone in heart anymore and thus the Ln RMSSD is decreased. If the case is more about the sympathetic overactivity, the decrease is seen in both Ln RMSSD and R-R-interval, which means smaller effect on the ratio. The interpretation of Ln RMSSD/R-R-ratio also has some challenges: Firstly, every athlete seems to have individual Ln RMSSD/R-R-ratio profile, which must be considered when making conclusions based on the ratio. Secondly, the ratio is also affected by the phase of the training cycle and thus only longitudinal monitoring from months to years is enough to provide a proper base for optimal monitoring process and conclusions. (Buchheit 2014.)

Smallest worthwhile change (SWC). SWC is a relatively new concept for interpretation of the HRV to program athlete´s daily training. It can be described as the individual optimal zone of HRV and is determined based on the longitudinal data of the athlete. In the studies using this method for HRV-guided training programming this optimal zone has been most often determined as +/- 0,5 standard deviations (Kiviniemi et al. 2007; Plews et al. 2012; Plews et al. 2013). (Buchheit 2014.)

6 HRV RESPONSES TO EXERCISE IN DIFFERENT TIME SCALES

The HRV characteristics described in previous chapter are mainly related to resting state. In addition to this, the HRV dynamics during and post-exercise as well as the chronic HRV changes following continuous exercise training are important to understand and may provide useful information for training impact assessment in competitive sports. In this chapter, HRV dynamics during and after exercise and HRV adaptations following exercise training programs are first discussed. After that, HRV-studies related to different training load assessment protocols are reviewed.

6.1 Acute and chronic HRV-related adaptations to endurance training

6.1.1 HRV changes during exercise

In general, acute physical or mental stress is known to reduce HRV. More detailed information about HRV dynamics corresponding different exercise intensities was gathered in a case study of Hottenrott et al. (2006). They used power spectral analysis to describe the acute effects of endurance exercise in a recreational cyclist and found clear changes in LF_{nu}/HF_{nu}-ratio associated with the changes in exercise intensity (figure 12.).

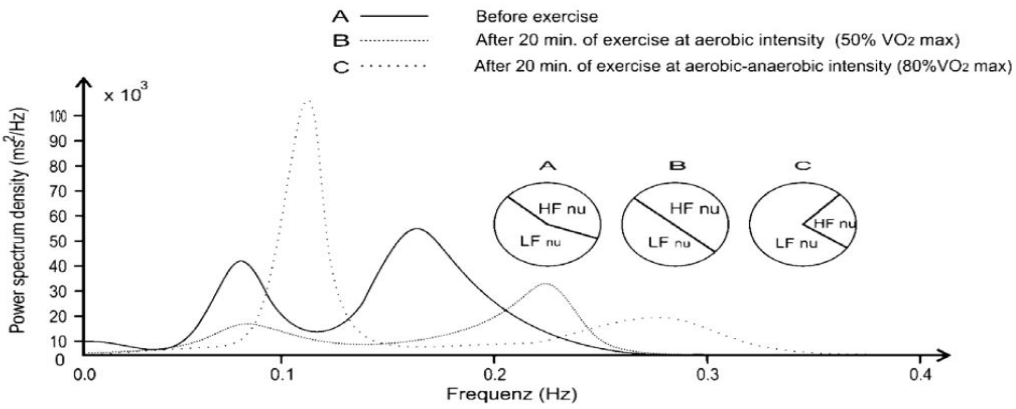

FIGURE 12. Acute alterations in HRV during aerobic exercise (Makivic et al 2013; translated from Hottenrott et al. 2006). VO_2max = maximal oxygen uptake; HFnu = normalized unit of high frequency power: HFnu =HF/(HF+LF); LFnu = normalized unit of low frequency power: LFnu =LF/(HF+LF).

Acute HRV changes during exercise were observed in the study of Cottin et al. (2004). They found higher absolute values of HRV parameters at moderate intensities below the anaerobic threshold when compared to the intensities over the threshold. They also found significantly higher LFn values below the threshold, but the opposite results emerged when intensity was lifted above the ventilatory threshold. This finding indicates that sympathetic predominance occurs during moderate intensity exercise, but in high-intensity exercise predominance of parasympathetic input can be observed. This can be due to the changes in respiratory rate combined with the disappearance of heart´s autonomic control, which occurs in heavy exercise conditions. The gradual switch in LF/HF-ratio at around VT2 can be used to estimate individual´s VT2 noninvasively. The similar dynamics were observed in comparison to blood lactate measurements by Aimet et al. (2001) (figure 13.). They found that TP, HF, LF and VLF decrease with growing intensities until the lactate turn point 2 (LTP2) and after that it slightly increase towards the maximal power output (P_{max}). The third study (Röttger et al. 2005) also used progressive cycling test to examine HRV changes with increasing intensities. Young male soccer players showed HRV minimums (RMSSD, SD1) that matched with 2-4 mmol lactate levels) depending on individual.

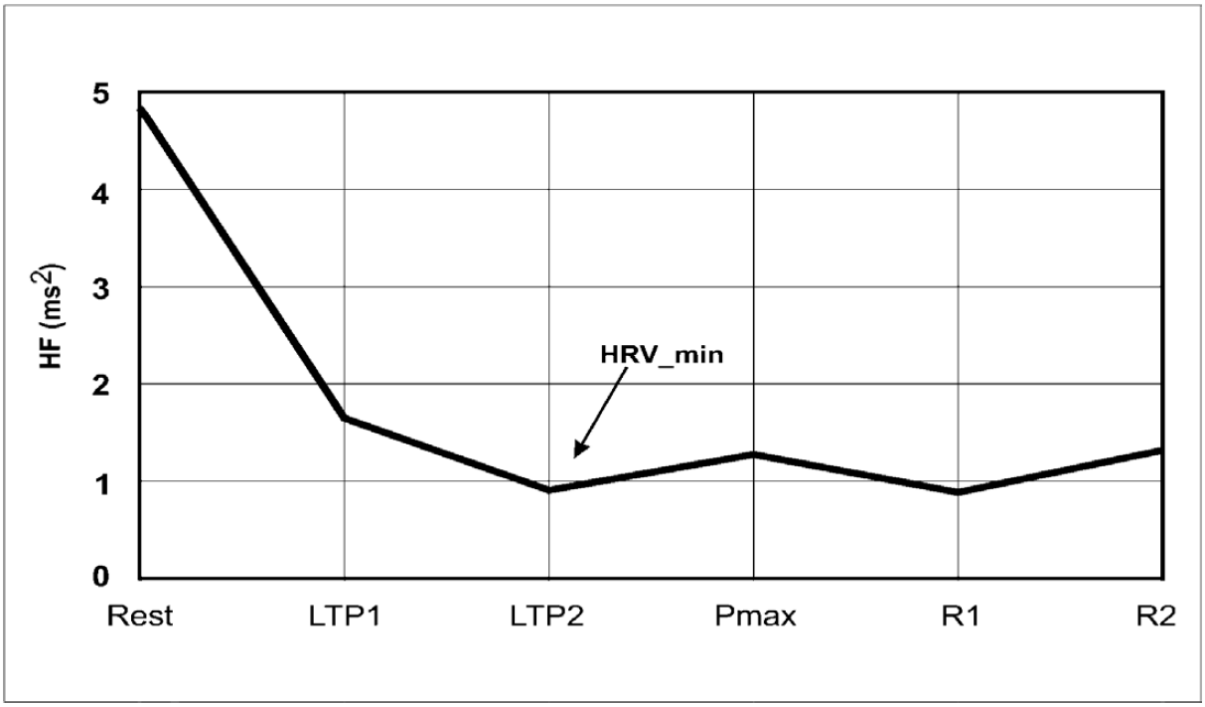

FIGURE 13. HRV minimum correlates well with lactate turn point 2 and can be a potential tool for non-invasive threshold estimation (Makivic et al 2013, modified from Aimet et al 2001). LTP = lactate turn point; P_{max} = maximal power output.

6.1.2 HRV changes post-exercise

HRV has been shown to be lower post-exercise when exercise intensity is higher and continuous exercise with same intensity leads to lower post-exercise HRV compared to interval exercise (Kaikkonen et al. 2007). Post-exercise HR and HRV recovery have been proposed as potential tools for estimating the training load cumulated from the preceding exercise. Kaikkonen and coworkers (2007) found that HRV (HF) recovery was determined by exercise intensity, but not by the duration. Borresen and Lambert (2007) studied the impact of changing training load on exercise HR response and post-exercise HR recovery. They observed significant changes in HR recovery in the increasing training load training group but also noticed that HR during submaximal exercise was not affected by the growing training load. (Borresen & Lambert 2007.)

6.1.3 HRV changes in nocturnal monitoring following an exercise day

The changes in nocturnal HRV were observed in studies of Myllymäki et al. (2011), Hynynen et al. (2010) and Yoshida et al. (2018). Myllymäki and coworkers (2011) studied the effects of vigorous late-night activity on sleep and HRV parameters. They did not find any significant changes in HRV or sleep parameters, but HR was significantly higher after the exercise day compared to control day. Hynynen and colleagues (2010) compared the nocturnal HRV responses following rest day, moderate training day and marathon race (table 4.). As can be supposed, HRV parameters were attenuated after the marathon, but also after the moderate exercise day. The extent of changes in HRV shows that there is likely a dose-response relationship between the training load and the nocturnal HRV response. (Hynynen et al. 2010.)

TABLE 4. Nocturnal HR- and HRV values measured after rest day, moderate exercise day and marathon race day (Hynynen et al 2010).

	Rest			Moderate exercise				Marathon			
HR (bpm)	47	±	2	50	±	3	***	61	±	6	*** ###
SDNN (ms)	142	±	16	126	±	15	***	91	±	19	*** ###
RMSSD (ms)	61	±	19	53	±	19	**	34	±	18	***###
LFP (ms^2)	1798	±	543	725	±	780	*	1031	±	667	** ##
HFP (ms^2)	1214	±	920	952	±	754	**	445	±	612	*** ###
TP (ms^2)	3011	±	1230	2677	±	1408	*	1476	±	1255	*** ###
LF/ HF ratio	2.56	±	2.38	3.17	±	3.51		3.75	±	2.57	
CVRRI (%)	11	±	1.0	10.6	±	1.1		9.2	±	1.7	** #

*, **, *** Statistically significant differences to rest, $p < 0.05$, $p < 0.01$, $p < 0.001$, respectively.
#, ##, ### Statistically significant differences to moderate exercise, $p < 0.05$, $p < 0.01$, $p < 0.001$, respectively.
Abbreviations: average heart rate, HR; standard deviation of RRI, SDNN; square root of the mean of the sum of the squares of differences between adjacent RRI, RMSSD; low frequency power, LFP; high frequency power, HFP: total power, TP; low to high frequency ratio, LF / HF; coefficient of variation of RRI, CVRRI.

In the recent study of Yoshida and coworkers (2018) they used PPG-signal from wrist-computer to measure nocturnal PRV following the intensive futsal exercise. The results were consistent with the study of Hynynen et al. (2010), which underlines the usability of wrist-HR also in night measurements. The nocturnal HRV has been studied also from psychological point-of-view: Hynynen and coworkers (2011) examined the interaction of psychological stress and HRV in nocturnal and orthostatic settings (the provocation of physiological changes by standing up). The morning orthostatic HRV was affected by the mental stress, but in the nocturnal values no significant changes were observed. Thus, nocturnal measurements seem to be less affected by the mental factors and can be better measures of physiological stress compared to morning monitoring. However, when interpreting the nocturnal HRV, the effect of circadian rhythms (Sammito et al. 2010; Huikuri et al. 1990) and sleep stages (Murali et al. 2003) must be considered in order to avoid misinterpretations.

6.1.4 HRV changes following continuous exercise training

The chronic HRV changes following endurance type exercise programs in athletes are related to the anatomical and physiological changes in cardiovascular system. The effects of continuous endurance training on HRV have been widely studied, mostly because of the known health benefits of increased HRV levels. Most studies have reported increased HRV, but also decrease

or no effect has been reported (Hynynen et al. 2007; Pichot et al. 2000; Sandercock et al. 2005: Uusitalo et al. 1996).

HRV in athletic and sedentary groups. Tonkins (1999) observed increased levels of HRV time domain parameters in the group 39 athletes but found only nonsignificant differences between aerobically and anaerobically trained athletes. However, Aubert et al. (2001) found higher RMSSD and pNN50 in aerobically trained athletes when compared to anaerobically trained or rugby athletes. Verlinde et al. (2001) had similar trend in HF spectrum. Aubert et al. (1996) compared middle-aged athletes to sedentary age-matched group and found higher RMSSD in athletic group. The same has been shown with younger endurance athletes in several studies (Dixon et al. 1992; Goldsmith et al. 1992; Jensen-Urstad et al. 1997; Macor et al. 1996; Puig et al. 1993; Shin et al. 1997).

Janssen et al (1993) found significantly different supine HRV values in athletic and sedentary groups, but proposed that this difference mostly occurs in quality, not that much in quantity of HRV. De Meersman (1993) studied HR variation that occurs with breathing in different age-groups of recreational runners and found significantly higher variation in recreational runners compared to sedentary age-matched controls. On the other hand, Migliaro et al (2001) did not find any significant differences between sedentary and non-sedentary groups, but they did not observe bradycardia either, which may implicate that active group was not trained enough to have attained the long-term training effects.

There has been some controversy about the main contributing mechanisms of training induced bradycardia. Already in 1982, Katona and coworkers suggested that bradycardia found in elite oarsmen occurs primarily due to the changes in intrinsic firing rate of the heart (S-A-node). Bonaduce et al. (1998) and Smith et al. (1989) had similar conclusions few years later, and Stein et al (2000) also proposed that lower HR after double blockade of ANS in endurance athletes is a sign of electrophysiological changes in S-A-node following continuous endurance training. Two years later the same research group (Stein et al. 2002) found parallel shifts in sinus automaticity after the double blockade in athletic and sedentary groups. Goldsmith et al. (1997) emphasized the role of physical fitness in vagal modulation of HR and some other studies (Dixon et al. 1992; Jensen-Urstad et al. 1997; Shin et al. 1997) also insisted that PNS activity strongly contributes to resting HR, but also mentioned that it is only one contributor of exercise

induced sinus bradycardia. In the recent study of König and colleagues (2018), HRV parameters were found to weakly reflect the chronic changes in cardiac autonomic nervous activity.The training induced bradycardia seem to be mostly due to the structural changes in S-A-node (D´Souza et al 2014). The potential limitations for these studies are mostly related to uncontrolled respiratory patterns, that may affect the frequency domain measures and incomplete blockades that may question the results related to changes in heart intrinsic firing rate.

Training interventions for sedentary populations. The changes in autonomic control of the HR following endurance training has been studied in sedentary and recreational populations. The adaptations seem to be age- and gender dependent, but also the content and extent of the training seem to be major determinants of HRV changes following different training programs.

Studies reporting no significant changes in HRV measures (Boutcher & Stein 1995; Perini et al. 2002; Loimaala et al. 2000; Davy et al. 1997), mostly had relatively short training periods. Loimaala and colleagues (2000) even proposed that training program aiming to attain significant HRV changes in middle-aged population should last over one year. Their 5-month 4-6 times per week low- to moderate intensity (55-75 % of VO_2max) aerobic training programs were not enough to induce significant changes in HRV. Correspondingly, Nummela et al. (2016) found significant increases in nocturnal HRV following their 4-week moderate-, 4-week high-intensity training program in sedentary individuals. The increased HRV was also associated with increased aerobic capacity VO_2max. (Loimaala et al. 2000.) Similarly, Melanson & Freedson (2001) found significant increase in HF, pNN50 and RMSSD following their 12 weeks, 3 times a week, moderate- to vigorous intensity stationary cycling exercise program. Catai et al. (2002) had similar length running training program with more moderate intensity (70-80 % of HR_{max}). They did not find any significant changes in either young or elderly training groups. Thus, the intensity of the training seems to be one of the main determinants of the exercise induced chronic HRV changes. It has been concluded that low- to moderate intensity training interventions are not enough to induce significant changes in resting HRV, especially if the duration of the training program has been short. The age of the individual also likely has important contribution to the HRV adaptations. (Aubert et al. 2003.) In middle-aged population changes seem to require long-lasting training programs proposed Loimaala et al. (2000), but in younger individuals, adaptations can happen even within 6 weeks of training. (Aubert et al. 2003.) However, interestingly in the study of Levy et al. (1998) the elderly training group had

much greater increase in SDNN compared to younger training group completing similar training program of 6 months. Also, Schuit et al. (1999) observed increases in SDNN, but also in LF and VLF in elderly population after 6-month training intervention. The increase in overall HRV was concluded as beneficial adaptation regarding the cardiovascular health, but the association of LF and cardiovascular health still seems to be controversial (Schuit et al. 1999). As it has been mentioned, the duration and the intensity of the training program seem to important determinants of HRV adaptations achieved by endurance training programs, and limitations of these interventions are also often related to short training periods, too low intensities or too small sample sizes.

Studies in elite and sub-elite endurance athletes. In addition to the training interventions done with sedentary and recreational athletic groups some studies have investigated same effects in competitive and elite athletes. Hynynen et al (2007) studied changes in nocturnal HRV following overload training in international level cross country skiers. Hard training days did not induce significantly different changes in nocturnal HRV compared to easy training days, but significant reductions in LF and HF were found in the end of overreaching period compared to recovered state. Pichot et al. (2000) followed French national level middle-distance runners during their normal 3+1-week (3x hard +1x recovery week) training cycles. They found increased HR and reduced PNS tone (e. g. HF, pNN50, RMSSD) towards the end of 3-week hard training period and the values returned to baseline or even exceeded it after the recovery week. Furlan et al (1993) compared competitive cyclists in detraining and peaking periods and found elevated PNS and SNS activities in peaking period, which would indicate that overall HRV is increased in peak shape.

6.2 HRV for monitoring training load and performance level

Different HRV monitoring settings are potential non-invasive and cost-efficient tools to optimize athlete´s training. Morning supine HRV (Buchheit 2014) and nocturnal slow wave sleep (SWS) HRV (Herzig et al. 2017) measurements have been recommended by different research groups. The both have their advantages: SWS is very steady state for the measurements and nocturnal measurement provides time-efficient alternative for morning measurements. On the other hand, well-standardized morning monitoring with sufficient stabilization time (60- s, Krejci et al. 2018) are also easy and contain often less artifacts in the signal. The both

measurements must be completed at same time of the day, since HRV has certain circadian patterns that may affect the attained values significantly (Sammito et al. 2010). Nocturnal measurements should also have accurate detection of slow-wave sleep stages to make sure that other sleep stages do not distort the results. As mentioned previously, Hynynen et al (2006) used a combination of nocturnal and orthostatic HRV in investigation of overtained athletes. This combination of resting supine and orthostatic HRV measurements was recently emphasized by Schmitt et al. (2015). They also highlighted the importance of the spectral indices in contrast to Buchheit´s (2014) simplified approach to athlete monitoring. The use of weekly averages of HRV measures has been proposed as a superior indicator of fatigue compared to daily values (Plews et al. 2013).

HRV can be used to either monitor and optimize the internal training load of the system or to estimate how the body is responding to the training stimulus and how the exercise performance level is developing. Manzi et al. (2009) found HF decrease to correlate with increased training load (calculated individualized training impact, TRIMPi) and conversely LF increased by growing training load. The LF measured from the last session (~20 days) before the competition positively correlated with the race result. Thus, LF could be useful measure to noninvasively assess the training load in recreational athletes. Plews et al. (2017) found parasympathetic saturation in 3 out of 4 world champion rowers during the successful preparatory period leading to rowing world championships (ln RMSSD: R-R-ratio decreased towards the end of 7-week period). Earlier Plews et al. (2014) had studied elite rowers´ 23wk training period towards the Olympics and found that increases in low intensity (below lactate threshold (LT1)) and high intensity (above lactate threshold 2 (LT2)) training induced considerable changes in ln RMSSD weekly average. Ornelas et al (2017) performed a case study with a recreational distance runner (4 trainings/ week) and the subject had an increase in ln RMSSD already after 3 weeks. The changes in HRV were positively correlated with increased aerobic capacity. Buchheit et al. (2010) studied resting and post-exercise HRV and HR recovery in 14 recreational level runners and found progressive increase in vagal-related HRV indices in 11 positive responders after 9 weeks training period. A positive correlation between the increased running performance and HRV parameters was observed again. These studies indicate that positive changes in aerobic capacity are likely related to increased vagally-mediated HRV indices, but the interpretation of the HRV changes in elite level athletes must be done with caution, since the HRV values can

be biased by the parasympathetic saturation. Also, the possible role of LF in athletic monitoring needs to be studied further.

Bellenger et al. (2016) gathered a systematic review about the monitoring of athletic training status by autonomic heart rate regulation. They grouped the included studies based on the outcomes of the performance development and found small to moderate increase in vagally-mediated HRV parameters of resting and post-exercise states and post-exercise HR recovery within the positive responders. Furthermore, the HR acceleration was largely affected in the one study that assessed it. The studies leading to performance decrements had similar but more trivial changes in HRV parameters and post-exercise HR recovery, but HR acceleration was decreased with attenuated performance. The authors concluded that additional measures of training tolerance might be needed to determine, which changes in HRV parameters are related to positive adaptations. (Bellenger et al. 2016.)

As described above, the use of HRV to estimate how the body is reacting to the training stimulus still needs to be studied to find the right ways interpret the values. However, HRV guided training seems to be a promising tool for endurance coaching. Kiviniemi et al (2007) were the first to investigate the effect of HRV-guided training. They observed significant increases in maximal sustained load ($Load_{max}$) in both groups, but in HRV-guided training group the increase was significantly higher compared to the pre-determined training group and peak oxygen uptake (VO_2peak) was also increased significantly. Vesterinen et al. (2016) also compared pre-determined training program to HRV-guided training. The HRV-guided group improved their running time in 3000m time trial significantly and also a small between group difference was observed in the change of running test time. However, the pre-determined training resulted in greater improvements in VO_2max. (Vesterinen et al. 2016.)

Finally, HRV has been considered as a good non-invasive marker of physiological stress in the body, but to be more holistic within the coaching process it is always important to combine it with subjective feeling and recovery questionnaire. HRV and subjective ratings of recovery interpreted together may provide additional insights in the athlete´s recovery profile. Flatt et al. (2018) studied athlete self-reported measures (ASRM) and found HRV to correlate with mood, sleep quality, stress and fatigue, but not with muscle soreness. This finding may indicate that

HRV is not a complete indicator of the recovery state and thus it must be combined with suitable subjective questionnaire.

7 THE AIM OF THE STUDY AND HYPOTHESES

7.1 Research questions and hypotheses

1. **Are the changes in HRV (measured by EMFIT Qs) consistent with the changes in subjective ratings of recovery?**

Hypothesis 1

Nocturnal HRV RMSSD measured by sleep tracker (EMFIT Qs) follows the trends of subjective ratings of recovery in endurance athletes like it was found in the study of Flatt and coworkers (2018) in sprint swimmers. They found a good correlation between HRV indices and athlete-self-report measures of recovery -questionnaire (mood, stress, fatigue, sleep quality) in NCAA division 1 sprint swimmers. Resting HR and ln RMSSD were measured by fingertip pulse sensor and Ithlete software (HRV Fit Ltd, UK).

2. **Do training intensity and duration determine the nocturnal HRV response?**

Hypothesis 2

Training intensity is a more prominent factor determining the nocturnal HRV response compared to training duration. Myllymäki and colleagues (2012) studied effects of late-night high intensity training on sleep quality in physically active men and cardiac autonomic modulation and found no significant differences in HRV indices compared to control nights (following non-exercise day). However, HR was higher after training days. Hynynen and coworkers (2010) investigated nocturnal HR and HRV indices in physically fit men after rest day, moderate exercise day and marathon race day. They found significantly elevated HR and lowered HRV indices after marathon race compared to rest and moderate exercise days. Thus, the duration and/or intensity of training (training load) seems to determine the magnitude of the changes in nocturnal HRV after a training day.

8 METHODS

8.1 Subjects

Thirty-five (26 men, 9 women), endurance athletes (31 orienteers, 4 triathletes) were recruited from the training groups of Turku regional academy of sports. Participants represented a wide variety of athletic level: 13 of them were high school athletes (aged 14-20), while the best athletes participating were representing the international level in the two named disciplines. The participation criteria were not determined for the athletic level, but all the athletes were representing at least good national level (top10 or Finnish championship medalist) in juniors or seniors. Three athletes did not complete all the measurements. The characteristics of the subjects completing the study are presented in table 5.

TABLE 5. Characteristics of the subjects at the beginning of the study (mean ± SD).

	Women (n=8)	Men (n=24)	All (n=32)
Age (yr)	22.1 ± 3.2	22.2 ± 5.2	22.2 ± 4.7
Body mass (kg)	58.7 ± 7.0	71.0 ± 7.1	67.9 ± 8.7
Height (cm)	169.1 ± 5.4	181.1 ± 6.5	178.1 ± 8.0
BMI (kg/m^2)	20.6 ± 2.0	21.6 ± 1.5	21.4 ± 1.6
VO_2max (ml/kg/min)	52.5 ± 3.6	65.2 ± 5.0	62.0 ± 7.2
HR_{max} (bpm)	189 ± 5	195 ± 11	193 ± 10

BMI, body mass index; VO_2max, maximal oxygen uptake; HR_{max}, maximal heart rate

Before the study, the participants were informed about the practices they would undergo during the study by sending the research information form attached with the consent of participation form. The HRV measurements started immediately after a subject received the Emfit QS-device, but the final written consent to participate the study was given while the athletes were tested first time on treadmill. The participants also underwent blood draws to check the blood profiles in order to ensure that the participants were healthy. In case of abnormalities were obtained in athlete´s blood profile, the further healthcare was guided by the doctor of the study. The study was approved by the Ethics Committee of University of Jyväskylä, Finland.

8.2 Study protocol

The study was carried out as a part of the weekly training schedule in Turku Regional academy of Sports. The measurement period took place between the December 2017 and June 2018 and consisted of two maximal treadmill tests and nocturnal HRV measurements. In addition, the participants were asked to report their training, subjective feeling and sleep quality on daily basis. The athletes were training according to their own training plans. They were asked to report their endurance trainings in the three following categories (table 6):

TABLE 6. Endurance training categories used in this study.

1) Over 90 min low intensity trainings, intensity below aerobic threshold (LT1)
2) Under 45min high intensity trainings, avg intensity (HR) at least 5 beats over the aerobic threshold (LT1), either interval or continuous trainings
3) Over 45 min high intensity trainings, avg intensity (HR) at least 5 beats over the aerobic threshold (LT1)

The main trainings were counted and in case of two trainings from different categories in the same day, the training with the highest intensity was counted. LTP1 = lactate turn point; HR =heart rate.

8.3 Data Collection

The data collection took place between December 2017 and June 2018. During this period, HRV, subjective scores and training data was collected. Not all the participants had exactly the same measurement period, but all of them had at least 4 months of HRV monitoring. Furthermore, physiological data was collected in two maximal treadmill tests. The treadmill running tests were performed in Paavo Nurmi Center, Turku.

Performance tests. The aerobic and anaerobic thresholds, used for the categorization were, determined in the beginning of the study (within the first 4 measurement weeks) by testing each of the athletes in the progressive treadmill running test, which was performed until exhaustion. The same test was performed again in the middle of the data collection period to ensure that the drastic changes in levels of aerobic fitness will not cause false categorization of the training.

The treadmill tests were performed progressively until exhaustion and consisted of 3-minute constant loading stages. The 3 min included the time that was used for the blood sample taking

from fingertip (approximately 15 s). During the sample taking, the treadmill was stopped. The blood lactate concentrations were determined by YSI 2300 STAT+ -lactate analyzer (Yellow Springs Instruments, USA) and the pulmonary gases were measured with Oxycon Pro- pulmonary gas analyzer (Viasys Healthcare GmbH, Hoechberg, Germany). HR was measured with Polar RS800CX- heart rate monitors (Polar electro Oy, Kempele, Finland). The test results in the end of the test were considered as maximal when the respiratory exchange ratio (RER) was over 1.05 and when HR and VO_2 started to plateau. The highest 60s average value was considered as individual´s VO_2max. The reported maximal blood lactate was the highest lactate value measured in 1 min and 4 min post-measurements. The thresholds were determined manually based on the inclination of the lactate curve and breath by breath – respiratory data. Aerobic threshold (mean 1.3 mmol/L, SD ± 0.2 mmol/L) was set at 0.3 mmol/L above the lowest lactate level. Anaerobic threshold (2.8 ± 0.4 mmol/L) was determined as a combination of ventilatory threshold 2 and lactate turn point 2.

The participants reported also their subjective ratings of recovery and sleep quality (after waking-up in the morning) and the physical stress (before going to bed in the evening)**.** Each of these were reported in scale of 1-10, 1 meaning the worst sleep quality and recovery or the lowest level of physical stress and 10 corresponding the best sleep quality and state of recovery as well as the highest level of physical stress. The subjective scores and the training data were self-reported by the subjects.

The nocturnal HRV measurements were performed with EMFIT Qs, a noninvasive, pressure-based (ballistocardiograph) sleep monitor (Emfit Oy, Vaajakoski, Finland, figure 14). The subjects were instructed for the use of the device and the installation was done by the subjects. When travelling, subjects were told to take the device with them. The HRV data was automatically collected to the Emfit-server via Wi-Fi-connection. The HRV-parameters of time (RMSSD) and frequency (TP, LF/HF) domains were also automatically calculated by algorithm (Emfit Oy, Jyväskylä, Finland).

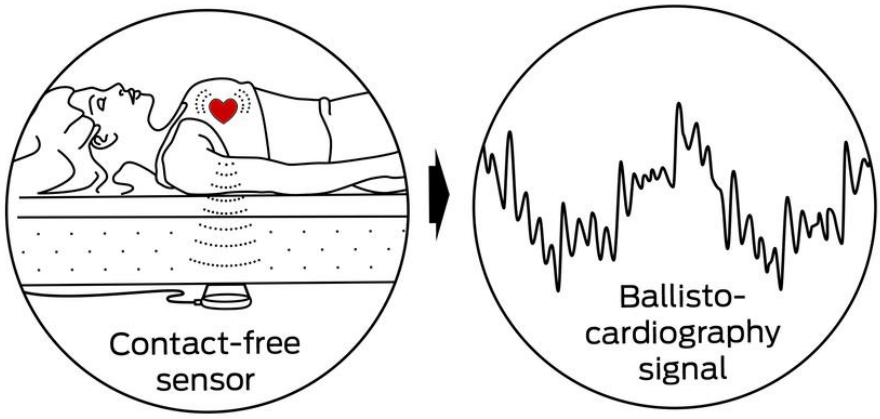

FIGURE 14. Noninvasive EMFIT Qs- sleep tracker was used in the study. The collection of pulse rate variability was done by using ballistocardiograph signal (https://www.emfit.com/science-of-ballistocardiography).

8.4. Statistical analyses

The results are expressed as means ± standard deviations. Statistical analyses were performed for HR- and HRV- parameters, subjective ratings of recovery, stress and sleep quality and self-reported (selected) endurance trainings. Sleep tracker (EMFIT Qs, Emfit Oy, Vaajakoski, Finland) provided also data about sleep stages, movement activity during the sleep time and breathing rate, but this data was excluded from the analyses due to the questionable reliability. All the included data was analyzed at individual and group levels. Additional analyses of HRV parameters averaged on monthly basis were conducted to check the overall trends of HRV during the preparatory and competitive seasons.

The HRV responses and subjective ratings after each training type were analyzed at individual and group-levels by using univariate ANOVA and Tukey´s post-hoc test. Correlations between HRV parameters and subjective ratings of physical stress, recovery and sleep quality were analyzed using Spearman´s correlation coefficient. Changes in threshold values and maximal aerobic performance values, as well as long term HRV were analyzed by using paired samples T test.

9 RESULTS

Participants. 32 out of 35 recruited athletes completed the measurements. However, only 24 athletes had sufficient data for all the analyses. Data was collected from the pre- and mid endurance performance tests, nocturnal EMFIT-measurements and self-reported questionnaires. Sample size for different analyses was variable: HRV data was collected from 33 athletes, but 9 athletes failed to provide self-reported data. Thus, the analyses of training vs. HRV and HRV vs. subjective ratings were performed for 24 athletes, but the HRV trends over the measurement period were analyzed from the data of 33 athletes.

Anthropometrics. The changes in anthropometrics between the pre and mid tests were very small and statistically nonsignificant. The anthropometrical characteristics of the participants in beginning of the study are shown in table 7.

TABLE 7. Anthropometrical characteristics of the participants in beginning of the study.

	Women (n=9)	Men (n=24)	All (n=33)
Body mass (kg)	58.7 ± 7.0	71.0 ± 7.1	67.9 ± 8.7
Height (cm)	169.1 ± 5.4	181.1 ± 6.5	178.1 ± 8.0
BMI (kg/m^2)	20.6 ± 2.0	21.6 ± 1.5	21.4 ± 1.6

Reported endurance trainings. 9-52 category 1 trainings, 11-39 category 2 trainings and 1-13 category 3 trainings were reported by the athletes (figure 15.). Means and standard deviations for training counts are listed in the table 8. To remind about the training categorization, reader is referred to table 6.

TABLE 8. Means and standard deviations for training counts of each training category.

	Training 1	Training 2	Training 3
Mean	22	22	6
SD	11	8	3

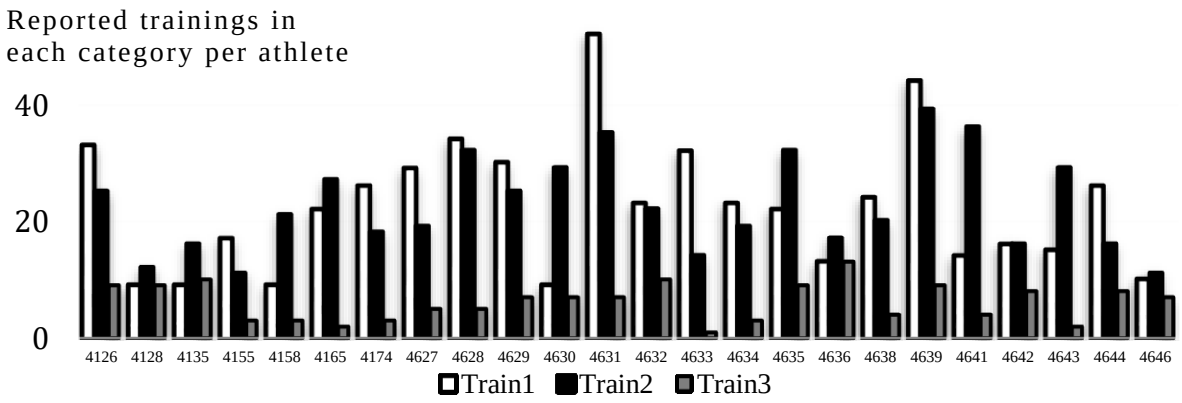

FIGURE 15. Reported endurance trainings of each athlete. Athletes are here coded by device numbers.

Aerobic endurance characteristics. The running speed at aerobic and anaerobic thresholds and maximal speed of the treadmill tests in the beginning and in the middle of the measurement period are listed in the table 9. The tests were separated by 95 days (mean) (84 days median: some athletes had to postpone tests due to injuries). Small, yet significant improvements were seen in aerobic and anaerobic threshold speeds in both men and women. The changes in maximal speed of the treadmill test (V_{max}) were nonsignificant. However, the maximal lactate decreased significantly in the whole group level.

TABLE 9. Maximal treadmill test results in the pre- and mid measurements.

PRE	MEN		WOMEN		ALL	
Aerobic threshold (km/h)	13.9 ±	1.2	11.6 ±	0.9	13.3 ±	1.5
Anaerobic threshold (km/h)	16.7 ±	1.3	13.9 ±	0.9	16 ±	1.7
V_{max} (km/h)	19.8 ±	1.2	16.8 ±	1.1	19.1 ±	1.8
$_{Theor.}VO_{2max}$ (ml/kg/min)	65 ±	3.9	56 ±	3.2	62.7 ±	5.4
VO_{2max} (ml/kg/min)	65.2 ±	5	52.5 ±	3.6	62 ±	7.2
HR_{max} (bpm)	195 ±	11	189 ±	5	193 ±	10
LA_{max} (mmol/l)	10.3 ±	2.4	9.9 ±	2.2	10.2 ±	2.3
MID	MEN		WOMEN		ALL	
Aerobic threshold (km/h)	14.2 ±	1.1**	12.4 ±	0.8**	13.8 ±	1.3**
Anaerobic threshold (km/h)	17.0 ±	1.1**	14.4 ±	0.9*	16.4 ±	1.6**
V_{max}, (km/h)	20 ±	1.1	16.9 ±	0.9	19.2 ±	1.7
$_{Theor.}VO_{2max}$ (ml/kg/min)	65.4 ±	3.6	56 ±	2.1	63 ±	5.3
VO_{2max} (ml/kg/min)	62.3 ±	14.3	52.4 ±	3.8	59.7 ±	13.1
HR_{max} (bpm)	195 ±	11.2	190 ±	5	194 ±	10
LA_{max} (mmol/l)	9.6 ±	1.7	8.5 ±	2.7	9.3 ±	2*

**= $p<0.01$, *$p<0.05$, significant changes from PRE to MID tests.

9.1 Subjective and objective measures of stress and recovery

The first research question was to investigate the association of nocturnal HRV and subjective ratings of recovery. In this study, self-reported measure of recovery (scale 0-10) correlated statistically significantly with HRV parameters in some, but not in all the athletes.

Individual analysis. The highest count of individuals with significant correlation was found in RMSSD values (90min avg and linear fit) (table 10.). Also, in HR_{avg} over 20 % of the athletes had significant ($p<0.01$), yet low correlations ($r= -0.4 – 0.4$). Significant correlations were found in all HRV parameters in at least one athlete. The correlations noticeably varied depending on the athlete, so that one athlete had significant correlation in one parameter, while the other athlete had significant correlation in other parameter.

TABLE 10. The number of statistically significant correlations between HRV parameters and perceived feeling of recovery in the morning, expressed as the number of individuals having significant (positive or negative) correlation (and % of the individuals in the group).

	HR_{avg}	LF_{nu}	HF_{nu}	Morning RMSSD 90min avg	Morning RMSSD linear fit
$p<0.05$	5 (26 %)	7 (37 %)	6 (32 %)	11 (58 %)	12 (63 %)
$p<0.01$	4 (21 %)	3 (16 %)	1 (5 %)	6 (32 %)	5 (26 %)

HR_{avg} = whole night HR_{avg}; LF_{nu}= HRV low frequency power normalized unit, LF_{nu}=LF/(HF+LF); HF_{nu}= HRV high frequency power normalized unit, HF_{nu}=HF/(HF+LF); RMSSD 90min avg= mean of the RMSSD during the last 90min of bed time; RMSSD linear fit= RMSSD linear fit end value, produced from the whole night data.

Group analysis. On the group level significant positive correlations were found in LFnu ($r= 0.85$; $p<0.001$) and RMSSD 90min avg ($r=0.33$; $p= 0.41$).

9.2 Effects of endurance training to nocturnal HRV

Individual analysis. The mean values of RMSSD (morning linear fit end value) are illustrated in figure 16. The responses to reported endurance trainings were highly individual and still very few (n=3) individuals had significantly different RMSSD values after different trainings.

11 out of 24 athletes had decreasing trend in HRV with increased training load (intensity x duration), but also inconsistent (n= 6) and opposite (n= 4) trends were found in some athletes.

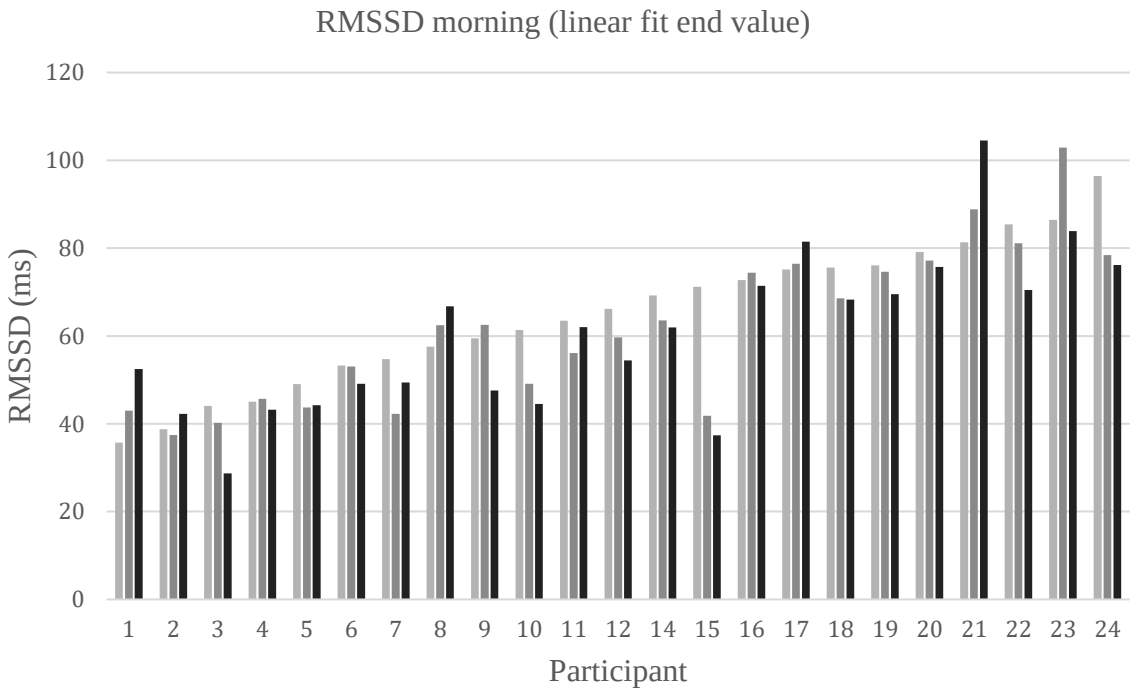

FIGURE 16. Individual RMSSD (linear fit end value in morning) after each of the three training types. 1st, 2nd and 3rd bars indicate individual means of the RMSSD following training types 1, 2 and 3 respectively.

Group analysis. When averaged to group level, RMSSD was found to be significantly different after type 1 training compared to type 2 and 3 trainings (figure 17). However, there was no significant difference between mean RMSSD values after type 2 and 3 trainings.

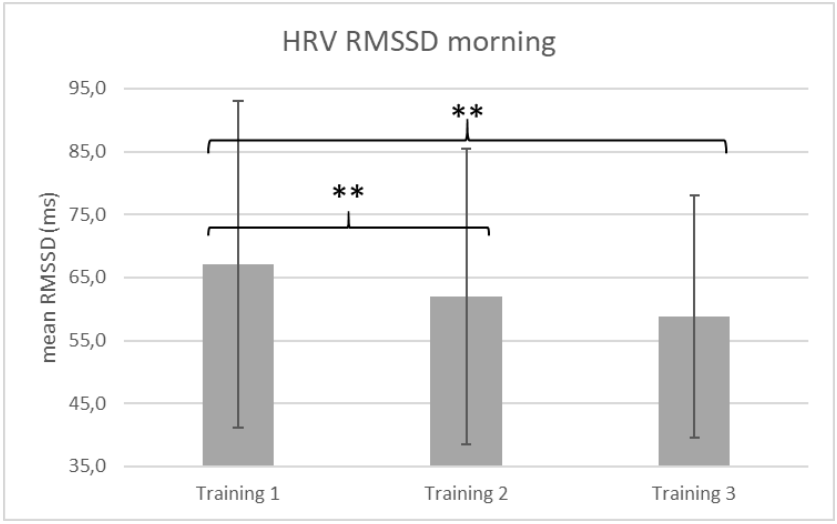

FIGURE 17. Mean (+ SD) RMSSD (morning linear fit end value) after each of the three training types. p<0.01=**

9.3 Additional analyses: Mean HRV changes over the measurement period.

In addition to the analyses regarding to the hypotheses, mean HRV analyses over the 6 months measurement period were performed. HRV monthly average was found to decrease in 31 out of 32 athletes during the measurement period (figure 18). Also, a decreasing trend can be seen in the monthly average values of morning RMSSD (figure 19).

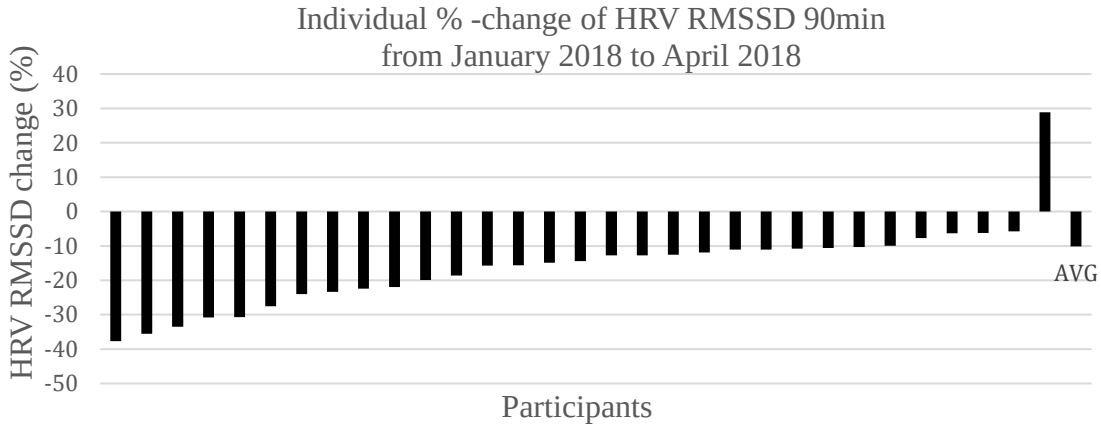

FIGURE 18. Percentual changes in HRV RMSSD since January 2018 until April 2018.

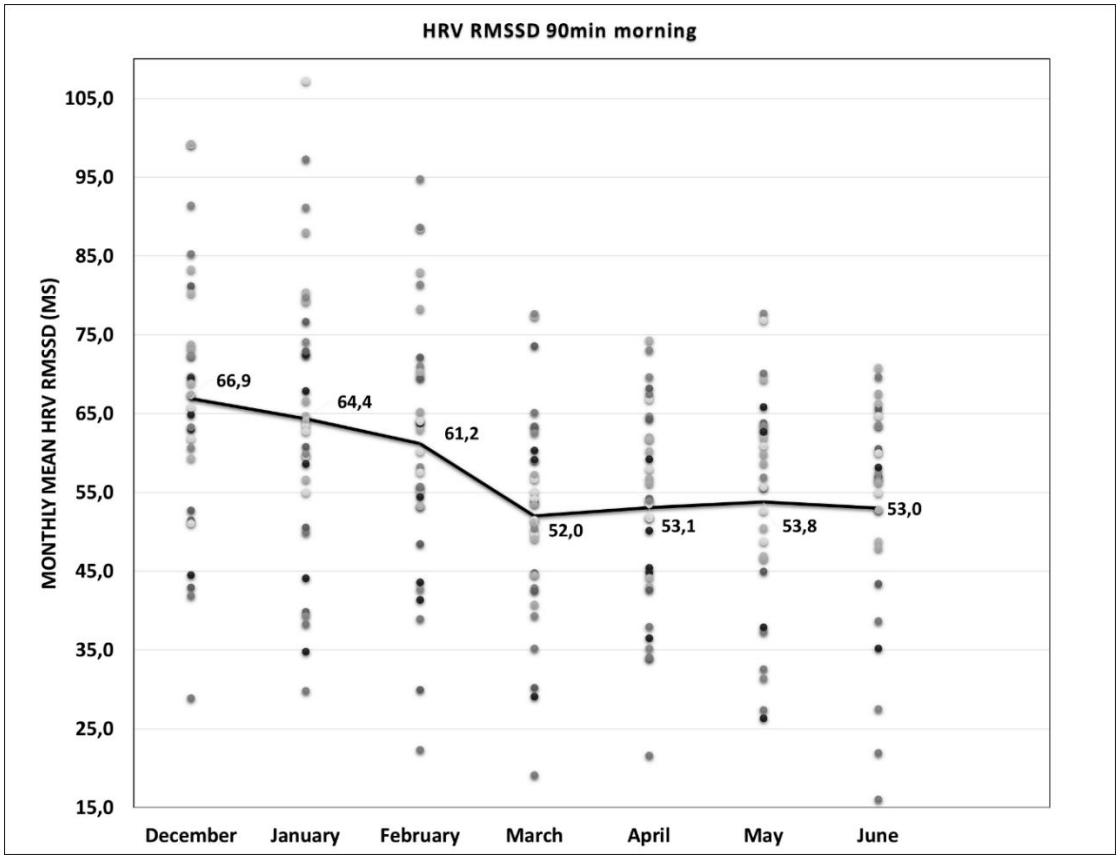

FIGURE 19. Monthly mean values of RMSSD over the measurement period.

10 DISCUSSION

This study was aiming to combine research interests and practical recovery monitoring in a regional sports academy. HRV has been studied in athletes from very different perspectives, but most of these studies have had either low level athletes as test subjects or cross-sectional design. In this study, research questions were addressed to recovery and acute physiological stress induced by different endurance trainings. Due to the unstandardized training and the other factors contributing to HRV levels, the results deliver only descriptive information that can be used to conduct more specific and standardized research settings of HRV responses in endurance athletes.

The main findings of the study were 1) the relatively low (yet partly significant) correlations of subjective ratings of recovery and ballistocardiograph-based HRV and 2) significantly reduced HRV after moderate to high intensity trainings regardless of the training duration (≥15 min Z3-Z5) on the group level. On the individual level, the variation of the HRV to subjective correlations and HRV changes after endurance trainings was extremely high, and significant correlations and changes were found in different parameters in different athletes. In addition to the analyses regarding to the study questions, the extra analyses of HRV long term trends and the descriptive physiological data from the VO_2max-tests were performed for practical coaching purposes.

Association of subjective and objective measures of recovery. A combination of subjective and objective measures of training load has been proposed to be the optimal way for training stress follow-up settings in athletes (Borresen & Lambert 2008). Thus, when evaluating the value/validity of new objective measurement (device), it can be thought that the numbers from objective physiological measurement should match at least at some level with the subjective feeling. In this study self-reported measure of recovery correlated significantly with at least one HRV parameters in almost all the athletes (17/19) that reported their perceived recovery. On the group level, significant, yet low correlations were found in LF_{nu} and RMSSD 90min average. To author´s knowledge this was the first study with competitive endurance athletes using EMFIT Qs sleep tracker and in the light of the results, the device seems to have potential in athlete monitoring. However, the variation in correlations between the athletes was large and not a single athlete had significant correlation between subjective recovery and all the HRV parameters.

The reliability and validity of the device for this kind of setting still requires more detailed and standardized research settings with competitive athletes, even if the validation data seems to be matching ECG signal fairly well (Vesterinen et al. 2019). In this study the issues with the data quality were probably mostly related to the standardization of the testing procedures, as the athletes were using the sleep trackers without continuous supervision.

Compared to previous studies, HRV was matching with athlete-self reported measures (ASRM) of recovery at similar level. In the study of Flatt and coworkers (2018), 15 of 17 sprint swimmers had at least one significant relation with ASRM variables (sleep quality, stress, mood, muscle soreness, fatigue). However, the systematic review of Saw et al. (2016) revealed that in general subjective measures correlate weakly with objective measures. In the review, HRV was compared to Profile of Mood States-questionnaire and positive changes in HRV were reported with regard of overall mood disturbance and fatigue. Authors of this review proposed that subjective measures are the superior to the objective ones and should always be included in training load monitoring setting either alone or combined with objective measures. Schmitt and colleagues (2013) published exceptionally wide follow-up study that aimed to quantify the association of HRV and self-reported fatigue in elite endurance athletes. Their finding was that all the measured HRV parameters were significantly lower in subjectively determined ´fatigued´ condition compared to ´non-fatigue´ condition. Another finding was increased intra-individual variance of HRV parameters in fatigued state, which might indicate that fatigue can result in varying HRV changes.

Effect of endurance training intensity and duration to acute nocturnal HRV responses. The effects of training stress to HRV have been studied on cumulative and acute bases. This study was designed to determine acute effects of training duration and intensity on nocturnal HRV. The intensity was hypothesized to play more important role in determining acute HRV response and results on the group level were consistent with this assumption as the HRV values were significantly lower after moderate-to-high-intensity training days compared to low-intensity training days. The difference between moderate-to-high-intensity trainings of different duration (<45min vs. >45min) was nonsignificant, but there was a trend indicating that higher training load (intensity x duration) leads to lower HRV. On the individual level, most of the athletes (14 of 24) had decreasing trend from low-intensity training to moderate-to-high-intensity training, but there were also athletes with opposite trend. The significant changes between different

training conditions were unfrequent at individual level and the significant changes were found in different HRV parameters in different athletes.

Training counts per each training type varied a lot between individuals and the counts for training type 3 were consistently very low (mean=5.9) compared to other two training types (mean=21.6). This can be a major reason why the difference between training types 2 and 3 was not found. Also, the classification cut off itself enables that the differences of the training load between these two categories can be very small (44 min vs 46 min).

Previous studies about the training intensity and duration effects to HRV have shown that intense acute exercise loading leads to increased nocturnal resting HR (Hynynen et al. 2010; Myllymäki et al. 2012) and decreased HRV (Hynynen et al. 2010), while chronic, successfully implemented exercise loading seems to have opposite effects in long-term (Nummela et al. 2016).

Long-term HRV trends. Even if this study focused on the acute HRV responses, the long-term, averaged HRV data was analyzed also to see the trends over the 6 months measurement period. Interestingly the HRV RMSSD (90min, morning) monthly average decreased over the measurement period. This trend is consistent with the findings of Heikura et al (2015) with recreational runners (VO_{2max} 55.5 ± 5.3 ml/kg/min), but opposite with finding of Raczak et al (2006) in good level runners (VO_2max= 61.5 ± 4.9 ml/kg/min). With this regard, it is relevant to note that athletes´ maximal treadmill performance remained similar between pre- and mid-measurements, but unfortunately corresponding data from the termination of the study is not available. A prominent change in RMSSD90 monthly average can be seen between February and March, which can be an interesting finding for the coaches of the athletes. The collected training data is not sufficient to explain this change, but coaches may speculate this with the training periodization of their athletes.

Limitations of the study. There were several limitations in this study, and they must be considered to ensure that wrong conclusions based on the results will not be made. The starting point, when planning the research setting was to combine practical training stress follow-up to valid research setting. The primary idea was to collect physiological data from good to elite level endurance athletes and provide them tools to optimize their training adaptations. Athletes as the

test subjects in practicum meant that the training standardization would not be possible. Different solutions regarding to training evaluation and selection of the subjective feeling questionnaire were discussed. Initially training evaluation was to be made with TRIMP-calculation and short POMS-questionnaire was proposed to be the measure for the subjective feelings, but finally these were left out. Thinking afterwards, this was a mistake. Also, other limitations could have been avoided during the preparation of the study. In overall, the setting was too wide and demanding for the participants, which reduced the data quality quite much. Problems arose in reporting of subjective data as well as training data, which led to questionable data quality and loss of data.

The data quality was also potentially affected by the sleep tracker used in the HRV measurements. The validity and reliability of this new technology has not been proven by good scientific papers so far. The methodological issues regarding to this device are mostly related to data handling, packing and analyzes, since the peak-to-wave-detection (ECG vs. BCG signal) seems to match quite well (Vesterinen et al. 2019). In this study, raw BCG signal was not evaluated or analyzed, but used HRV data was provided in 3 minutes averaged segments. The treatment of signal was made automatically, and researcher does not have information about the used algorithm. Some issues related to sleep tracker usage also caused data loss and open questions regarding to data quality when sleeping in double bed still remain. The length of the measurement period was relatively long, which also seemed to be too demanding for the participants and which can be seen in data of the last two months. The use of one simple system would be easier for the participants as well.

Almost all these limitations could have been tackled with good research plan, including sufficient knowledge about the certain limitations and proper pilot measurements. Due to the limited time resources, these were however compromised, which reduced the scientific quality of the study. For this size of research project, the measurements were also too broad and with better focus on shorter and more standardized settings would have probably given better results. Importantly, also the data collection should have happened with one simple system, instead of combination of different forms and automatic HRV collection. The lesser manual handling of the data would also affect the data quality positively by reducing the amount of errors.

Strengths of the study. This study focused on the effects of acute training stress to nocturnal HRV in good to elite level athletes. The relatively good athletic level of the participants is a valuable factor when discussing about the results, even if there were problems with the data quality. Achieved results may not be scientifically prominent, but they may serve as a descriptive data about good level endurance athletes, which could guide researchers, when planning new research settings on this field. It is also good note, that the data collected for this study is so large that when averaged, for example, on group or monthly levels, relevant trends may occur despite of the issues with data quality.

Conclusions. Due to the issues with the data quality and standardization, conclusions based on this data are much about speculation. The HRV parameters matched significantly with subjective ratings of recovery in most of the athletes, but the correlations were low and the results were very different in different individuals. Reported endurance trainings also led to quite varied responses in different individuals. HRV was known to be very individual physiological marker, and these results underline that. Yet, on the group level, HRV parameters responded to training as it was hypothesized (greater training impact led to lesser HRV). None of the analyzed HRV parameters was superior to other in this context. In addition to the main results, HRV monthly average was decreasing over the measurement period (from December to June) and a significant drop was seen between February and March. This might question the training periodization of the orienteering athletes during the winter season, since 29 out of 32 participants were orienteers. In overall, more standardized research setting is required even in elite athletes to achieve desirable data quality and be able to make more extensive conclusions.

Practical applications and further research topics. HRV is affected by numerous factors and thus the follow-up and research settings should include standardized monitoring of training and other stressors as well as subjective feeling. Implementation of HRV measurements in athlete´s daily life must be done carefully, so that sufficient data can be collected with the smallest possible disturbance of daily routines. As the scientific evidence linking HRV to recovery state is still somewhat controversial, HRV alone cannot be considered as a comprehensive tool to measure physiological training load and recovery state. However, HRV clearly has potential for this purpose and when combined with other physiological and subjective measures, it can provide important information about the training readiness and long term, cumulative training stress. HRV has been successfully used in HRV-guided training studies to enhance endurance training

adaptations, but at the same time, individual and natural day-to-day variations are found to be really high. Thus, HRV measurements should be standardized well, but also individualized when working with elite athletes to achieve the best possible results. Further studies on the HRV background factors and effects of training in elite athletes are needed to better understand the data that is provided by the new technology in this field.

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APPENDIX

HR and HRV mean characteristics of the participants over the measurement period.

ID	Night count	HR avg	SD	HR min	SD	RMSSD morn-ing	SD	RMSSD morn-ing 90	SD	HF nu	SD
4126	169	42,5	2,0	37,5	0,6	72,2	12,1	67,4	10,2	53,5	5,1
4128	47	54,0	9,1	40,5	2,5	24,0		38,0	19,0	49,2	7,5
4130	122	54,3	2,3	44,0	1,9	61,4	14,7	57,2	11,1	56,9	7,1
4135	154	64,4	2,6	49,2	3,0	50,1	15,9	52,2	14,8	69,7	7,3
4140	131	50,8	3,6	43,2	3,1	20,5	6,7	24,3	7,9	39,8	13,0
4148	100	47,8	3,6	39,4	2,8	52,9	7,2	54,5	10,6	51,8	11,4
4150	137	54,2	2,9	41,9	1,6	79,7	16,6	76,9	12,8	61,8	5,2
4155	75	53,1	4,2	44,2	3,3	38,4	13,5	37,4	13,9	57,4	15,0
4158	113	50,5	3,9	40,4	2,4	79,7	22,2	78,5	19,5	52,1	11,7
4162	77	42,3	1,8	37,8	0,7	54,4	20,1	54,8	16,5	56,9	6,9
4165	168	67,1	9,2	46,3	2,5	73,8	29,5	78,9	30,8	45,8	11,8
4174	118	47,1	3,0	38,9	1,5	68,0	12,4	66,4	12,6	52,3	17,4
4627	177	40,8	0,8	37,0	0,2	71,8	12,4	78,5	31,6	54,9	7,6
4628	125	45,9	1,6	38,2	0,8	70,5	8,5	72,8	9,4	57,0	7,0
4629	197	49,8	4,8	40,1	2,3	53,2	6,8	51,8	12,3	50,6	12,1
4630	149	56,8	4,8	45,7	4,2	61,3	74,1	45,7	33,5	49,0	19,7
4631	186	55,2	5,3	42,3	3,2	62,8	19,1	61,9	18,2	63,3	7,7
4632	118	47,3	2,3	40,3	1,5	61,1	11,0	59,9	10,8	59,7	9,0
4633	97	49,9	4,7	39,0	1,9	81,9	13,1	84,0	21,8	59,3	14,8
4634	145	59,8	6,6	45,0	4,6	56,9	13,1	51,9	26,4	40,5	25,1
4635	201	47,2	2,8	40,2	2,6	70,2	11,1	69,6	10,9	61,8	12,6
4636	160	52,6	2,8	41,8	2,2	74,8	15,4	68,1	15,0	55,4	8,9
4637	125	48,1	3,9	40,5	2,5	57,9	7,5	56,4	9,6	60,8	10,9
4638	92	55,2	6,0	42,7	4,1	44,6	12,5	42,7	7,4	55,2	8,9
4639	195	54,2	3,3	43,7	1,8	53,2	6,2	53,5	7,6	53,3	7,3
4640	163	52,7	3,8	41,9	2,7	65,2	11,4	60,6	10,7	58,2	4,8
4641	193	63,2	2,4	47,3	3,3	38,4	7,7	37,1	10,6	50,9	12,1
4642	136	52,1	3,4	44,6	2,2	46,8	6,9	47,3	9,0	52,2	18,6
4643	164	51,8	7,5	40,5	3,0	63,3	9,2	61,6	11,4	57,3	10,7
4644	115	42,3	1,2	38,0	3,5	77,6	15,3	74,6	16,6	37,5	27,4
4645	175	48,6	3,1	40,1	2,2	72,0	11,7	62,8	9,6	56,2	7,3
4646	145	56,4	3,2	44,1	3,4	56,4	12,0	60,4	11,7	54,9	15,0