CHANGES IN NOCTURNAL HEART RATE VARIABILITY AND ENDURANCE PERFORMANCE DURING A HIGH-INTENSITY OR HIGH-VOLUME ENDURANCE TRAINING PERIOD IN RECREATIONAL ENDURANCE RUNNERS

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ABSTRACT

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It is known that endurance training affects the modulation of the autonomic nervous system and heart rate variability (HRV). As a method HRV may be a potential tool to monitor trainability and endurance training adaptation. The purpose of this study was to examine changes in nocturnal HRV indices and endurance running performance during high-intensity versus high-volume endurance training. In total, 28 recreational male and female endurance runners (35 ± 8 year, VO_{2max} 50 ± 5 ml/kg/min) were matched into two training groups after the 8-week basic training period (BTP) according to HRV, endurance performance and training adaptation during BTP. During the 8-week hard training period (HTP), the high-intensity training (HIT) group (n=14) increased training intensity and the high-volume training (HVT) group (n=14) increased training volume from level of BTP. Basal nocturnal HRV indices (RMSSD, SDNN, LFP, HFP, TP) and endurance running performance were measured at baseline, after BTP and HTP. In the HIT group, VO_{2max} (3.7 %, p=0.005) and V_{peak} (2.4 %, p=0.002) improved significantly during HTP, whereas no changes were observed in the HVT group. Similarly, nocturnal HRV indices (RMSSD: 11.6 %, p=0.034; SDNN: 11.4 %, p=0.005; TP: 2.4 %, p=0.040) increased only in the HIT group but not in the HVT group. Significant correlations were observed between endurance training adaptation and changes in nocturnal HRV indices (ΔVO_{2max}, ΔTP: r=0.54, p=0.045 and ΔV_{peak}, $\Delta SDNN$: r=0.55, p=0.050) in the HIT group. This study showed that high-intensity endurance training induced greater changes in nocturnal HRV indices and endurance running performance compared with high-volume training. In order to lead significant changes in nocturnal HRV indices among recreational endurance runners high-intensity endurance training seems to be needed. Finally, the present findings support the suggestion of HRV as a monitoring tool of endurance training adaptation.

Key words: *heart rate variability, autonomic nervous system, endurance performance, endurance training, high-intensity training, high-volume training, training adaptation*

ABBREVIATIONS

AerT	aerobic threshold
AnT	anaerobic threshold
ANS	autonomic nervous system
BMI	body mass index
BTP	basic training period
ES	effect size
HTP	hard training period
HFP	high frequency power
HIT	high-intensity training
HR	heart rate
HRV	heart rate variability
HVT	high-volume training
LFP	low frequency power
pRR50	percentage of interval differences of adjacent R-to-R peak intervals greater than 50 ms
RMSSD	square root of the mean squared differences between adjacent R-to-R peak intervals
RR	R-to-R peak
SD	standard deviation
SDNN	standard deviation of the R-to-R peak intervals
SDANN	standard deviation of the 5-minute mean R-to-R peak intervals
SWC	smallest worthwhile change
TIRRI	triangular interpolation of R-to-R peak interval histogram
TP	total power
TRIMP	training impulse
VLFP	very low frequency power
V_{AerT}	running velocity at aerobic threshold
V_{AnT}	running velocity at anaerobic threshold
V_{peak}	maximal running velocity
VO_{2max}	maximal oxygen consumption

CONTENTS

1 INTRODUCTION

Regular physical activity related to good physical fitness is accepted to be associated with health, reduced all-cause mortality (Kesäniemi et al. 2001) as well as success in endurance sport (Jones & Carter 2000). Physical training, mainly consisting of aerobic endurance training, improves aerobic fitness with various physiological adaptations, including an altered electrophysiology of the heart that can be observed as changes in heart rate variability (HRV) (Aubert et al. 2003). The term HRV has been used to illustrate the variations in the intervals between the subsequent pacemaker depolarization, R-to-R peak (RR) intervals (Figure 1), as an indicator of cardiac autonomic control (TaskForce 1996). In addition, cardiac autonomic control has been suggested to have an important role as a determinant of training adaptation (Hautala et al. 2009). Understanding of the existing relationship between cardiac autonomic control and endurance training adaptation is still minor. However, several research groups all over the world are interested in it and trying to innovate new practical applications of HRV (e.g. Plews et al. 2013; Vesterinen et al. 2013).

The measurement of HRV offers relatively simple, reliable and a non-invasive tool for assessing autonomic heart rate (HR) control instead of complex invasive methods (Akselrod et al. 1981). As a valid method, the measurement of HRV has received positive acceptance among medical experts, researchers, exercise physiologists, endurance athletes and their coaches. For example, abnormalities in HRV can have diagnostic value in clinical world (Bigger et al. 1992), whereas endurance athletes can monitor the state of overloading and recovery during a training period (Aubert al. 2003). Furthermore, the measurement of RR intervals can be performed with modern HR monitors being as accurate and valid as electrocardiography devices (e.g. Gamelin et al. 2006).

The aims of this study were to examine 1) whether high-intensity and high-volume endurance training induce changes in nocturnal HRV indices and endurance performance and 2) whether a relationship exists between nocturnal HRV indices and endurance training adaptation. It is hypothesised that increases in nocturnal HRV indices and endurance performance will occur and the changes will correlate with each other.

2 AUTONOMIC CONTROL OF HEART RATE

HR, like numerous other bodily functions, is controlled via the autonomic nervous system (ANS) that consists of sympathetic and parasympathetic nerves, even though cardiac contraction would be initiated by self-excitatory fibres also (Guyton & Hall 2006, 112–115, 748). However, the cardiac autonomic input, and subsequently HR, is originally modulated by the following factors: respiration (Berntson et al. 1993), central command, arterial baroreflexes as well as cardiopulmonary reflexes (Rowell & O'Leary 1990). The final common pathway for their neural effect on HR is the cardiac sympathetic and parasympathetic afferents. Thus, the effects of those modulating factors may be determined by the sympathetic and parasympathetic, or vagal, response (Saul 1990). Although the central command seems to play an important role in HR control (Williamson et al. 2006), little emphasis will be placed on it in this literature review because it is difficult to measure and control.
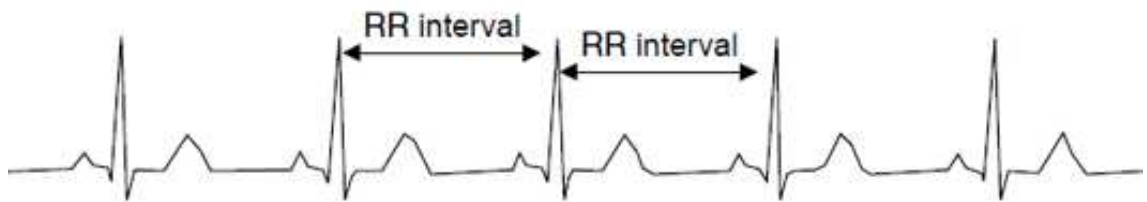

FIGURE 1. An electrocardiography signal with R-to-R peak (RR) intervals marked. Modified from Aubert et al. (2003).

2.1 Sympathetic control of heart rate

The preganglionic neurons of the sympathetic branches originate in the intermediolateral column of the cervical spinal cord and synapse to postganglionic neurons in the stellate ganglion located close to the spinal cord (Smith et al. 1970). Upon entering the pericardial sac, the postganglionic neurons innervate the sinoatrial node (James 2002), atrioventricular node or -bundle (James 2003) and myocardium (Figure 2) (Kapa et al. 2010). With the knowledge of topographic studies, some sympathetic preganglionic neurons synapse to postganglionic neurons in the cardiac ganglion located close to the

sinoatrial and atrioventricular node (Singh et al. 1996). The neural stimulus via sympathetic afferent fibres increase HR by the action of noradrenaline released from the postganglionic neurons binding to the β-adrenergic receptor (Boyett et al. 2000) speeding up the rhythm of the sinoatrial node, from which the cardiac impulse is initiated (Schuessler et al. 1996). Sympathetic response in the sinoatrial node is relatively slow, occurring after 1–2 seconds after stimulus at the earliest and returning to baseline within about 15 seconds (Spear et al. 1979).

Additionally, HR can also be modulated through the hormones, especially noradrenaline. The adrenal medulla secretes both adrenaline and noradrenaline into circulation when stimulated sympathetically (Guyton & Hall 2006, 207). Noradrenaline affects the heart so that it increases depolarization rate of the sinoatrial node (Boyett et al. 2000) and myocardial contractility (Goldberg et al. 1960) by activating β-receptors as explained earlier. However, the neural control is the primary regulation mechanism of HR (Hainsworth 1998).
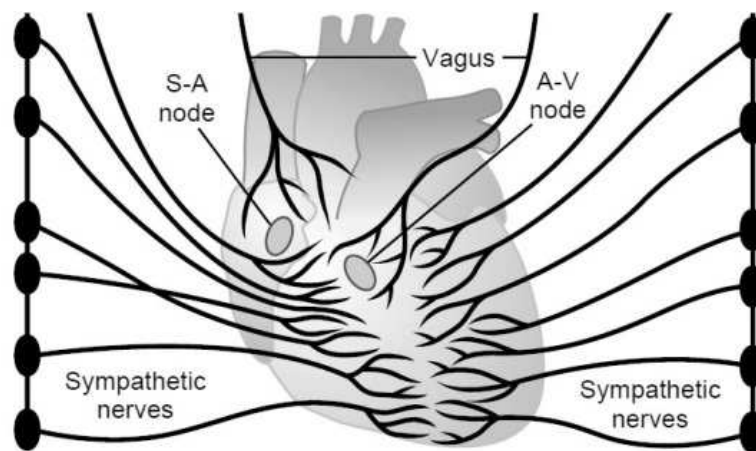
FIGURE 2. Cardiac sympathetic and parasympathetic (vagus) nerves. S-A node, sinoatrial node; A-V node, atrioventricular node. Modified from Guyton & Hall (2006, 113).

2.2 Vagal control of heart rate

The parasympathetic preganglionic neurons arise from the tenth cranial nerve, also termed as vagus nerve, originating in the dorsal motor nucleus of the medulla (Van Stee 1978). The preganglionic neurons synapse to postganglionic neurons in the cardiac gan-

glion like some of the sympathetic preganglionic neurons (Singh et al. 1996). Anatomical studies have illustrated that the vagus nerve provide a rich innervation to the sinoatrial and atrioventricular node (Hainsworth 1998) (Figure 2). In addition to previous areas, some branches of the parasympathetic fibres may also innervate myocardium directly (Johnson et al. 2004). Contrary to sympathetic stimulus, vagal activation decrease HR by releasing acetylcholine from the postganglionic neurons. The mechanism is still unclear but it has been speculated that the binding of acetylcholine to the muscarinic receptors slow the depolarization rate of the sinoatrial node (Boyett et al. 2000).

Respiratory sinus arrhythmia. Beat-to-beat fluctuation of HR at the respiratory frequencies (>0.15 Hz) is termed as respiratory sinus arrhythmia; HR increases during inspiration and decreases during expiration (Figure 3) (Berntson et al. 1993). On the other hand, RR intervals shorten and lengthen, respectively. Respiratory sinus arrhythmia is affected mostly by reflexive modulation of vagal control during breathing cycle (Richter & Spyer 1990). It has been shown that an increase in tidal volume increases the magnitude of respiratory sinus arrhythmia whereas an increase in respiratory frequency decreases it (Pöyhönen et al. 2004). Although the respiratory frequency affects respiratory sinus arrhythmia, the level of vagal HR control is not altered (Hayano et al. 1994).
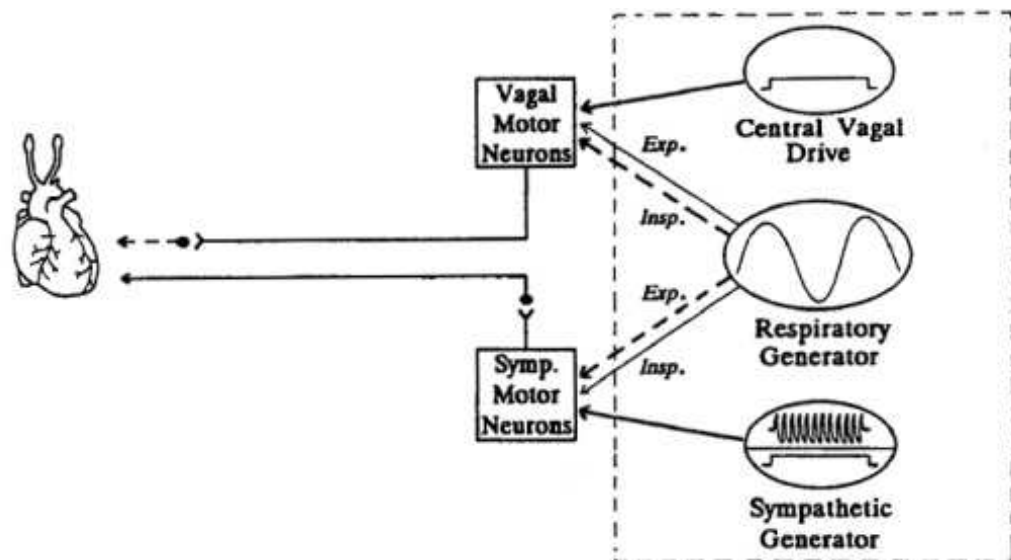

FIGURE 3. Components of central cardiorespiratory mechanisms. *Solid lines* represent excitatory effects and *dashed lines* inhibitory effects of central vagal drive, respiratory and sympathetic generator. Exp., expiration; Insp., inspiration; Symp., sympathetic. Modified from Berntson et al. (1993).

2.3 Reflexes affecting autonomic control

In addition to the central command and respiration, arterial baroreflex plays an important role in the modulation of cardiac autonomic control (Rowell & O'Leary 1990). The baroreceptors, located in the walls of great arteries, regulate HR with specific reflexes initiated by stretch receptors. The baroreflex respond extremely rapidly to changes in arterial pressure. A decrease in arterial blood pressure diminishes the impulses to the vasomotor centre resulting in a decrease in vagal activity and an increase in sympathetic activity. The net effects are increased HR and vasoconstriction of the blood vessels throughout the circulatory system. (Guyton & Hall, 2006, 209–210.) The baroreflex may regulate HR also vice versa; an increase in arterial blood pressure results in decreased HR and vasodilation.

The role of the chemo-, metabo- and mechanoreceptors are much minor than the baroreceptors. Chemoreflex initiated by the chemoreceptors operates in much the same way as the baroreflex. The chemoreceptors are located in aortic and carotid bodies with opposite functions. The stimulation (i.e. oxygen lack, carbon dioxide or hydrogen ion excess) of the aortic chemoreceptors results in increased sympathetic activity via the vasomotor centre, whereas the stimulation of the carotid chemoreceptors elicits increased vagal activity. (Guyton & Hall 2006, 211–212.) The metabo- and mechanoreceptors that are located in skeletal muscles may induce an increase in sympathetic activity after becoming activated (Rowell & O'Leary 1990).

3 HEART RATE VARIABILITY (HRV) – A MEASURE OF AUTONOMIC CONTROL

3.1 Methods of analysing HRV

Power spectral density analyses together with the time domain analysis are perhaps the most commonly used methods to analyse HRV according to the references concerning HRV measurements. Also non-linear methods have been developed based on the theory of non-linear dynamics (Akay 2000). However, the non-linear methods are disregarded in this review. Time domain analysis and classical power spectral density analyses assume that the RR interval signal is stationary fluctuating with the time (Saalasti 2003, 40) being at the same time a major limitation of these methods.

Time domain analysis. As an advantage of the analysing method HRV indices can be calculated with simple statistical methods assuming that the length of RR intervals is determined (TaskForce 1996). The time frame of the analysis may differ between 5 minutes (e.g. Buchheit et al. 2010) and 24 hours (e.g. Furlan et al. 1990). The indices such as the standard deviation of the RR intervals (SDNN) and the standard deviation of the 5-minute mean RR intervals (SDANN) are derived from direct measurements of RR intervals (TaskForce 1996). It must be mentioned that SDNN increases while the duration of analysed recording increases (Saul et al. 1988). Unlike SDNN and SDANN, the square root of the mean squared differences between adjacent RR intervals (RMSSD) and the percentage of interval differences of adjacent RR intervals greater than 50 milliseconds (pRR50) are derived from differences between consecutive RR intervals (TaskForce 1996).

Geometrical methods offer an alternative technique to analyse time series data of RR intervals. The lengths of RR intervals are plotted on the x-axis of the plot and the numbers of each RR interval lengths are plotted on the y-axis presenting the distribution of the sample density (Rajendra et al. 2006). The triangular interpolation of RR interval histogram (TIRRI) is determined as the width of the baseline distribution measured as a base of triangle, whereas the HRV triangular index is calculated by dividing the total

number of all RR intervals by the maximum of the sample density distribution (Figure 4) (TaskForce 1996). HRV triangular index has been considered to be highly insensitive to artefacts and ectopic beats, because they are left outside of the triangle (Scherer et al. 1993).
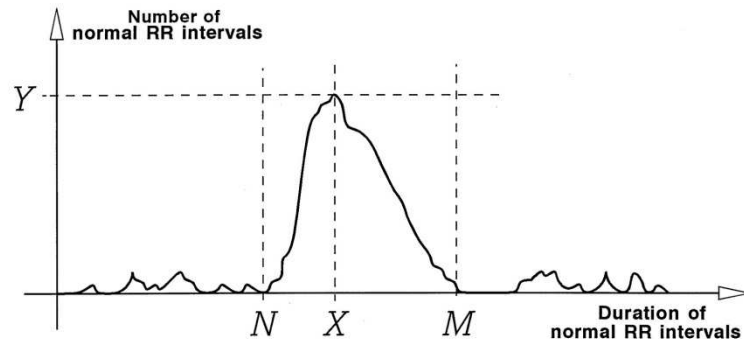

FIGURE 4. The sample density distribution of the R-to-R peak (RR) intervals. X, the most frequent RR interval length; M, N, markers of the baseline distribution; Y, the maximum of the sample density distribution. Modified from TaskForce (1996).

Spectral analysis. In the power spectral density analysis the equidistant RR interval signal is decomposed into its frequency components, which are then quantified in terms of their relative intensity (TaskForce 1996). Nonparametric fast Fourier transformation and parametric autoregressive model represent the classical power spectral density analysing methods with the limitation of stationarity. The modern power spectral density analysing methods, such as wavelet transformation (e.g. Verlinde et al. 2001), short-time Fourier transformation (e.g. Martinmäki & Rusko 2008) or goarse graining spectral analysis (Yamamoto & Hughson 1991), can also be used for non-stationary RR interval signals. Traditionally, the frequencies of the RR interval signal is distinguished into three spectral components: very low frequency power (VLFP, <0.04 Hz), low frequency power (LFP, 0.04–0.15 Hz) and high frequency power (HFP, 0.15–0.40 Hz) components (Akselrod et al. 1981). The frequency ranges listed above are suitable for resting conditions recommended by the committee of TaskForce (1996).

To obtain the fast Fourier transformation spectrum from the tachogram (the length of RR interval plotted as a function of time), RR interval data is divided into overlapping segments, which are then windowed with a Hann, Hamming or triangular window. The fast Fourier transformation spectrum is calculated for each windowed segment separate-

ly. (Niskanen et al. 2004.) Finally, the spectrum of the segments is averaged and can be plotted against the frequency (Figure 5a) (Pichon et al. 2006). Computational efficiency, simple implementation and a representative graphical output are listed as the advantages of fast Fourier transformation. However, limited frequency resolution occurs due to windowing when used short time frames. (Kay & Marple 1981.)

Parametric autoregressive spectral analysis has better frequency resolution for short frames of data than nonparametric fast Fourier transformation spectral analysis (Marple 1977). Consequently, smoother spectral components (Figure 5b) can be distinguished with independently pre-selected frequency bands (Bartoli et al. 1985). The order p (see e.g. Rajendra et al. 2006) for the autoregressive model that best represents the selected series of RR intervals must be estimated prior to the spectral analysis (Akselrod et al. 1985). The selection of the model order to be used may affect inaccuracy to final analysis even though different kinds of criteria for the estimation have been published (Fagard et al. 1998).
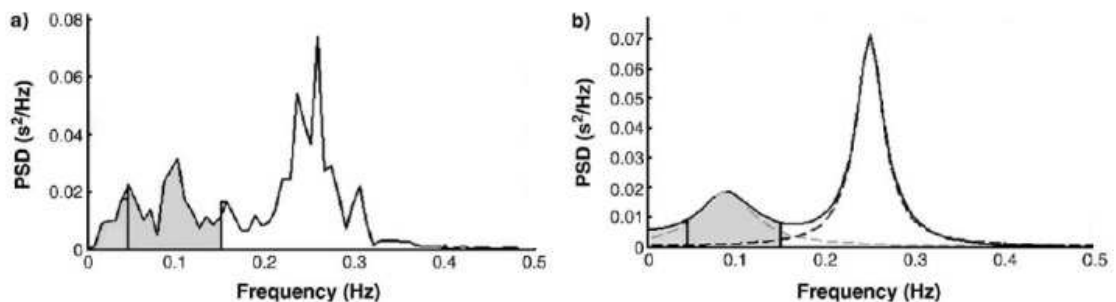
FIGURE 5. Heart rate variability representations of fast Fourier transformation (a) and autoregressive model (b) spectrum. The spectral components, very low-, low- and high frequency power, are separated with a vertical line. PSD, power spectral density. Modified from Pichon et al. (2006).

3.2 Relationship between autonomic control and HRV

HRV results from a dynamic relationship between sympathetic and parasympathetic (vagal) control of HR that can be modulated by a co-activation, co-inhibition or activation of one with an inhibition of another one (reciprocal control) of the divisions of the ANS (Berntson et al. 1991). Since the control of the two autonomic divisions is oppo-

site, an increase in HR may be due to vagal withdraw, sympathetic activation or both (Allen et al. 2007). The level of sympathetic and vagal activity can be quantified with the methods discussed previously using the length of RR intervals instead of HR. HRV indices derived from HR may misrepresent the concrete level of autonomic activity because of inherent nonlinearity between autonomic control and HR (Berntson et al. 1995), although HR and RR intervals have similar statistical and distributional characteristics (O'Leary 1996).

The relationship between autonomic control and HRV has been examined by blockade studies with graduated dose of administration of certain drug (e.g. Pichot et al. 1999). Especially the development of spectral analyses has enhanced the knowledge of the autonomic control of HR. According to the several studies, the absolute HFP corresponds to the modulation of vagal tone, and further vagal activity, as shown in blockade studies (Akselrod et al. 1981; Malik et al. 1993; Bloomfield et al 1998; Akselrod et al. 2001; Golberger et al. 2001). An equally explicit correspondence between sympathetic control and HRV has not been reported. However, Pagani et al. (1986) have suggested that LFP reflects sympathetic activity when expressed as normalized units; LFP divided by total power (TP, a sum of LFP and HFP). Later Montano et al. (1994) have supported the suggestion of Pagani et al. (1986) with their findings of changes in normalized LFP during passive orthostatic task. Additionally, it has also been suggested by several researchers that LFP includes both sympathetic and vagal modulation (e.g. Akselrod et al. 1981; Saul et al. 1990). Recent studies have reported findings that support the suggestion of the joint effect of sympathetic and vagal activity (Uusitalo et al. 1996; Taylor et al. 1998; Martinmäki et al. 2006). Finally, the ratio of LFP and HFP has been used to measure the balance between sympathetic and vagal activity in some circumstances (Aubert et al. 2003).

Most time domain and spectral HRV indices are strongly correlated with each other when recording duration is close to 24 hours (Table 1). High correlations exist because of the mathematical and physiological relationship (TaskForce 1996). SDNN, TIRRI, HRV triangular index and TP represent overall HRV encompassing all frequency components during the period of recording (Kleiger et al. 1992), whereas SDANN has been used as an estimate of long-term components of HRV, linked to sympathetic activity (TaskForce 1996; Brennan et al. 2001). Furthermore, RMSSD together with pRR50 has

been shown to estimate HFP variation in HR representing vagal modulation (TaskForce 1996). Alternatively, the insufficiency of discrimination between the sympathetic and vagal activity has been regarded as the major limitation of the time domain analysis (Aubert et al. 2003). However, when used the long-term recordings the results of spectral analyses are equivalent to those of time domain analysis, which are moreover easier to calculate (TaskForce 1996). Furthermore, Al Haddad et al. (2011) have shown that HRV indices provided by the time domain analysis have smaller coefficient of variation than spectral HRV indices and therefore reflect cardiac autonomic control more reliable than spectral indices.

TABLE 1. Approximate correspondence of heart rate variability indices of the time domain and spectral analyses applied to long-term (24-hour) recordings, based on TaskForce (1996).

Time domain index	Approximate spectral index correlate	Autonomic activity
SDNN	Total power	Overall HRV
SDANN	VLFP	Sympathetic activity
RMSSD	HFP	Vagal activity
pRR50	HFP	Vagal activity
TIRRI	Total power	Overall HRV
Triangular index	Total power	Overall HRV

HRV, heart rate variability; SDNN, standard deviation of the R-to-R peak (RR) intervals; SDANN, standard deviation of the 5-minute mean RR intervals; RMSSD, square root of the mean squared differences between adjacent RR intervals; pRR50, percentage of interval differences of adjacent RR intervals greater than 50 ms; TIRRI, triangular interpolation of RR interval histogram, VLFP, very low frequency power; HFP, high frequency power

3.3 Effects of age, gender and aerobic fitness on HRV

Several studies have indicated that HRV decreases during adult life associated with physiological aging (Carter et al. 2003a). Reduction in vagal control of HR at rest, observed as a decrease in HFP, may be due to a decrease in physical fitness with age (Goldsmith et al. 2000; Ingram 2000) or decline in autonomic modulation in general (McNarry & Lewis 2012). A similar decrease has also been reported in LFP in elderly individuals linked to a reduction in cardiac responsiveness to sympathetic activity

(Lipsitz et al. 1990). However, possible changes in cardiac autonomic modulation with aging do not apparently affect resting HR of elderly individuals (Ryan et al. 1994). On the other hand, many researchers suggest that endurance training, described deeply in the following main chapter, will increase HRV, particularly vagal activity, and resting bradycardia also in the older individuals (Menard & Stanish 1989; Gregoire et al. 1996; Davy et al. 1998; Banach et al. 2000). Thus, it can be hypothesised that the decline in HRV indices associated with aging may be partly due to sedentary lifestyle (Yataco et al. 1997).

Gender might have an effect on HRV, although the differences between genders may depend on age and measured HRV index (Carter et al. 2003a). The findings related to gender difference in HRV are not entirely comparable because of heterogeneous group of subjects (age, training level and aerobic fitness), different duration of HRV recordings and analysing methods (Ryan et al. 1994; Gregoire et al. 1996; Ramaekers et al. 1998; Kuo et al. 1999; Hedelin et al. 2000b; Barantke et al. 2008). Nevertheless, it can be cautiously concluded that men show higher HRV indices than women younger than 40 (or 50) years of age. Some researchers suggest, however, that young women have greater vagal activity than men showing higher HFP (Kuo et al. 1999; Hedelin et al. 2000b). Kuo et al. (1999) hypothesise that gender difference of vagal activity will disappear over the menopause. The effect of the menstrual cycle should not be ignored as has been done in the most HRV studies (Aubert et al. 2003).

Researchers have been aware of the effect of aerobic fitness on HRV and cardiac autonomic control for a long time (e.g. Yataco et al. 1997). More and more studies have been published afterwards revealing that cardiac vagal modulation of HR at rest is higher in trained than in sedentary individuals (e.g. Buchheit & Gindre 2006; Hautala et al. 2009; Buchheit et al. 2010). In the other words, the cardiac vagal activity is greater in individuals having high aerobic capacity. A similar difference has also been shown in elderly athletes and an age-matched sedentary population (e.g. Banach et al. 2000). However, there exist few studies that could not have shown any correlation between aerobic capacity and vagal activity in athletes of a different aerobic fitness level (e.g. Tonkins 1999). These kinds of findings might be explicable in large individual variation of HRV (Hautala et al. 2009) not forgetting the inherited factors (Singh et al. 1999).

4 CHANGES IN HRV AND ENDURANCE PERFORMANCE INDUCED BY AEROBIC ENDURANCE TRAINING

4.1 Improvements in aerobic endurance performance

Endurance training induces several physiological adaptations that are reflected in improvements of aerobic fitness. According to the identification by Whipp et al. (1982) aerobic fitness can be divided into several components: the maximal oxygen uptake (VO_{2max}), exercise economy, the lactate or ventilatory threshold and oxygen uptake kinetics. The magnitude of training response to the components of aerobic fitness depends on the duration, intensity and frequency of the exercise bouts (Wenger & Bell 1986). Additionally, genetics, age, gender, nutrition, prior training, level of aerobic fitness, sleep, rest and stress has been proposed to result in large variation in the adaptation to endurance training (Bouchard & Rankinen 2001; Hedelin et al. 2001; Hautala et al. 2003, 2009; Buchheit et al. 2010; Nummela et al. 2010). Interestingly, age, gender, race and level of aerobic fitness together explain only 11 % of the variance in the adaptation to standardized endurance training (Bouchard & Rankinen 2001).

Mean improvements in VO_{2max} following 6–28 weeks of endurance training has been within 5–23 % of baseline values (Billat et al. 1999; Bouchard & Rankinen 2001; Hautala et al. 2003, 2009; Vollaard et al. 2009; Buchheit et al. 2010; Vesterinen et al. 2013). However, individual adaptations vary between negative values to as much as over 40 % improvement (Hautala et al. 2003; Vesterinen et al. 2013). The training intensity and volume play a key role when discussed the improvements in VO_{2max}. It is believed that the high-intensity interval training elicits greater improvements in maximal endurance performance than the continuous submaximal endurance training despite the baseline level of aerobic fitness (Laursen & Jenkins 2002). However, an additional increase in submaximal training volume may compensate the effect of relatively low training intensity and lead to significant improvements in VO_{2max} similar to the high-intensity interval training (Ingham et al. 2008). On the other hand, in the light of the current researches it appears that athletes having VO_{2max} greater than 60 ml/kg/min can achieve further improvements through high-intensity interval training only (Londeree 1997).

The mean improvements in the other components of aerobic fitness range within 3–6 % for exercise economy (Franch et al. 1998; Saunders et al. 2006; Vesterinen et al. 2013) and 10–16 % for lactate threshold (Helgerud et al. 2001; Esfarjani & Laursen 2007) in recreational athletes with large individual variation. Instead, elite endurance athletes have much smaller improvements in aerobic fitness than listed above (Laursen & Jenkins 2002); even 1 % improvement in VO_{2max} may, however, be the determining factor in the competitive performance (Hopkins 2005). In the future, the understanding of the mechanisms behind the individual adaptation to endurance training may develop new tools to guide endurance training with optimum training load of each individual.

4.2   Endurance training-induced changes in HRV at rest

Numerous longitudinal studies have shown that long-term endurance training induces several cardiac autonomic adaptations that can be obtained with changes in HRV indices. An increase in absolute HFP with a decrease in resting HR after endurance training period has been observed by several researchers (Figure 6) (Hautala et al. 2009), although contradictory findings exist as well. However, it can be hypothesised that endurance training increases vagal control of HR regardless of age, gender or aerobic fitness. The changes in the absolute level of LFP are more inconsistent and, therefore, any conclusions cannot be made. Some studies have reported increased absolute LFP in sedentary subjects (Tulppo et al. 2003; Hautala et al. 2004; Pichot et al. 2005), whereas some studies have found no changes in absolute LFP (Loimaala et al. 2000; Carter et al. 2003b; Vesterinen et al. 2013).
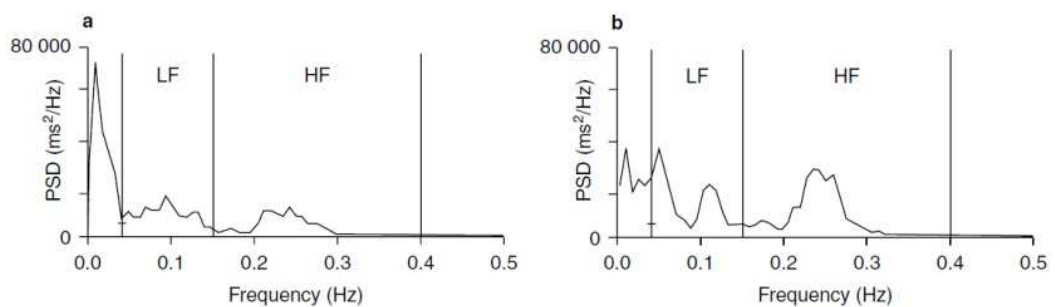

FIGURE 6. Power spectral density (PSD) of a recording in a young sedentary individual before training, high frequency power (HFP) 812.3 ms^2, (a) and after a 6-month aerobic training, HFP 1878.4 ms^2 (b). LF, low frequency; HF, high frequency. Modified from Aubert et al. (2003).

The conflicting results of endurance training-induced changes in sympathetic and vagal control of HR are possibly due to the differences in the duration and intensity of the training periods as well as the frequency of training between studies (Hautala et al. 2009). The duration of training interventions used in the studies varies typically from 4 weeks (e.g. Kiviniemi et al. 2007) to 6 moths (e.g. Levy et al. 1998). The changes in the ANS and electrophysiology of the heart take time from weeks to months, like many other physiological changes too. However, even the short-term (6 weeks) endurance training has been demonstrated to increase vagal activity of the heart (Yamamoto et al. 2001). On the other hand, it has been suggested by Loimaala et al. (2000) that the duration of endurance training interventions should be extended up to years to induce changes observed in HRV, at least among middle-aged population. Afterwards, Iwasaki et al. (2003) have observed that HRV increased only during the first 3 months of endurance training in sedentary subjects, although the endurance performance was improved over a whole 1-year progressively loaded training period (Figure 7). In consequence of that, a long duration of endurance training may not necessarily lead to greater enhancement in HRV. Finally, a recent meta-analysis reveals that an increase in HFP, and therefore in vagal activity, during short-term (4 weeks) endurance training interventions is influenced by the young age of the study population instead of endurance training itself (Sandercock et al. 2005).
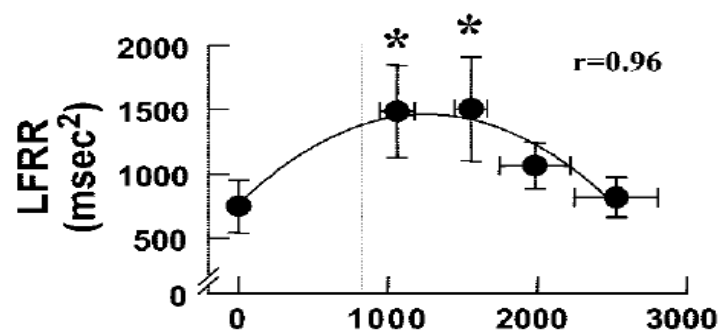
FIGURE 7. A dose-response relationship between exercise intensity (monthly training impulse) and low frequency power (LFRR) in sedentary subjects over a 1-year progressively loaded training period. Resting heart rate variability levels were measured at baseline and months 3, 6, 9 and 12. * $p < 0.05$ compared with the pre-training baseline. Modified from Iwasaki et al. (2003)

In the previous studies, the intensity of endurance training has mainly been limited to moderate or vigorous (e.g. Levy et al. 1998; Loimaala et al. 2000; Kiviniemi et al. 2007) yet Achten and Jeukendrup (2003) have suggested that at least vigorous training

intensity is needed to induce changes in HRV. For example, Loimaala et al. (2000) had an effort to determine whether training intensity would affect HRV. Two groups of middle-aged men trained with the intensities either at 55 % or 75 % of VO_{2max} for 20 weeks. No differences were found in any of the time domain or spectral HRV indices in either of the training intensity groups (Loimaala et al. 2000). Similarly, Vesterinen et al. (2013) found no differences in HRV among recreational endurance runners during the 14-week basic training period when training was performed primarily below the intensity of the aerobic threshold. However, an increase in HFP and TP with a decrease in HR was found after the 14-week intensive training period when training intensity was significantly increased (Vesterinen et al. 2013). This finding by Vesterinen et al. (2013) supports the hypothesis suggested by Achten and Jeukendrup (2003) that vigorous training intensity is needed to induce changes in HRV.

The role of the frequency of training still remains unclear. Perhaps the training volume has a more important role to changes in HRV indices induced by endurance training than the frequency of training (Achten & Jeukendrup 2003). Increases in training volume have been observed to elicit significant changes in HRV indices among both sedentary (Iwasaki et al. 2003) and trained individuals (Buchheit et al. 2004; Manzi et al. 2009). However, too excessive increases (over 50 %) in training volume may rather decrease the absolute level of HRV indices (Iwasaki et al. 2003). In addition, age may also affect the magnitude of endurance training-induced changes in HRV; there are indications that young individuals present greater changes in HRV compared to older individuals (Mourot et al. 2004; Hynynen et al. 2010; Tulppo et al. 2011). It should not also be forgotten that HRV decreases as a result of acute exercise (Aubert et al. 2003). For example, nocturnal HRV can be reduced with raised HR after moderate or vigorous endurance exercise (Mourot et al. 2004) being not a chronic adaptation, however. On the other hand, prolonged and intensive endurance training without adequate recovery may lead to the state of over-reaching, or even overtraining syndrome, that can be observed in HRV indices (Halson & Jeukendrup 2004).

5 AIM OF THE STUDY

The aim of this study was to examine the effects of high-intensity and high-volume endurance training on nocturnal HRV indices and endurance performance in recreational endurance runners. The primary focus was to examine the changes in basal nocturnal HRV indices and endurance running performance as a response to endurance training and to assess possible relationships between HRV indices and endurance performance. The secondary focus was to examine possible differences in individual vagal-related HRV profiles between training responders and non-responders.

Research problems.

1) Does the combined 16-week basic and hard training intervention induce changes in basal nocturnal HRV indices?

2) Does the combined training intervention induce changes in endurance performance?

3) Are there differences in changes of basal nocturnal HRV indices or endurance training adaptation between the high-intensity and high-volume training groups during the hard training period?

4) Are there relationships between HRV indices and endurance training adaptation?

5) Are there differences in individual vagal-related HRV profiles between the responders and non-responders within the training groups?

It is hypothesised that the combined basic and hard training intervention increase basal HRV indices in recreational endurance runners together with improved endurance running performance, based on the findings of Buchheit et al. (2010) and Vesterinen et al. (2013). High-intensity endurance training may elicit greater endurance training adaptation compared with high-volume endurance training (Laursen & Jenkins 2002). In addition, positive endurance training adaptation is expected to be associated with high HRV values at baseline (Hautala et al. 2003; Vesterinen et al. 2013) and great increases in HRV indices (Buchheit et al. 2010). The fifth research problem tries to describe qualitatively the differences in HRV profiles and to explain why the other individuals improve their endurance running performance during the training periods while the other individuals possibly do not improve.

6 METHODS

6.1 Subjects

In total, forty male (n=20) and female (n=20) recreational endurance runners were recruited to the study. All subjects were healthy, non-smokers, non-obese (BMI <30 kg/m^2), free of any diseases and regular medication. Resting electrocardiography (Cardiofax ECG–9320, Tokyo, Japan) was measured and analysed to ensure that subjects had no cardiac abnormalities, which would have affected the HRV analysis or prevent from endurance training. All subjects were fully informed of the procedures, possible risks and benefits of the study and they signed an informed consent document. Five subjects dropped out due to lack of motivation or injury and two subjects were excluded because of insufficient compliance with the training during the study. Furthermore, five subjects were excluded from HRV analyses due to erroneous RR interval recordings. Finally, the results of 28 subjects (14 men and 14 women) were available for the final analyses. Anthropometric characteristics of the subjects are presented in Table 2. According to the questionnaire, subjects had trained on average 5.0 ± 1.9 times and 6.6 ± 2.8 hours per week prior to the study. The endurance training background of the subjects was on average 14 years and varied from 2 to 30 years. The study was approved by the Ethics Committee of the University of Jyväskylä, Finland.

TABLE 2. Anthropometric characteristics of the subjects. Values are means ± SD.

n	Age (year)	Height (m)	Weight (kg)	BMI (kg/m^2)	Body fat %
28	34.7 ± 7.6	1.70 ± 0.08	69.3 ± 11.5	23.9 ± 2.3	20.4 ± 6.0

SD, standard deviation; BMI, body mass index

6.2 Experimental design and training

The 16-week training intervention was divided into an 8-week basic training period (BTP) and 8-week hard training period (HTP) separated by the measurement week (Figure 8). All subjects performed the BTP as one group doing the same training pro-

gram. After the BTP, subjects were separated into two training groups, high-intensity training (HIT, n=14) and high-volume training (HVT, n=14) group. The training groups were matched for gender, endurance performance, improved endurance performance during the BTP and basal HRV indices. An incremental treadmill test was performed prior to the BTP, between the training periods and after the HTP to measure endurance performance characteristics. The subjects were asked to avoid strenuous physical exercise during the preceding two days of the test. All the tests were performed during daytime between 8am and 4pm.
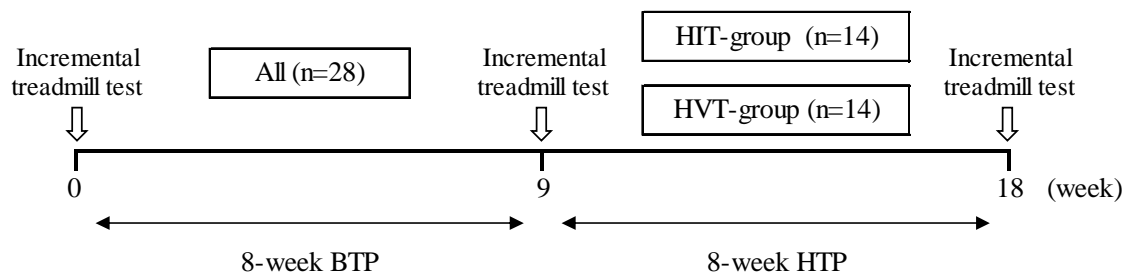

FIGURE 8. The experimental design. BTP, basic training period; HTP, hard training period; HIT, high-intensity training; HVT, high-volume training.

As the secondary focus of the present study was to examine individual responses to endurance training, subjects were separated into subgroups, *post hoc*, within their respective training groups. The subjects who improved their maximal running velocity during the HTP more than 2 % were included in the training responders subgroup and the subjects who did not improve or improved less than 2 % were included in the non-responders subgroup. A 2 % cut-off point should correspond to the typical variation in running performance of recreational athletes since the typical variation of distance runners is ~1.5 % in 3000–10 000 m running events (Hopkins 2005). In the HIT group, eight subjects out of 14 were classed as responders and the rest six subjects as non-responders, whereas in the HVT group, four subjects out of 14 and the rest ten subjects were classed as responders and non-responders, respectively. *Post hoc* separation into the responders and non-responders subgroups was based on maximal running velocity because it has been shown to be related to endurance performance better than VO_{2max} (Paavolainen et al. 1999).

The outline of the 16-week training program is presented in Table 3. During the 8-week BTP the subjects were asked to complete three to six endurance training sessions per

week according to the individual training volume prior the study. Endurance training consisted mostly of running but occasionally included also cycling, Nordic walking and/or cross country skiing. In addition, the subjects were asked to perform one circuit training session per week. During the BTP training intensity was asked to be kept primarily below the individually determined aerobic threshold (Aunola & Rusko 1986). During the following 8-week HTP either the training intensity (HIT) or training volume (HVT) was increased compared to the BTP. The subjects of the HIT group were asked to replace three low-intensity (below aerobic threshold) training sessions with one moderate-intensity (between aerobic and anaerobic thresholds, Aunola & Rusko [1986]) continuous (20–40 min) and two high-intensity (above anaerobic threshold) interval training sessions (4 x 4 min at 90 % V_{peak} with 3 min recovery and 6 x 2 min at 100 % V_{peak} with 2 min recovery) per week. In the HVT group, the subjects were asked to prolong the duration of their running training sessions by 30–50 % and maintain the training intensity primarily below aerobic threshold. The 16-week training program was periodized to cycles of four weeks; three weeks of hard training was followed by an easy training week when the subjects were asked to train only at low-intensity.

TABLE 3. The outline of the 16-week training program.

| | BTP (weeks 1–8) | HTP (weeks 9–16) | |
		HIT-group	HVT-group
Training sessions			
High-intensity	None	2 sessions *	None
Moderate-intensity	1–2 sessions *	1 session *	1–2 sessions *
Long low-intensity	1 session	None	2 sessions
Basic low-intensity	1–3 sessions	1–3 sessions	1–3 sessions
Circuit training	1 session	1 session	1 session

* Exercises were not performed during recovery weeks
BTP, basic training period; HTP, hard training period; HIT group, high-intensity
training group; HVT group, high-volume training group

The subjects controlled their training intensity by measuring their HR during all exercises using a HR monitor (Garmin Forerunner® 610, Garmin Ltd.) equipped with the integrated GPS sensor to measure running distance. All the subjects were required to keep a training diary throughout the study to record training mode, duration of the training session, average HR and running distance of each training session. Exercise RR interval data was used to determine the durations at three different intensity zones; low-,

moderate- and high-intensities. A novel approach to the traditional training impulse (TRIMP) method was used to estimate the total training load (i.e. intensity × volume) of the subjects by using the following formula introduced by Foster et al. (2001):

$$TRIMP = 1 \times t_1 + 2 \times t_2 + 3 \times t_3$$

where t_1 is a duration in the low-intensity zone, t_2 is a duration in the moderate-intensity zone and t_3 is a duration in the high-intensity zone.

6.3 Procedures

Anthropometry. All anthropometric measurements were performed before and after both training periods prior the incremental treadmill test. In addition to height, body mass was measured using a calibrated digital scale. Body fat % was determined using skin-fold thickness from four different skin folds (subscapular, biceps brachii, triceps brachii and iliac crest) (Durnin & Womersley 1974).

Incremental treadmill test. The initial velocity of 7 km/h (women) or 8 km/h (men) was used in the incremental treadmill test with the 0.5° incline. Thereafter, velocity was increased by 1 km/h every third minute until voluntary exhaustion. Oxygen uptake was measured breath-by-breath using a portable gas analyser (Oxycon Mobile®, Jaeger, Hoechberg, Germany). Also HR was measured continuously using the HR monitor (Suunto t6, Suunto Ltd., Finland). Fingertip blood sample (20 μl) was taken at the end of each 3-min stage for blood lactate analysis (Biosen S_line Lab+, EKF Diagnostic GmbH, Magdeburg, Germany). The highest 60-s VO_2 value was considered as maximal oxygen uptake (VO_{2max}). The maximal running velocity (V_{peak}) was determined as the highest velocity of the test. If the subject could not complete the 3-min stage of the last velocity, the V_{peak} was calculated as follows: the last completed velocity (km/h) + [duration of the last uncompleted velocity (s) – 30 s / 150 s] x 1 km/h. Aerobic (AerT) and anaerobic (AnT) thresholds were determined using blood lactate, ventilation, oxygen uptake and production of carbon dioxide according to Aunola & Rusko (1986).

Nocturnal HRV analysis. Subjects were asked to measure nocturnal RR intervals during four consecutive nights per week throughout the training periods as well as measure-

ment weeks. A HR monitor (Garmin Forerunner® 610, Garmin Ltd., Great Britain) was used to record RR intervals with a sampling frequency of 1000 Hz. The recordings were started before retiring to bed and stopped after waking up in the morning. The first 30 min of recording was excluded and the following continuous four hour period was accepted for the analysis if the imposed cut-off of the erroneous RR intervals was lower than 33%. The acceptable RR interval data was processed and analysed using the Firstbeat PRO heartbeat analysis software (version 2.0.0.9, Firstbeat Technologies Ltd., Jyväskylä, Finland). RR interval recordings were first scanned through an artefact detection filter of the Firstbeat PRO software to exclude all falsely detected, missed and premature heart beats (Saalasti 2003). The consecutive artefact corrected RR intervals were then re-sampled at the rate of 5 Hz by using linear interpolation to obtain equidistantly sampled time series. From the re-sampled data, the software calculated HRV indices second-by-second using the short-time Fourier Transform method. For a given segment of data, a Hanning time window with a length of 256 samples was applied, and last Fourier transform was calculated and power spectrum was obtained. Thereafter, the window was shifted one sample to another and the same process was repeated. Low frequency power (LFP; 0.04–0.15 Hz) and high frequency power (HFP; 0.15–0.40 Hz) were calculated as integrals of the respective power density curve. Total power was determined as the sum of low and high frequency power (TP = LFP + HFP). In addition, average HR, standard deviation of RR intervals (SDNN) and root mean square of differences between adjacent RR intervals (RMSSD) were analysed with time domain methods. Basal HRV indices were provided as averages of two nights after a light training day according to TRIMP.

6.4 Statistical analyses

The values are presented as means ± standard deviations (SD). The normal distribution of the data was assessed with the Shapiro-Wilk goodness-of-fit test. Due to skewness, ln-transformation was used with the spectral HRV indices in order to meet the assumptions of the parametric statistical analysis. Changes in endurance performance and HRV indices as a result of the present training were first analysed using repeated-measures analysis of variance (ANOVA), followed by paired samples t-test within group and in-

dependent samples t-test between groups. Pearson's product moment correlation coefficient was used to determine the relationships between HRV indices and endurance performance. In addition to the measures of statistical significance, the following criteria were adopted to interpret the magnitude of the correlation (r): <0.1, trivial; 0.1–0.3, small; 0.3–0.5 moderate; 0.5–0.7, large; 0.7–0.9, very large; and 0.9–1.0, almost perfect. The data was analysed using SPSS software (PASW Statistics 20.0; SPSS Inc., Chicago, Illinois). The statistical significance was accepted as $p < 0.05$.

Additionally, the data of endurance performance and HRV indices was assessed for significance using an approach based on the magnitudes of change (Hopkins et al. 2009). At first, the magnitude of change after training or difference between the groups was expressed as standardized mean differences (Cohen's effect sizes, ES) calculated using the pooled standard deviations (Cohen 1988). Threshold values for Cohen's ES statistics were <0.2 (small), 0.5 (moderate) and >0.8 (large). 90% confidence intervals (CI) for the (true) mean changes or between-group differences in the training response were estimated (Hopkins et al. 2009). For within- and between-group comparisons, the percentual chances that the (true) changes in performance or HRV indices were greater (i.e., greater than the smallest worthwhile change, SWC [0.2 multiplied by the pooled standard deviation, based on Cohen's ES principle], unclear or smaller) than these changes in compared group were calculated. Quantitative chances of higher or smaller training effects were assessed qualitatively as follows: <1%, almost certainly not; 1–5%, very unlikely; 5–25%, unlikely; 25–75%, possibly; 75–95%, likely; 95–99%, very likely; and >99% almost certain. The true difference was assessed as unclear, if the chance of having better or poorer performances, as well as HRV indices, were both >5% (Hopkins et al. 2009).

7 RESULTS

Training load. The training data of the training periods is summarized in Table 4. The training- and running volume during the initial 8-week BTP did not differ from the training prior to the study whereas training frequency (p=0.034) was increased. The running volume (p<0.001) and percentage of training duration at the high-intensity zone (p=0.008) were increased during the following 8-week HTP compared to the BTP in the HVT and HIT group, respectively. No other significant differences were observed in the training data between the two training periods. Greater total training (p=0.005) and endurance training volumes (p=0.016) as well as TRIMP (p=0.003) were observed in the HVT group compared to the HIT group during the HTP. However, the percentage of training duration at the high-intensity zone (p=0.044) was greater in the HIT group compared to the HVT group.

TABLE 4. Training data of the groups during the training periods. Values are means ± SD.

	Prior to study	BTP	HTP	
			HIT-group	HVT-group
Training volume (h/week)	6.6 ± 2.8	6.8 ± 2.1	5.5 ± 1.7	7.2 ± 1.2 [&&]
Training frequency (times/week)	5.0 ± 1.9	5.6 ± 1.4 *	5.8 ± 2.2	5.8 ± 0.7
TRIMP (a week)		463 ± 137	395 ± 104	508 ± 76 [&&]
HR below AerT (%)		86 ± 8	82 ± 10	83 ± 10
HR between AerT and AnT (%)		13 ± 1	13 ± 6	15 ± 10
HR above AnT (%)		1 ± 1	4 ± 4 [##]	1 ± 2 [&]
Running volume (km/week)	29 ± 17	32 ± 16	37 ± 17	44 ± 11 [###]
Endurance training volume (h/week)		5.6 ± 1.8	4.8 ± 1.5	6.2 ± 1.4 [&]

* p<0.05 (significant difference from value prior to study)
[#] p<0.05, [##] p<0.01, [###] p<0.001 (significant difference between BTP and HTP)
[&] p<0.05, [&&] p<0.01 (significant difference between the training groups)
SD, standard deviation; BTP, basic training period; HTP, hard training period;
HIT, high-intensity training; HVT, high-volume training; TRIMP, training impulse;
HR, heart rate; AerT, aerobic threshold; AnT, anaerobic threshold

Anthropometrics. Body mass after the BTP was significantly smaller compared to the baseline level (69.3 ± 11.5 kg vs. 68.2 ± 11.0 kg, p=0.001). Also body fat % was lower

after the BTP compared to the baseline level (20.4 ± 6.0 % vs. 19.4 ± 6.1 %, p<0.001). During the following HTP body mass and body fat % decreased only in the HVT group (-0.9 ± 0.4 kg, p=0.038 and -0.7 ± 0.4 %, p=0.027, respectively), although no significant differences between the training groups were observed.

Endurance performance. V_{peak} improved by 2.8 ± 2.9 % (p<0.001) during the 8-week BTP (Figure 9, Table 5). In addition, velocities at the anaerobic (V_{AnT}) and aerobic (V_{AerT}) thresholds increased by 4.2 ± 3.9 % (p<0.001) and 5.5 ± 4.8 % (p<0.001), respectively, whereas VO_{2max} did not improve during the BTP. No significant gender differences were observed in the changes in any of the endurance performance characteristics. However, great individual heterogeneity of training adaptation was observed, especially in VO_{2max} (Figure 9).
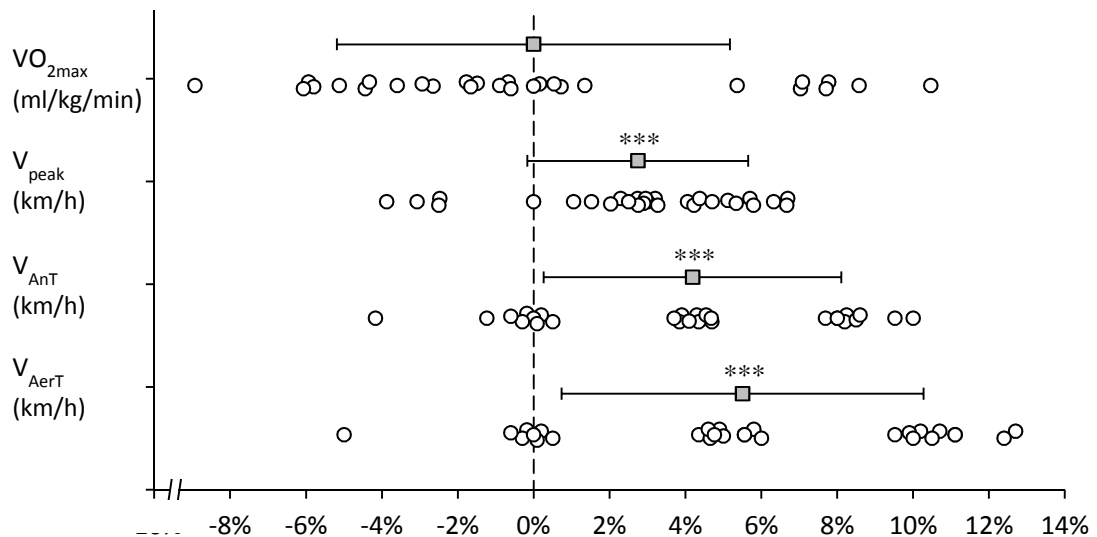

FIGURE 9. The percentual changes in endurance performance characteristics during the 8-week basic training period. *Squares* are means ± standard deviations. *Circles* represent individuals. *** p<0.001 significant change from the baseline level. The zero line is shown with *broken line*. VO_{2max}, maximal oxygen uptake; V_{peak}, maximal running velocity; V_{AnT}, running velocity at anaerobic threshold; V_{AerT}, running velocity at aerobic threshold.

During the HTP, VO_{2max} (3.7 ± 4.2 %; p=0.005), V_{peak} (2.4 ± 2.3 %; p=0.002), V_{AnT} (3.8 ± 4.4 %; p=0.005) and V_{AerT} (2.7 ± 3.7 %; p=0.020) were significantly increased in the HIT group, whereas in the HVT group a significant increase was observed in V_{AerT} (1.4 ± 2.2 %, p=0.040) only (Table 5). No significant differences were found in the changes in endurance performance characteristics between the training groups or gen-

ders. However, the qualitative analysis based on the magnitudes of change revealed that the HIT group improved V_{peak} and V_{AnT} more than the HVT group. The true improvements of the HIT group were greater, trivial or smaller with percentual chances of 60/40/0 % and 72/28/0 %, respectively (see Table 5 for details, Figure 10).
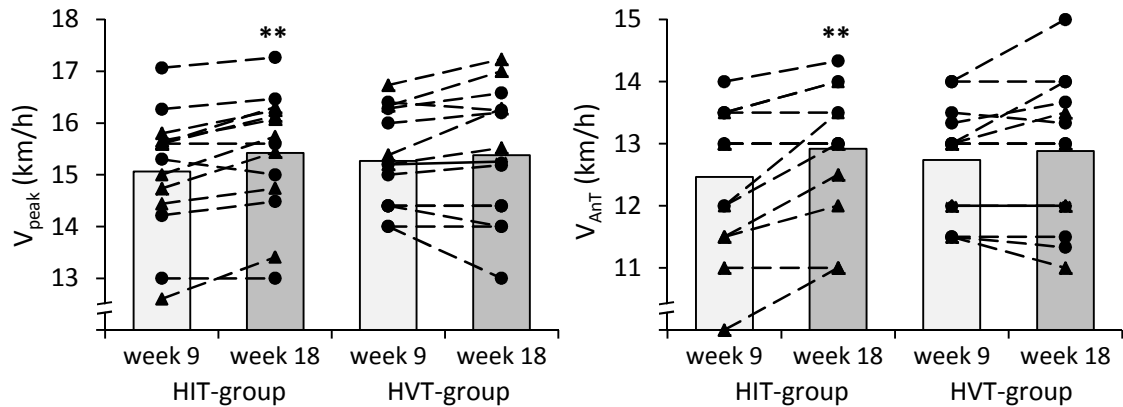

FIGURE 10. The absolute changes in V_{peak} (left) and V_{AnT} (right) during the hard training period in the HIT and HVT groups. *Bars* are means. *Triangles* (responders) and *circles* (non-responders) represent individuals. ** $p<0.01$ significant difference from week 9 within training group. V_{peak}, maximal running velocity; V_{AnT}, running velocity at anaerobic threshold; HIT, high-intensity training; HVT, high-volume training.

Basal HRV indices. RMSSD ($p=0.038$) was slightly decreased after the 8-week BTP compared with the baseline level. However, no significant changes were observed in nocturnal HR or other HRV indices during the BTP. After the following 8-week HTP, SDNN ($p=0.005$), RMSSD ($p=0.034$) and ln TP ($p=0.040$) were increased significantly in the HIT group compared with the level prior to ITP, whereas no significant changes were observed in the HVT group (Table 6). No significant differences were observed in the basal levels or the changes in HRV indices between the training groups. However, it was found by the qualitative analysis that the true changes in all HRV indices were greater in the HIT group compared with the HVT group (see Table 6 for details).

Table 7 summarizes the HRV indices of the *post hoc* subgroups within their respective training groups during the 8-week HTP. In the HIT group, the responders showed lower SDNN values prior to the HTP compared with the non-responders (ES=0.7). Nocturnal HR ($p=0.046$) decreased and SDNN ($p=0.003$) and RMSSD ($p=0.026$) increased during the HTP in the responders subgroup. These results were confirmed by the consistent

TABLE 5. Endurance performance characteristics at baseline, week 9 (after the BTP) and 18 (after the HTP). Values are means ± standard deviations.

	VO_{2max} (ml/kg/min)	V_{peak} (km/h)	V_{AnT} (km/h)	V_{AerT} (km/h)
All (*n=28*) – BTP				
Baseline	49.6 ± 5.3	14.8 ± 1.1	12.1 ± 1.0	9.6 ± 0.9
Week 9	49.5 ± 5.1	15.2 ± 1.1 ***	12.6 ± 1.0 ***	10.1 ± 0.9 ***
ES (rating)	0.0 (trivial)	0.4 (small)	0.5 (moderate)	0.6 (moderate)
HIT-group (*n=14*) – HTP				
Week 9	49.8 ± 6.2	15.1 ± 1.2	12.5 ± 1.2	10.1 ± 1.0
Week 18	51.6 ± 6.3 **	15.4 ± 1.3 **	12.9 ± 1.0 **	10.4 ± 0.9 *
ES (rating)	0.3 (small)	0.3 (small)	0.4 (small)	0.3 (small)
HVT-group (*n=14*) – HTP				
Week 9	49.2 ± 3.8	15.3 ± 0.9	12.7 ± 0.9	10.1 ± 0.9
Week 18	50.7 ± 4.8	15.4 ± 1.3	12.9 ± 1.2	10.3 ± 1.0 *
ES (rating)	0.3 (small)	0.1 (trivial)	0.1 (trivial)	0.1 (trivial)
Magnitude of between-groups differences prior to HTP				
ES (rating)	0.1 (trivial)	– 0.2 (small)	– 0.3 (small)	0.0 (trivial)
Magnitude of between-groups differences in responses to training during HTP				
ES (rating)	0.1 (trivial)	0.6 (moderate)	0.7 (moderate)	0.4 (small)
Mean difference (90% CI)	0.3 (–1.4;2.0)	0.3 (0.0;0.5)	0.3 (0.0;0.6)	0.1 (–0.1;0.3)
% chances of true value being better/trivial/poorer	23/67/10	60/40/0	72/28/0	27/72/1
Outcome	Unclear	Possibly	Possibly	Possibly trivial

* $p<0.05$, ** $p<0.01$, *** $p<0.001$ (significant difference from baseline or week 9)

ES, effect size (qualitative classification based on threshold values by Cohen [1988])

Magnitude of between-groups differences in responses to training are expressed as percentual chances and qualitative outcome for

HIT group to have better/trivial/poorer responses than HVT group (see "Methods" for thresholds of percentual chance used)

BTP, basic training period; HTP, hard training period; VO_{2max}, maximal oxygen uptake; V_{peak}, maximal running velocity; V_{AnT}, running velocity

at anaerobic threshold; V_{AerT}, running velocity at aerobic threshold; HIT, high-intensity training; HVT, high-volume training; CI, confidence interval

TABLE 6. Nocturnal HRV indices of the training groups at week 9 and 18 (before and after the HTP, respectively). Values are means ± SD.

	HR (bpm)	SDNN (ms)	RMSSD (ms)	ln LFP (ms^2)	ln HFP (ms^2)	ln TP (ms^2)
HIT-group (*n=14*)						
Week 9	53.0 ± 8.1	114 ± 28	64 ± 25	8.10 ± 0.59	7.75 ± 0.83	8.67 ± 0.63
Week 18	51.0 ± 6.9	127 ± 28 **	71 ± 24 *	8.30 ± 0.67	7.98 ± 0.67	8.88 ± 0.60 *
ES (rating)	– 0.3 (small)	0.5 (moderate)	0.3 (small)	0.3 (small)	0.3 (small)	0.3 (small)
HVT-group (*n=14*)						
Week 9	52.2 ± 5.5	120 ± 25	71 ± 28	8.12 ± 0.63	8.15 ± 1.16	8.88 ± 0.82
Week 18	51.6 ± 5.2	124 ± 26	71 ± 28	8.08 ± 0.52	8.00 ± 0.88	8.79 ± 0.59
ES (rating)	– 0.1 (trivial)	0.2 (small)	0.0 (trivial)	– 0.1 (trivial)	– 0.1 (trivial)	– 0.1 (trivial)
Magnitude of between-groups differences prior to HTP						
ES (rating)	0.1 (trivial)	– 0.2 (small)	– 0.3 (small)	0.0 (trivial)	– 0.4 (small)	– 0.3 (small)
Magnitude of between-groups differences in responses to training						
ES (rating)	– 0.4 (moderate)	0.8 (large)	0.6 (moderate)	0.6 (moderate)	0.8 (large)	0.8 (large)
Mean difference (90% CI)	– 1.5 (–4.4;1.3)	11.5 (1.0;22.0)	8.4 (–1.4;18.2)	0.23 (–0.04;0.50)	0.37 (0.05;0.69)	0.30 (0.03;0.57)
% chances of true value being better/trivial/poorer	4/42/54	84/15/1	71/18/1	76/22/1	82/18/0	85/15/0
Outcome	Possibly	Likely	Possibly	Possibly	Likely	Likely

* p<0.05, ** p<0.01 (significant difference from week 9)

ES, effect size (qualitative classification based on threshold values by Cohen [1988])

Magnitude of between-groups differences in responses to training are expressed as percentual chances and qualitative outcome for

HIT group to have better/trivial/poorer responses than HVT group (see "Methods" for thresholds of percentual chance used)

SD, standard deviation; HTP, hard training period; HR, heart rate; SDNN; standard deviation of R-to-R peak intervals; RMSSD, root mean square of the

differences between adjacent R-to-R peak intervals; ln LFP; natural logarithm of low frequency power; ln HFP, natural logarithm of high frequency power;

ln TP, natural logarithm of total power; HIT, high-intensity training; HVT, high-volume training; CI, confidence interval

qualitative analysis. Further, the responders presented greater change in SDNN (with the chances of 87/11/2 %) during the HTP than the non-responders (Table 7). In the HVT group, *post hoc* subgroups differed significantly in RMSSD (p=0.038) prior to the HTP; the non-responders showed higher RMSSD values. In addition, higher nocturnal HR and lower SDNN, ln HFP and ln TP values were observed among the responders compared with the non-responders (all ES≥0.8). According to the qualitative analysis ln HFP and ln TP decreased slightly during the HTP in the non-responders subgroup (with the chances of 63/36/1 % and 55/41/4 %, respectively).

Basal HRV indices and endurance performance. At baseline, VO_{2max} was significantly correlated with SDNN (r=0.46, p=0.015, n=28; Figure 11) and ln LFP (r=0.38, p=0.046, n=28). In addition, a significant correlation was observed between the change in V_{peak} during the BTP and ln LFP at baseline (r=–0.42, p=0.027, n=28) as well as the change in ln LFP during the BTP (r=0.38, p=0.049, n=28). Furthermore, significant correlations were observed between the change in V_{AnT} and the change in ln LFP and ln TP during the BTP [(r=0.60, p=0.001, n=28) and (r=0.51, p=0.005, n=28), respectively].
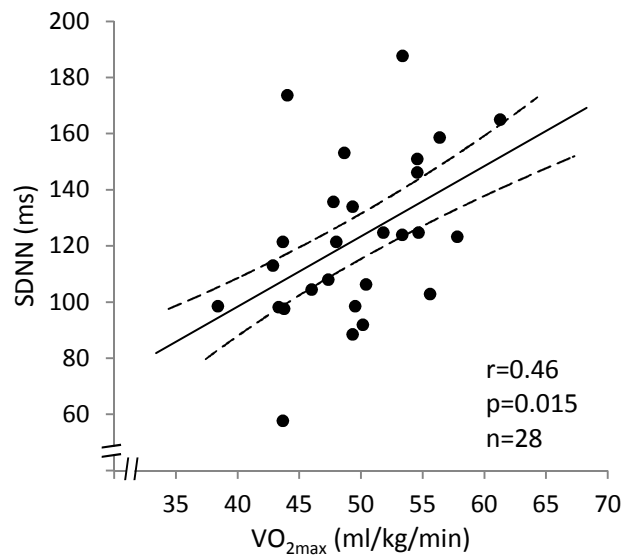

FIGURE 11. A correlation between baseline maximal oxygen uptake (VO_{2max}) and standard deviation of R-to-R peak intervals (SDNN) in all subjects. The regression line and the 90 % confidence intervals are shown with *continuous* and *broken lines*, respectively.

In the HIT group, the change in VO_{2max} during the HTP correlated significantly with the change in ln TP during the HTP (r=0.54, p=0.045, n=14), when the training groups were

examined separately. Similarly, a significant correlation was found between the change in V_{peak} and the change in SDNN (r=0.55, p=0.050, n=13; Figure 12). In the HVT group the changes in V_{peak} and V_{AnT} during the HTP were significantly correlated with the RMSSD basal level prior to the HTP [(r=–0.54, p=0.049, n=14; Figure 12) and (r=–0.67, p=0.009, n=14), respectively]. Also, a significant correlation between the change in V_{AerT} and nocturnal HR during the HTP (r=0.62, p=0.019, n=14) was found.
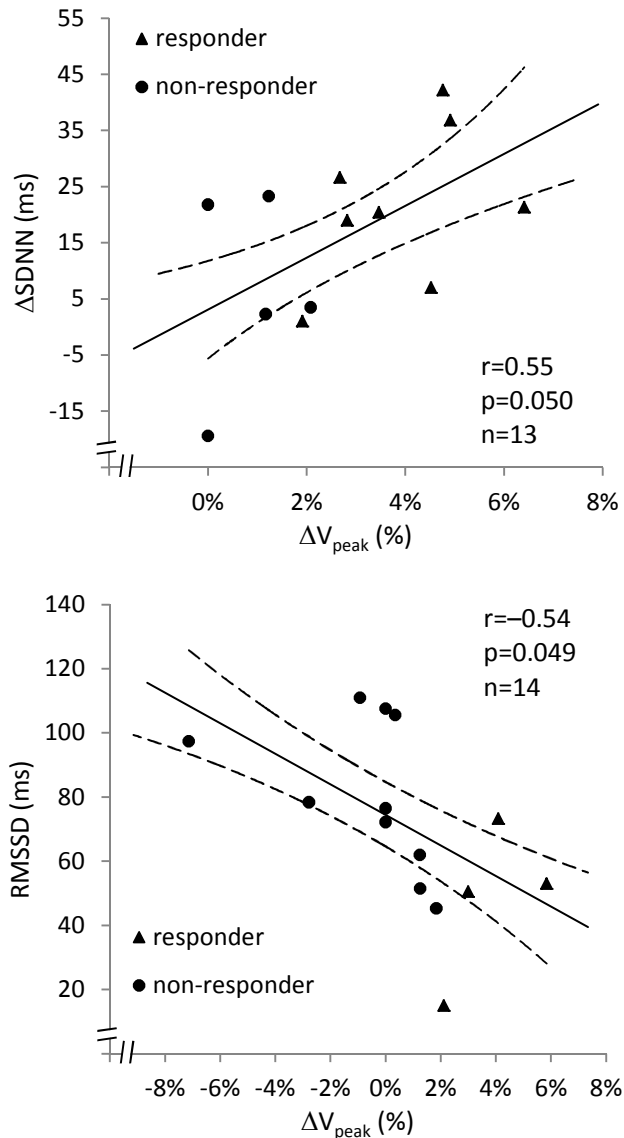
FIGURE 12. The correlations between the relative changes in maximal running velocity (ΔV_{peak}) and the absolute changes in standard deviation of R-to-R peak intervals (ΔSDNN) during the hard training period in the high-intensity training group (top) and the root mean square of the differences between adjacent R-to-R peak intervals (RMSSD) at week 9 in the high-volume training group (bottom). The regression line and the 90 % confidence intervals are shown with *continuous* and *broken lines*, respectively.

TABLE 7. Nocturnal HRV indices in the *post hoc* subgroups at week 9 and 18 (before and after the HTP, respectively). Values are means ± SD.

	HR (bpm)	SDNN (ms)	RMSSD (ms)	ln LFP (ms^2)	ln HFP (ms^2)	ln TP (ms^2)
HIT, responders (*n=8*)						
Week 9	53.6 ± 8.8	107 ± 26	63 ± 24	8.04 ± 0.60	7.75 ± 0.65	8.63 ± 0.54
Week 18	50.8 ± 8.3 *	129 ± 29 ** [a]	72 ± 25 *	8.20 ± 0.79	7.94 ± 0.64	8.81 ± 0.67
HIT, non-responders (*n=6*)						
Week 9	52.4 ± 7.9	124 ± 30 [b]	66 ± 29	8.18 ± 0.62	7.75 ± 1.10	8.72 ± 0.79
Week 18	51.2 ± 5.2	124 ± 27	68 ± 26	8.43 ± 0.51	8.03 ± 0.78	8.98 ± 0.53
Magnitude of between-groups differences in responses to training						
% chances of true value being better/trivial/poorer	11/35/54	87/11/2	37/48/15	18/39/43	14/54/32	14/45/41
Outcome	Unclear	Likely	Unclear	Unclear	Unclear	Unclear
HVT, responders (*n=4*)						
Week 9	55.0 ± 8.8	101 ± 19	48 ± 24	7.95 ± 1.10	7.33 ± 1.69	8.42 ± 1.26
Week 18	53.9 ± 4.3	111 ± 29	53 ± 22	8.03 ± 0.91	7.48 ± 1.36	8.52 ± 1.02
HVT, non-responders (*n=10*)						
Week 9	51.0 ± 3.6 [d]	127 ± 23 [d]	81 ± 24 [#] [d]	8.19 ± 0.40	8.47 ± 0.76 [d]	9.06 ± 0.55 [d]
Week 18	50.6 ± 5.4 [c]	130 ± 24 [e]	79 ± 27 [e]	8.10 ± 0.34	8.21 ± 0.58 [e]	8.90 ± 0.33 [c]
Magnitude of between-groups differences in responses to training						
% chances of true value being better/trivial/poorer	23/35/43	58/32/10	55/32/13	56/28/16	74/23/3	66/25/9
Outcome	Unclear	Unclear	Unclear	Unclear	Possibly	Unclear

* p<0.05, ** p<0.01 (significant difference from week 9), [#] p<0.05 (significant difference between *post hoc* subgroups)

[a] moderate effect size of change during the training period within *post hoc* subgroup

[b, c] moderate and [d, e] large effect size of difference between *post hoc* subgroups within respective training group prior to and after the HTP, respectively

Magnitude of between-groups differences in responses to training are expressed as percentual chances and qualitative outcome for

HIT group to have better/trivial/poorer responses than HVT group (see "Methods" for thresholds of percentual chance used)

SD, standard deviation; HTP, hard training period; HR, heart rate; SDNN; standard deviation of R-to-R peak intervals; RMSSD, root mean square of the differences between adjacent R-to-R peak intervals; ln LFP; natural logarithm of low frequency power; ln HFP, natural logarithm of high frequency power; ln TP, natural logarithm of total power; HIT, high-intensity training; HVT, high-volume training; CI, confidence interval

Individual HRV profiles. Eight representative subjects, four subjects from each training group, were selected for the further inspection of the HRV profiles at individual level. The individual HRV profiles in selected subjects of the HIT and HVT groups over the 8-week HTP are presented in figures 13 and 14, respectively. HRV profiles showed that the variation in vagal activity was individual; for example, subject #29 had less intra-individual variation than subject #7. In a group level, the HVT group showed trend to greater intra-individual variation compared to the HIT group (coefficient of variation being 41.5 ± 14.4 % vs. 29.3 ± 13.4 %, p=0.053).
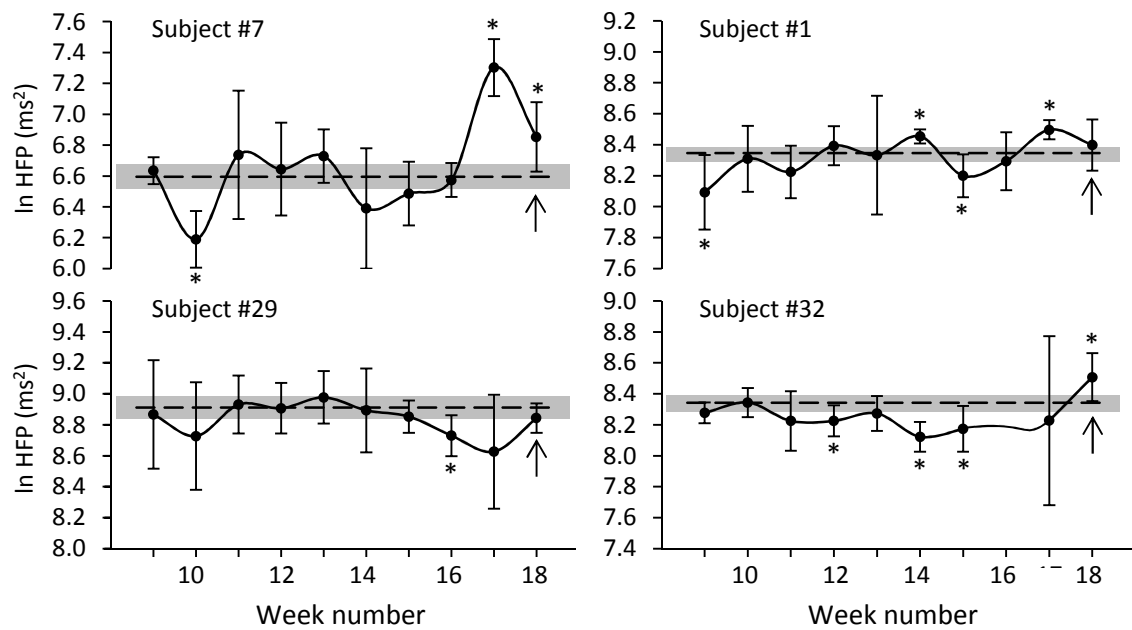

FIGURE 13. The nocturnal weekly averaged values of the natural logarithm of the high frequency power (ln HFP) with 90% confidence intervals over the 8-week hard training period in four subjects of the high-intensity training group. Subjects #7, #29 represent responders and subjects #1 and #32 non-responders. The *black circles* indicate the weekly averaged lnHFP value. The *arrows* indicate the final measurement week. The *grey shaded area* indicates the individual smallest worthwhile change (SWC) in ln HFP. * indicates a 'clear' change in weekly averaged ln HFP values above/below the zero line (the mean of ln HFP values during the basic training period) of the SWC (the *black dashed line*).

In the HIT group, vagal activity of both responders (subjects #7 and #29) was within the SWC (see "Methods" for details) over the first seven weeks of the HTP when training load, according to TRIMP, was mainly within the SWC. In subject #7, ln HFP values increased 'clearly' above the SWC at weeks 17–18 being most likely due to decreased (74 %) training load during the week's 16–18. On the other hand, ln HFP values of sub-

ject #29 decreased 'clearly' below the SWC at weeks 16–17 when training load was 53 % higher than mean TRIMP during the HTP. Despite this minor vagal withdraw at the end of the HTP, subject #29 could improve maximal endurance performance the most of the HIT group (V_{peak} increased 6.4 %).

The non-responders of the HIT group (subject #1 and #32) both presented lower ln HFP values at week 9 than their SWC was. In subject #1, ln HFP values fluctuated very typically below and above the SWC according to weekly TRIMP; ln HFP was low when TRIMP was high, and vice versa. Interestingly, subject #1 had two times less intra-individual variation in ln HFP values than subject #7 (responder). Subject #32 had substantial high (28 % higher than mean TRIMP) training load during the weeks 9–12 that might have resulted in decreased ln HFP values during the entire training period. However, vagal activity increased at week 18 when training load was decreased deliberately.
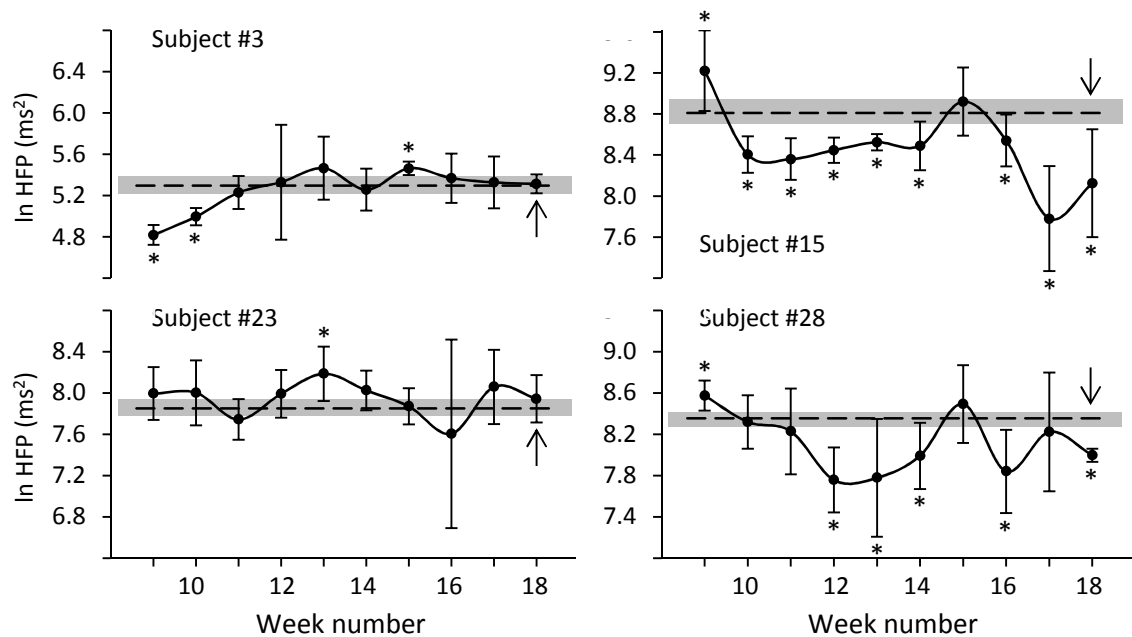

FIGURE 14. The nocturnal weekly averaged values of the natural logarithm of the high frequency power (ln HFP) with 90% confidence intervals over the 8-week hard training period in four subjects of the high-volume training group. Subjects #3, #23 represent responders and subjects #15 and #28 non-responders. The *black circles* indicate the weekly averaged lnHFP value. The *arrows* indicate the final measurement week. The *grey shaded area* indicates the individual smallest worthwhile change (SWC) in ln HFP. * indicates a 'clear' change in weekly averaged ln HFP values above/below the zero line (the mean of ln HFP values during the basic training period) of the SWC (the *black dashed line*).

In the HVT group, subject #3, responder, had 'clearly' reduced vagal activity during the weeks 9–10. The extremely high training load during the weeks 6–7 (243 % greater TRIMP than the mean during the HTP) may explain this reduction in ln HFP values. The training load and ln HFP were mainly within the SWC from to the week 10 to the end of the training period. In subject #23, ln HFP values were within the SWC throughout the training period despite the weeks when training load was reduced by ~50 % due to periodization at weeks 13 and 17.

Vagal activity of both non-responders of the HVT group (subjects #15 and #28) was 'clearly' above the SWC at week 9. However, ln HFP values decreased dramatically at beginning of the training period and remained generally below the SWC to the end of the training period. The training was periodized according to the plan so that TRIMP was clearly lower at every four weeks; in subject weeks 12 and 16, and in subject #28 weeks 13 and 17. TRIMP during the HTP increased ~11 % from the level of BTP similarly in both subjects. To conclude, the shape of the HRV profiles of the representative non-responders of the HVT group was very similar.

8 DISCUSSION

The main findings of the present study demonstrated that maximal endurance performance (VO_{2max}, V_{peak}) and nocturnal HRV (RMSSD, SDNN, ln TP) increased only in the HIT group during the HTP. Submaximal velocities (V_{AnT}, V_{AerT}) improved similarly in both training groups. Contrary to the pre-expectations, no significant changes in HRV indices were observed in the HVT group; not even in the responder's subgroup. However, low HRV prior to the HTP was observed to result in improved maximal endurance performance. In addition, the improvements in maximal endurance performance were associated with the changes in nocturnal HRV indices (SDNN, ln TP) in the HIT group.

Endurance training adaptation. All endurance performance characteristics were improved significantly during the 8-week BTP, except VO_{2max}. In total, seventeen subjects (61 %) could not improve their VO_{2max} explaining the observed insignificant improvement in a group level. However, all except five subjects (18 %) improved V_{peak}. The present data agrees with the findings of previous studies (Paavolainen et al. 1999; Kiviniemi et al. 2007) suggesting that the maximal running performance can be improved without changes in VO_{2max}. One explanation for lack of improvements in VO_{2max} during the BTP might be the insufficient training stimulus with relation to the pre-training fitness level. It is known that, for recreationally trained individuals, further improvements in maximal endurance performance can only be achieved by increasing training stimulus (Laursen & Jenkins 2002). In the present study, training volume in the BTP was not increased from the volume prior to the study and probably, therefore, training stimulus was not high enough. Thus, only minor improvements in VO_{2max} were expected. In addition, improvements in submaximal velocities (V_{AerT}, V_{AnT}) are congruent with the recent findings of Vesterinen et al. (2013), although they reported greater improvements in recreational endurance runners during similar training compared with the present study. This might be the result of the longer training period (Vesterinen et al. 2013).

Endurance training adaptations to increased training stimulus (i.e. intensity and/or volume) in recreationally active individuals have been widely examined (e.g. Laursen et al. 2002; Esfarjani & Laursen 2007; Helgerud et al. 2007; Ingham et al. 2008). Howev-

er, most of the previous training studies have mainly focused on physiological adaptations to high-intensity interval training. In the present study, the 8-week HTP consisted of either high-intensity (3 sessions per week) or high-volume (38 % increase from the BTP) endurance training. The HIT group could improve all endurance performance characteristics during the HTP, whereas the HVT group improved significantly only V_{AerT}. Between-groups differences in V_{peak} and V_{AnT} adaptations were observed as well (differences rated as moderate according to ES). The present findings are in line with Laursen et al. (2002), Esfarjani and Laursen (2007) and Helgerud et al. (2007) who showed that maximal endurance performance was improved after 4–10 weeks of high-intensity interval training in recreational endurance athletes. In addition, without a few exceptions, an additional increase in low-intensity training volume in highly trained individuals has not been demonstrated to enhance further improvements in endurance performance (Laursen & Jenkins 2002). However, the study of Ingham et al. (2008) showed that the 12-week high-volume (~95 rowing km per week) training period at the intensity below aerobic threshold (98 % of training duration) improved VO_{2max} significantly in sub-elite male rowers. Furthermore, low-intensity training was observed to elicit greater adaptation in power at aerobic threshold than a mix of low- and high-intensity endurance training. The authors concluded that both training regimes have similar training effects on performance characteristics (Ingham et al. 2008). However, the results of the present study do not support this claim, although the improvements in V_{AerT} were similar in both training groups (~2 %) despite the different training programs. This may be due to the fact that the time course for endurance training adaptation with increases in training volume may not occur as rapidly (Costill et al. 1991) as with increases in high-intensity training (Weston et al. 1997).

In addition to the group-level analyses, a retrospective analysis of the individual adaptation to training supports the above-mentioned finding of endurance training adaptation; the number of responders (V_{peak} improved 2 % or more) was double in the HIT group compared with the HVT group (8/14 vs. 4/14 subjects, respectively). Furthermore, eight subjects (57 %) could improve neither VO_{2max} nor V_{peak} in the HVT group during the HTP when the corresponding count in the HIT group was only four subjects (28 %). However, despite lack of significant high-volume endurance training-induced adaptations, the present results demonstrate that some individuals may enhance their endurance performance (e.g. VO_{2max} up to 18 %) after being exposed to high-volume training

load. Such great improvements have been normally reported after a similar type of training among individuals with VO_{2max} less than 40 ml/kg/min (Berger et al. 2006; Gormley et al. 2008). Interestingly, the fitness level of the responders in the HVT group was average; none was below the 25th percentile. Therefore no association between the fitness level and the endurance training adaptation was found. Thus, the substantial endurance training adaptation of these individuals cannot be explained by low baseline endurance performance level as has been suggested by Buchheit et al. (2010). Instead, different types of training background (i.e. high-intensity, low-volume), individual genotype (Nummela et al. 2010) and cardiac autonomic function (Hautala et al. 2003) may rather explain this phenomenon.

Early training studies have shown that females do not reach the same absolute levels of VO_{2max} or V_{peak} than males; the gap between genders varies between 24 % in sedentary people and 7 % in endurance athletes (Ogawa et al. 1992; Helgerud 1994). In the present study, the males had ~12 % higher absolute levels of endurance performance characteristics compared to the females. However, no gender differences were observed in the changes in endurance performance characteristics during either of the training periods. Similarly, Carter et al. (2003b) found no gender differences in endurance training response either. Despite the differences in absolute levels between the genders, it has been shown that both genders have similar qualitative cardiovascular pattern of response to endurance training and, therefore, can be treated statistically as one population when the focus is on the changes in endurance running performance (Raven et al. 1972).

Group-level HRV adaptation. Numerous studies have shown that endurance training increases cardiac autonomic activity and therefore HRV indices, especially vagal-related indices (Portier et al. 2001; Yamamoto et al. 2001; Kiviniemi et al. 2007; Buchheit et al. 2010; Nummela et al. 2010; Vesterinen et al. 2013). However, extremely intensive endurance training with inadequate recovery has been observed to diminish HRV indices (Pichot et al. 2000; Iellamo et al. 2002; Plews et al. 2012). In the present study, the 8-week BTP could not elicit relevant changes in HRV indices yet RMSSD was decreased; however, the decrease was practically meaningless according to the trivial effect size. Instead, vagal activity together with overall autonomic activity increased during the following 8-week HTP in the HIT group. The effect of high-intensity training on HRV indices is in line with previous studies concerning a similar type of training

(Portier et al. 2001; Buchheit et al. 2010; Vesterinen et al. 2013). In the recent study of Vesterinen et al. (2013), for example, no changes were observed in HRV indices after 14-week basic training period (on avg. 5 times/week at 68 % HR_{max}), but after hard training period of the same duration (on avg. 4 times/week at 72 % HR_{max} incl. 18 moderate- to high-intensity sessions) vagal-related HRV indices increased significantly. In addition, sympathetic withdrawal may occur due to increased vagal control (Portier et al. 2001). However, the above studies differ in fitness level and training background of the subjects with a range of sedentary individuals having VO_{2max} less than 40 ml/kg/min (Nummela et al. 2010) to elite athletes with VO_{2max} over 70 ml/kg/min (Portier et al. 2001; Plews et al. 2012). Based on the data of the present study, the increased cardiac vagal activity after high-intensity training may be a marker of a positive response to this type of training. The findings of Buchheit et al. (2010) support the previous suggestion.

A bit surprisingly, any of HRV indices was not altered in the HVT group despite the increased training volume (all ES were trivial or small). The present results are consistent with those reported by Uusitalo et al. (1998) and Hedelin et al. (2000a), although it was not hypothesised beforehand that no changes will occur. Furthermore, the effects of increases in training volume on basal HRV indices has been examined in at least four other studies (Iwasaki et al. 2003; Buchheit et al. 2004; Iellamo et al. 2004; Manzi et al. 2009). All these studies, except the study of Iellamo et al. (2004), demonstrated that vagal-related HRV indices (RMSSD, HFP) rose significantly when training volume was increased moderately. In addition, LFP has been shown to decrease at the same time indicating reduced sympathetic activity (Buchheit et al. 2004). However, further increases in training volume (~70 %) resulted in diminished HRV indices (Iwasaki et al. 2003; Manzi et al. 2009). The finding of Iellamo et al. (2004) supports this; HFP decreased in elite rowers when training volume was doubled. This so-called dose-response relationship of the cardiovascular adaptation expressed first time by Iwasaki et al. (2003) is unlikely to be the cause of unaltered cardiac autonomic control observed in the present study, based on the following facts: 1) the increase in training volume was only moderate (38 %) and 2) resting HR was not decreased either, contrary to Iwasaki et al. (2003) and Buchheit et al. (2004).

The reasons for unaltered HR and HRV indices in the HVT group are most probably related to the intensity and duration of the training period. Despite the increased training

volume and thus training load, training intensity (65 % HR_{max}) was obviously too low to induce any changes in cardiac autonomic activity. Similarly, Davy et al. (1997) found that both resting HR and HRV indices were unchanged from baseline levels after 12 weeks of aerobic training at the intensity between 60–75 % of HR_{max}. Later Achten and Jeukendrup (2003) concluded in their review that at least vigorous training intensity is needed to induce changes in basal HRV indices. This conclusion is supported by the findings of the present study in relation to HRV adaptation of the HIT group. On the other hand, the duration of the training period may not have been long enough if compared, for example, with the study of Iwasaki et al. (2003) (8 weeks vs. 6 months). Additionally, the methodological differences between the foregoing studies should not be overlooked. The correspondence of the results between these studies can be argued due to the wide range of HRV recording methods varying from short-term recordings after awakening (e.g. Buchheit et al. 2010) or during daytime (e.g. Manzi et al. 2009) to long-term recordings during night-time (e.g. Vesterinen et al. 2013). The most complex HRV indices cannot be calculated from the short-term recordings lasting contrary to those recorded over longer period (TaskForce 1996). In addition, the night-time recordings, used also in this study, have been considered to reflect a more standardized condition being barely influenced by behavioural pattern (Pichot et al. 2000).

There several studies that have focused on the influence of gender on cardiac autonomic tone (Ramaekers et al. 1998; Kuo et al. 1999; Barantke et al. 2008; Kiviniemi et al. 2010). The conclusion drawn from these studies is that males have higher HRV indices than females. In the present study, however, no gender differences existed in the basal levels or changes of nocturnal HRV indices during the training. This is in line with the finding of Nummela et al. (2010); no gender effects were found. A difficulty comparing the present finding to the previous studies is that the physical fitness of the subjects is different. For example, in the study of Kiviniemi et al. (2010) the males had ~33 % higher absolute levels of VO_{2max} compared to the females, whereas in the present study the difference was ~12 %. Individuals having high aerobic capacity (i.e. VO_{2max}) show also great HRV indices (e.g. Hautala 2009). Therefore, it can be suggested that the gender difference in HRV indices observed in the previous studies might be partly due to great gender difference in physical fitness, unlike in the present study. Finally, the non-observed gender difference in HRV indices supports the gender-mixed training group design of the present study in order to have sufficient statistical power.

Individual HRV adaptation. The present data together with previous intervention studies support the hypothesis of Hautala et al. (2009) that cardiac autonomic control plays an important role in endurance training adaptation. However, only a few researchers (Buchheit et al. 2010; Nummela et al. 2010), in addition to the present study, have tried to examine the differences in HRV adaptation between training responders and non-responders. The only way to examine the individual responses to training is to group the subjects into the responders and non-responders a posteriori. This may result in a small sample size for the one of the subgroups. Therefore, in the present study, the *post hoc* comparisons were assessed for significance using also an inferences-based method to be made with a small sample size (Hopkins et al. 2009). In addition, the individual ln HFP profiles were created to illustrate the time course of change in vagal activity.

In the HIT group, SDNN values of the responders were clearly lower than the non-responders ones prior to the HTP but increased significantly during the training period reaching the level of the non-responders indicating enhanced parasympathetic activity. In addition, most of the responders showed increased vagal activity, although not all of them (subject #29), together with greater day-to-day variation compared with the non-responders. These findings are in accordance with Buchheit et al. (2010) who showed that low resting HRV at baseline and increased vagal activity during the training period were associated with positive training adaptation to a 8-week interval training period among moderately trained runners. On the other hand, opposite findings have also been reported; high HFP or TP levels at baseline may predict positive training adaptation to high-intensity training (Hautala et al. 2003; Vesterinen et al. 2013). However, the use of basal HRV indices in order to predict endurance training adaptation may be problematic because of remarkable intra-individual differences in basal HRV indices (Al Haddad et al. 2011). The absolute values of spectral HRV indices may vary from hundreds (e.g. subject #7) to tens of thousands (subject #29) and, consequently, the reported 'high' or 'low' values in one study may be tenfold greater or smaller in another study. Instead, the time course of changes in HRV indices would be a more promising way to monitor the trainability and endurance training adaptation. Especially vagal-related indices have been used for this purpose recently (Plews et al. 2012) but changes in SDNN or TP values may be used as well. Finally, there was nothing to sug-

gest that the non-responders of the HIT group were overtrained during the training period, based on the individual ln HFP profiles and questionnaires.

In the HVT group, no significant changes in HRV indices were observed either in the responder's or non-responder's subgroup. This finding concerning unchanged HRV indices of the responders is novel, although a few studies (Loimaala et al. 2000; Vesterinen et al. 2013) have shown that low-intensity training does not induce changes in HRV indices despite improved endurance performance observed in a group-level. On the other hand, Nummela et al. (2010) found that nocturnal HFP of the responders increased significantly during moderate-intensity endurance training among sedentary subjects. Thus, in the present study it was expected that HRV indices would increase in individuals having positive endurance training adaptation. Although the present results do not support this hypothesis, slight trend towards increased vagal and overall ANS activity was observed in the responder's subgroup. Further, vagal activity increased significantly in two out of four responders (subject #3 and #37). Due to the extremely small number of responders (n=4) the powerful conclusions cannot be drawn. The underlying mechanisms of changes in ANS activity in relation with endurance training adaptation are unknown. The effect of everyday life psychological stressors, such as work and family, and quite typical upper respiratory tract infections for HRV indices cannot be ignored. For example, it can be speculated that HFP of subject #23 did not increase at week 18 anymore due to slight upper respiratory tract infection (observation from the training diary) as did at week 13, when training load was similar to week 18 (Figure 14).

Similarly to the HIT group, the responders of the HVT group had significantly lower basal HRV indices both prior to and after the HTP compared to the non-responders. This supports the findings of Buchheit et al. (2010), although training intensity and volume of the interventions are not identical. Low HRV indices may also be linked to a poor endurance performance level (Aubert et al. 2003) or minor endurance training background (Buchheit et al. 2004). In the present study, however, HRV indices prior to the HTP were not associated with baseline endurance performance or previous training activity. As described earlier, the results and conclusions of the previous studies have not been entirely consistent with each other. It has been suggested by Vesterinen et al. (2013) that low HRV can possibly predict an inability to cope with the training load and

the accumulation of fatigue. The data of the present study leads rather an opposite suggestion whereby low HRV may predict potentially to positive endurance training adaptation regardless of training intensity and volume. Instead, high HRV levels prior to the training period were observed among the non-responders of the HVT group with poor training adaptation. The ln HFP profiles of subject #15 and #28 illustrate the time course of changes in vagal modulation during the HTP. In both subjects the HFP levels decreased significantly by the effect of training load, obviously, resulting possibly in an over-trained condition (especially subject #15). Lack of periodization may partly explain poor training adaptation and this kind of HRV profiles; e.g. the training load according to TRIMP was constant at every week in subject #15, whereas periodization of subject #28 followed the plan. It appears unlikely that the higher training load of non-responders than responders would explain the opposite trends observed in HRV indices because training load did not differ significantly between the *post hoc* subgroups. Finally, it must be noted that great week-to-week variation exists in basal vagal-related indices (Figures 13 and 14) and thus long-term trend might be a more representative sign of changes in HRV indices instead of isolated weeks, although single values of the week are average of two recordings (Plews et al. 2012).

Association between HRV and endurance training adaptation. It has been widely reported that HRV indices are associated with endurance performance (Buchheit & Gindre 2006; Buchheit et al. 2010) and endurance training adaptation (Hautala et al. 2003; Nummela et al. 2010; Vesterinen et al. 2013). However, only a limited number of studies concerns recreational athletes (Buchheit et al. 2010; Vesterinen et al. 2013). In the present study, the significant correlation between the baseline HRV indices and endurance performance was observed only prior to the BTP, not HTP. SDNN showed to have the strongest relationship with VO_{2max} ($r=0.46$, $p=0.015$). This finding is in line with Buchheit & Gindre (2006) and Buchheit et al. (2010). In addition, basal HRV indices prior to the training periods correlated significantly with the endurance training adaptation. Interestingly, a positive relationship was found between the baseline ln TP values and the change in V_{AnT} during the BTP ($r=0.51$, $p=0.005$), whereas a negative relationship was observed between RMSSD values at week 9 and the change in V_{peak} and V_{AnT} during the HTP ($r=-0.54$, $p=0.049$ and $r=-0.67$, $p=0.009$, respectively). The differences between the training periods might be partly explained by the fact that RMSSD is thought to be vagal-related HRV index while TP reflects both vagal and sympathetic

activity. However, Vesterinen et al. (2013) have reported positive relationships between the endurance training adaptation and both baseline HFP and TP values in recreational endurance runners. In the HIT group only, changes in SDNN values during the HTP were associated with the changes in V_{peak} at the same time (r=0.55, p=0.050) accounting 30 % of the variance in the adaptation to endurance training. Consistent findings were also reported by Buchheit et al. (2010) after a similar type training intervention. Taken together, it can be suggested that there likely exists an interdependence between cardiac autonomic control and aerobic performance.

Limitations of the study. The RR interval recordings made at home may not be considered as highly standardized as those made in laboratory conditions. In the present study, this compromise had to be made for practical reasons. In addition to endurance training, there may be several other factors that affect HRV, such as irregular sleeping habits, everyday life physiological and psychological stressors. The incidence of these factors may be very common among sedentary and recreational athletes due to work and the family. Therefore, it cannot be concluded that the changes in HRV indices would have been totally consequence of training *per se* because the RR interval recordings are quite sensitive to all kind of stress (TaskForce 1996). To exclude, or at least reduce, the effect of everyday life stress on the RR interval recordings the subjects were asked to inform if the daily routines were abnormal during the training intervention (e.g. night shift instead of day shift). In these cases the single recordings were excluded from the final analyses. Furthermore, there was no control group that did not exercise. Thus, the present results must be interpreted cautiously.

Additionally, in the present study, the RR interval recordings were measured on four consecutive nights each training and measurement week, as a rule. The original purpose was to provide HRV indices as averages of two consecutive nights but this was not possible for all subjects due to erroneous RR interval recordings. Instead, recordings of two single isolated nights after a light training day were used to calculate the averages of HRV indices. This can be considered an obvious limitation of this study as has been suggested in general (Plews et al. 2012). However, almost all studies referred in this study have used single isolated recordings as well. Furthermore, the final sample size of the training groups was relatively small (n=14) and thus limited the statistical power.

This was compensated by a novel statistical analysis approach based on the magnitudes of change (Hopkins et al. 2009).

Conclusions. The present study showed that high-intensity endurance training induced greater changes in endurance running performance compared with high-volume endurance training. In addition, it seems that high-intensity endurance training is needed in order to lead significant changes in nocturnal HRV indices among recreational endurance runners (VO_{2max} ~50 ml/kg/min). However, high-volume endurance training load may induce changes in vagal activity of nocturnal cardiovascular autonomic regulation in some individuals together with altered endurance running performance. No gender differences existed in the changes in nocturnal HRV indices and endurance running performance. Furthermore, the subjects who improved their endurance performance most showed lower basal levels of HRV indices than the subjects who improved only slightly or did not improve at all. Finally, great increases in nocturnal HRV indices were associated with good endurance training adaptations after high-intensity training.

Practical applications. Based on the findings of the present study HRV measurements, as an inexpensive and practical method, may be of assistance in monitoring endurance training adaptation and stress among recreational endurance athletes. In order to achieve further improvements in endurance performance, the training programs of recreational athletes should consist of training periods with high training load (i.e. high-intensity or high-volume) and recovery periods. Thus, the knowledge of individual trainability and training status is extremely important. Weekly HRV monitoring of vagal activity may provide objective information of the training status and possibly prevent long-term states of overreaching.

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