

Heart rate variability dynamics during early recovery after different endurance exercises

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Abstract Since heart rate variability (HRV) during the first minutes of the recovery after exercise has barely been studied, we wanted to find out HRV dynamics immediately after five different constant-speed exercises. Thirteen sedentary women performed two low-intensity (3,500 m [3,500_{LI}] and 7,000 m [7,000_{LI}] at 50% of the velocity of VO_{2max} [vVO_{2max}]), two moderate-intensity (3,500 m [3,500_{MI}] and 7,000 m [7,000_{MI}] at $\sim 63\%$ vVO_{2max}) and one high-intensity (3,500 m at $\sim 74\%$ vVO_{2max} [3,500_{HI}]) exercises on a treadmill. HRV was analyzed with short-time Fourier transform method during the 30-min recovery. High frequency power (HFP) was for the first time higher than at the end of the exercise after the first minute of the recovery (3,500_{LI} and 7,000_{LI}, $P < 0.001$), after the fourth (3,500_{MI}, $P < 0.05$) and the fifth (7,000_{MI}, $P < 0.05$) minute of the recovery and at the end of the 30-min recovery (3,500_{HI}, $P < 0.01$). There were no differences in HRV between 3,500_{LI} and 7,000_{LI} or between 3,500_{MI} and 7,000_{MI} during the recovery. The levels of HFP and TP were higher during the whole recovery after 3,500_{LI} compared to 3,500_{MI} and 3,500_{HI}. We found increased HFP, presumably caused by vagal reactivation, during the first

5 min of the recovery after each exercise, except for 3,500_{HI}. The increased intensity of the exercise resulted in slower recovery of HFP as well as lower levels of HFP and TP when compared to low-intensity exercise. Instead, the doubled running distance had no influence on HRV recovery.

Keywords HRV · Dynamics · Recovery · Short-time Fourier transform · Constant-speed exercises

Introduction

During the last decades, there has been increasing interest to investigate heart rate variability (HRV) as an index of autonomic control of heart. Such studies have been accomplished mainly during rest but also during exercise (Arai et al. 1989; Cottin et al. 2004; Pichon et al. 2004; Tulppo et al. 1998) and recovery (Arai et al. 1989; Casties et al. 2006; Goldberger et al. 2006; Mourot et al. 2004; Terziotti et al. 2001). However, the dynamics of HRV during the immediate recovery after exercise have remained unclear, probably due to methodological issues. Traditional spectral analysis methods require stationarity of the processed signal (Task Force 1996) and therefore they cannot be used during exercise or early recovery. On that account, in most of the HRV studies the first minutes of the recovery have been excluded from analysis.

Short-time Fourier transform (STFT) method provides time–frequency information of the signal. It can be used during transient phases of RRI signal which allows the detection of dynamics of HRV during any given time (Elsenbruch et al. 1999). This method has been previously used to measure HRV during exercise (Pichon et al. 2004) and it has also been proved to detect fast changes in vagal

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responses (Keselbrener and Akselrod 1996, Martinmäki et al. 2006b).

During the immediate recovery after exercise, fast changes occur in the cardiac function. Increase in the parasympathetic activity, as well as decrease in the sympathetic activity, is known to occur after exercise cessation (Savin et al. 1982; Pierpont et al. 2000). However, neither the time course of the autonomic changes nor the contribution of vagal and sympathetic changes during early recovery is clear. It has been suggested that sustained sympathetic activity maintains high heart rate levels during the early recovery despite the parasympathetic reactivation (Furlan et al. 1993; Pierpont et al. 2000).

The relationship between HRV and autonomic modulation is not simple. However, it is well established that at rest high frequency power (HFP) is modulated essentially by fluctuations in the parasympathetic branch of the autonomic nervous system (Akselrod et al. 1981; Berntson et al. 1993; Cacioppo et al. 1994; Martinmäki et al. 2006a). Although HFP during rest and different physiological conditions have been studied widely, HFP dynamics during the early recovery after exercise has been investigated only in a few studies. Casties et al. (2006) studied HRV immediately after a 30-min progressive exercise (up to 90% VO_{2max}) and found a tendency of increasing HFP during the first 10 min of the recovery, although there were no significant differences when compared to exercise level. Also Goldberger et al. (2006) studied the immediate (5-min) recovery of HRV after a maximal bout of exercise. They investigated the recovery of HRV with and without a parasympathetic blockade (atropine), and concluded that HRV during the immediate recovery after exercise can be used to evaluate parasympathetic reactivation. Martinmäki and Rusko (2007) found increasing HRV during the first ten recovery minutes after high-intensity (61% of maximal power) and low-intensity (29% of maximal power) exercises. In addition, in a previous study, we investigated HRV immediately after different high-intensity (80, 85 and 93% of vVO_{2max}) exercises, and found increased TP during the first 5 min of recovery, but no recovery of HFP (Kaikkonen et al. 2007).

Low-intensity exercises, during which the sympathetic nervous system is not greatly activated, may be hypothesized to have different recovery pattern than high-intensity exercises, during which the sympathetic activity contribution is increased (Pierpont et al. 2000). However, the effect of different exercise models, e.g., increased intensity versus increased duration of the exercise, on immediate post-exercise HRV is not well known. Heart rate, blood lactate concentration and oxygen uptake are known to increase more during high-intensity exercise than during low-intensity exercise, which suggests that metabolic functions are elevated more during high-intensity exercise. Therefore

it can be hypothesized that also post-exercise HRV dynamics is influenced by this elevated metabolism. The prolonged distance of the exercise is not known to have similar effects on metabolism, so it may also be hypothesized that the post-exercise HRV dynamics is not drastically altered by prolonged distance.

The main purpose of the present study was to find out the HRV dynamics, especially vagal reactivation, during the immediate recovery after different constant-speed endurance exercises. We also wanted to find out the effects of increased intensity and prolonged running distance on post-exercise HRV. We hypothesized, according to previous studies (Casties et al. 2006; Goldberger et al. 2006), that HRV increases immediately after exercise cessation as a function of time, but the recovery after high-intensity exercise will be slower than after low-intensity exercises.

Methods

Subjects

Thirteen sedentary women were selected on volunteer basis to participate in this study. All subjects were non-smoking, had normal body mass ($BMI < 30 \text{ kg m}^{-2}$) and none of them was taking any regular medication. All subjects gave written informed consent and filled a brief health questionnaire concerning different chronic systemic diseases. Resting ECG (Cardiofax ECG-9320, Japan) was recorded to ensure they had no cardiac abnormalities. Subjects had the right to withdraw from the study at any time. The study was approved by the ethics committee of the University of Jyväskylä. Physical characteristics of the subjects are presented in Table 1.

Graded maximal treadmill test

After being accustomed to the study protocol and equipments, subjects performed a maximal graded treadmill test. Body height, body mass and body fat (%) of subjects were

Table 1 Mean (SD) physical characteristics of the subjects ($N = 13$)

Age (year)	35 (3)
Height (cm)	168 (5)
Body mass (kg)	63.4 (10.2)
BMI (kg m^{-2})	22.5 (3.0)
Body fat (%)	27.4 (4.3)
HR _{max} (bpm)	191 (10)
VO _{2max} ($\text{ml kg}^{-1} \text{ min}^{-1}$)	35.9 (3.2)
vVO_{2max} (km h^{-1})	10.8 (0.8)

BMI body mass index, vVO_{2max} the velocity at VO_{2max}

measured prior to the test. The initial test speed of 5 km h^{-1} (gradient 1%) was used in the treadmill test, and the speed was increased with 1 km h^{-1} every 3 min until voluntary exhaustion. First two speeds were performed walking and the following speeds running. Breath-by-breath respiratory data (V_{\max} 229, Sensor Medics, Palo Alto, USA) and R-R intervals (RRI) (Suunto t6 wristop computer, Suunto Oy, Vantaa, Finland) were measured continuously during the test. Fingertip blood samples for blood lactate analysis were taken at the end of each step. Anaerobic threshold (AT) was determined with blood lactate and respiratory parameters, according to Aunola and Rusko (1984). The highest 60-s VO_2 value was considered as maximal oxygen uptake ($\text{VO}_{2\max}$). The velocity at AT (v_{AT}) and at $\text{VO}_{2\max}$ ($v\text{VO}_{2\max}$) was also determined.

Exercises

Running speeds for exercises were determined individually based on the maximal treadmill test results. Exercises were designed so that the effect of increased intensity and prolonged running distance of the exercise on post-exercise HRV could be investigated. Subjects performed two low-intensity exercises (3,500 m [$3,500_{\text{LI}}$] and 7,000 m [$7,000_{\text{LI}}$] at 50% $v\text{VO}_{2\max}$), two moderate-intensity exercises (3,500 m [$3,500_{\text{MI}}$] and 7,000 m [$7,000_{\text{MI}}$] at [50% $v\text{VO}_{2\max}$ + (v_{AT} - 50% $v\text{VO}_{2\max}$)/2]) and one high-intensity exercise (3,500 m [$3,500_{\text{HI}}$] at v_{AT}), on a treadmill in random order. The mean intensity of the moderate- and high-intensity exercises was 63 and 74% of $v\text{VO}_{2\max}$. Exercises were performed once a week during the following 6 weeks after the maximal treadmill test. Subjects were guided to avoid severe physical stress 2 days preceding the exercise days, avoid caffeine and alcohol in the exercise days and heavy meal 4 h before exercises. They were also advised to take care of proper fluid balance prior to exercises.

Each exercise session started with the measurement of subjects' resting RRIs (Suunto t6 wristop computer, Suunto Oy, Vantaa, Finland) in sitting (5 min) posture. After the resting measurement, subjects started the previously determined exercise, during which RRIs and breath-by-breath respiratory data (V_{\max} 229, Sensor Medics, Palo Alto, USA) were collected. Rating of Perceived Exertion (RPE, scale 0–10+) and fingertip blood samples were obtained immediately after exercise cessation. Respiratory frequency (RF) was obtained from the breath-by-breath respiratory data. Immediately after the exercise cessation a chair was placed on the treadmill, on which subjects were seated. Controlled, passive recovery for 30 min was performed, during which the subjects were sitting. Moving and talking was prohibited during the recovery.

HRV analysis

RRI data was transferred to computer with Suunto Training Manager-program (Suunto Oy, Vantaa, Finland). Further signal processing and HRV calculation was performed with Matlab-program (Matlab 7, MathWorks, Inc., Natick, USA). RRIs were checked and edited for artefacts using a detecting algorithm and subsequently verified by visual inspection. The RRI series were then resampled at a rate of 5 Hz using linear interpolation to obtain equidistantly sampled time series. The data were further filtered with a band-pass filter to remove variance below 0.04 Hz and above 1.0 Hz.

Short-time Fourier transform method was used for HRV analysis. A series of 512 samples with the time window function of Hanning was used to obtain HRV data. The STFT method provides time–frequency decompositions of the consecutive RRI time series by calculating power spectra for each 200 ms sample. The time-frequency spectra of RRI series was obtained by calculating fast Fourier transform of the first sample and then by shifting the window from one RRI sample to another consisting each sample of the chosen period. Resting HRV was analyzed with the same method in order to be able to compare the results reliably.

High frequency power (HFP, 0.15–1.0 Hz) and total power (TP, 0.04–1.0 Hz) were calculated as integrals of the respective power spectral density curve. The higher frequency limit of 1.0 Hz was used to include the respiratory frequency during and immediately after exercise to the analysis. Further, averages of 3 min (pre-exercise sitting), 1 min (end of the exercise, during the recovery at 1–5 min) and 4 min (end of the recovery, minutes 27–30) of these parameters were calculated. These averages have been used in figures as rest (pre-exercise sitting), exe (last minute of the exercise), rec1–rec5 (the first 5 min of the recovery) and rec 30 (the last minutes of the 30-min recovery). We focused our study only on the mainly vagally mediated parameters HFP and TP, because the origins and implications of low frequency power (LFP) are more unclear.

Statistical analyses

To meet the assumptions of parametric statistical analysis, a natural logarithmic transformation of the values of HFP and TP was performed. A repeated measures analysis of variance (ANOVA) with contrasts was performed to study the main effect and interaction of (1) the exercise intensity and recovery time (3,500_{LI}, 3,500_{MI}, 3,500_{HI}) and (2) the exercise duration and recovery time (separately for 3,500_{LI} and 7,000_{LI} and for 3,500_{MI} and 7,000_{MI}) during the 5-min acute recovery. If the main effect of exercise intensity was

significant, further analysis was made between the 3,500_{LI}, 3,500_{MI} and 7,000_{HI} to test the difference between these exercises at each recovery minute. A repeated measures ANOVA with contrasts was also performed to study the main effect of recovery time between rest and the end of the recovery. A paired, two-tailed Student's *T*-test was further used to compare the within-exercise differences. In all statistical tests, differences were considered significant when $P < 0.05$.

Results

Rest and exercise

At rest, the main effect of repeated measures ANOVA showed no differences in HR, TP or HFP between the five exercises. The average pre-exercise HR, TP and HFP, including all exercises, in sitting posture were 75 ± 10 bpm, 7.6 ± 0.8 ln ms² and 6.8 ± 1.0 ln ms², respectively. The values of blood lactate (BLa), RPE and VO₂ at the end of the exercises are presented in Table 2. Increased intensity of the 3,500_{LI} (=3,500_{MI} and 3,500_{HI}) resulted in higher ($P < 0.01$) VO₂, BLa and RPE values and these parameters were also higher ($P < 0.001$) at the end of 3,500_{HI} when compared to 3,500_{MI}.

Comparisons of the end-exercise values between the three different intensities showed that HR was lower ($P < 0.001$, Fig. 1) and TP ($P < 0.001$, Fig. 2) and HFP ($P < 0.001$, Fig. 3) higher at the end of 3,500_{LI} when compared to 3,500_{MI} and 3,500_{HI}. There were no differences in HR, TP or HFP between the low-intensity (3,500_{LI} and 7,000_{LI}) or between the moderate-intensity (3,500_{MI} and 7,000_{MI}) exercises during the last minute of the exercise.

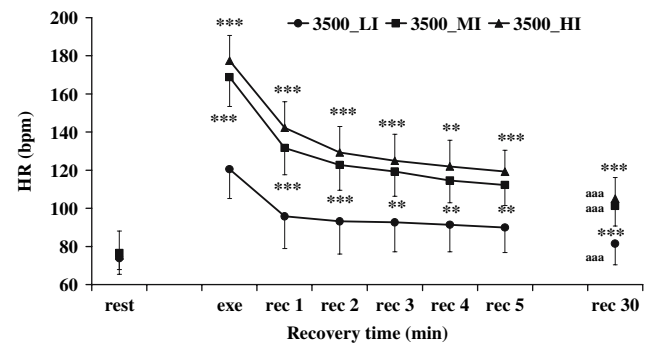


Fig. 1 The effect of increased intensity of the exercise on HR recovery. 3,500_{LI} = 3,500 m at 50% vVO_{2max} , 3,500_{MI} = 3,500 m at $\sim 63\%$ vVO_{2max} , 3,500_{HI} = 3,500 m at $\sim 74\%$ vVO_{2max} . Significantly different from 3,500_{LI} (* $P < 0.01$, *** $P < 0.001$) and significantly different from the resting value (aaa $P < 0.001$). The graphs of 7,000_{LI} and 7,000_{MI} are not presented, since they were similar to 3,500_{LI} and 3,500_{MI}

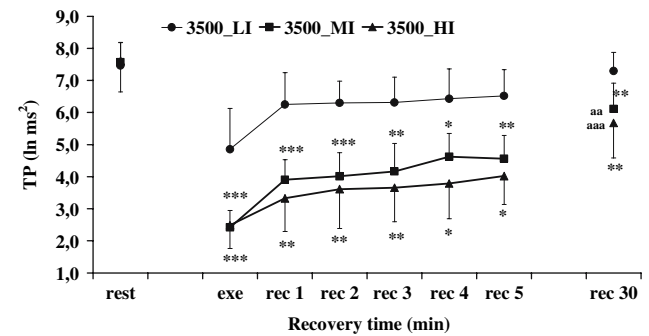


Fig. 2 The effect of increased intensity of the exercise on TP recovery. 3,500_{LI} = 3,500 m at 50% vVO_{2max} , 3,500_{MI} = 3,500 m at $\sim 63\%$ vVO_{2max} , 3,500_{HI} = 3,500 m at $\sim 74\%$ vVO_{2max} . Significantly different from 3,500_{LI} (* $P < 0.01$, *** $P < 0.001$) and significantly different from the resting value (aa $P < 0.01$, aaa $P < 0.001$). The graphs of 7,000_{LI} and 7,000_{MI} are not presented, since they were similar to 3,500_{LI} and 3,500_{MI}

Table 2 Descriptive parameters of the exercises (average \pm SD)

	3,500 _{LI}	3,500 _{MI}	3,500 _{HI}	7,000 _{LI}	7,000 _{MI}
Speed (km h ⁻¹)	5.5 \pm 0.4	6.9 \pm 0.6	8.0 \pm 0.6	5.5 \pm 0.4	6.9 \pm 0.6
HR (bpm)	121 \pm 15	169 \pm 15***	177 \pm 13***	125 \pm 18	172 \pm 14***
VO ₂ (% max)	45 \pm 5	76 \pm 11***aaa	89 \pm 12***	44 \pm 8	77 \pm 9***
VO ₂ (ml kg ⁻¹ min ⁻¹)	16 \pm 2	27 \pm 3***aaa	31 \pm 5***	16 \pm 3	27 \pm 3***
BLa (mmol l ⁻¹)	0.8 \pm 0.2	2.8 \pm 1.4***aaa	4.3 \pm 1.4***	1.3 \pm 0.8*	2.8 \pm 1.2***
RPE (0–10+)	2.3 \pm 1.5	5.8 \pm 2.3***	6.8 \pm 1.8***	3.4 \pm 2.3*	6.2 \pm 2.6***

BLa Blood lactate concentration at the end of the exercise, RPE rating of perceived exertion at the end of the exercise. 3,500_{LI} = 3,500 m at 50% vVO_{2max} , 3,500_{MI} = 3,500 m at $\sim 63\%$ vVO_{2max} , 3,500_{HI} = 3,500 m at $\sim 74\%$ vVO_{2max} , 7,000_{LI} = 7,000 m at 50% vVO_{2max} , 7,000_{MI} = 7,000 m at $\sim 63\%$ vVO_{2max} . Statistical differences between exercises when compared to 3,500_{LI}

* $P < 0.05$ ** $P < 0.01$, *** $P < 0.001$ and between 3,500_{MI} and 3,500_{HI}, aaa $P < 0.001$

No differences between 3,500_{MI} and 7,000_{MI}

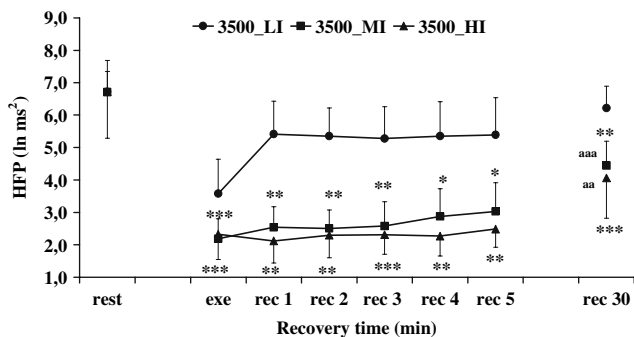


Fig. 3 The effect of increased intensity of the exercise on HFP recovery. 3,500_{LI} = 3,500 m at 50% vVO_{2max} , 3,500_{MI} = 3,500 m at ~63% vVO_{2max} , 3,500_{HI} = 3,500 m at ~74% vVO_{2max} . Significantly different from 3,500_{LI} (* $P < 0.05$, ** $P < 0.01$, *** $P < 0.001$) and significantly different from the resting value (aa $P < 0.01$, aaa $P < 0.001$). The graphs of 7,000_{LI} and 7,000_{MI} are not presented, since they were similar to 3,500_{LI} and 3,500_{MI}

The acute 5-min recovery

The main effect of exercise intensity ($P < 0.001$) and the main effect of recovery time ($P < 0.001$) on HR were significant between the three 3,500 m exercises during the acute 5-min recovery. Further comparisons between these three exercises showed that HR was higher after 3,500_{MI} ($P < 0.01$) and 3,500_{HI} ($P < 0.01$) when compared to 3,500_{LI} during the entire recovery. The main effect of exercise duration on HR was not significant between 3,500_{LI} and 7,000_{LI} or between 3,500_{MI} and 7,000_{MI}. The dynamics of HR during the recovery are presented in Fig. 1.

The main effect of exercise intensity ($P < 0.001$) and the main effect of recovery time ($P < 0.001$) on TP were significant between the three 3,500 m exercises during the acute 5-min recovery. Further comparisons between the three intensities showed that TP was lower after 3,500_{MI} ($P < 0.05$) and 3,500_{HI} ($P < 0.05$) when compared to 3,500_{LI} during each minute of the 5-min recovery (Fig. 2). The main effect of exercise duration on TP was not significant between 3,500_{LI} and 7,000_{LI} or between 3,500_{MI} and 7,000_{MI}.

The main effect of exercise intensity ($P < 0.001$) and the main effect of recovery time ($P < 0.001$) on HFP were significant between the three 3,500 m exercises during the acute 5-min recovery. Also the interaction between the intensity \times recovery time was significant ($P < 0.05$) between these exercises. Further comparisons between the three different intensities showed that HFP was lower after 3,500_{MI} ($P < 0.05$) and 3,500_{HI} ($P < 0.05$) when compared to 3,500_{LI} during each minute of the 5-min recovery (Fig. 3). The main effect of exercise duration on HFP was not significant between 3,500_{LI} and 7,000_{LI} or between 3,500_{MI} and 7,000_{MI}. The point of time where HFP was for the first time higher than at the end of exercise, was observed at the

first minute (3,500_{LI} and 7,000_{LI}, $P < 0.001$) of the recovery, at the fourth (3,500_{MI}, $P < 0.05$) and fifth minute (7,000_{MI}, $P < 0.05$) of the recovery and at the end of the 30-min recovery (3,500_{HI}, $P < 0.01$).

The increased intensity of the exercise resulted in higher RF after 3,500_{MI} ($P < 0.01$) and 3,500_{HI} ($P < 0.01$) when compared to 3,500_{LI} during each of the five recovery minute. The prolonged running distance had no influence on RF. A significant decrease within exercises, when compared to the preceding minute, was found during the first two (3,500_{MI} and 7,000_{MI}) or three (3,500_{LI}, 3,500_{HI}, 7,000_{LI}) minutes of the recovery, after which there were no further recovery. The dynamics of RF at the end of the exercise and during the acute recovery are presented in Fig. 4

HRV at the end of the 30-min recovery

Comparisons between the three different intensities showed that HR (Fig. 1) was higher, and TP (Fig. 2) and HFP (Fig. 3) were lower (ANOVA, $P < 0.001$) after 3,500_{MI} ($P < 0.01$) and 3,500_{HI} ($P < 0.01$) when compared to 3,500_{LI}. There were no differences between the low-intensity exercises (3,500_{LI} and 7,000_{LI}) or between the moderate-intensity exercises (3,500_{MI} and 7,000_{MI}) at the end of the recovery. TP (Fig. 2) and HFP (Fig. 3) were recovered to the resting level after 3,500_{LI} and 7,000_{LI}.

Discussion

The main purpose of the present study was to find out the HRV dynamics, especially vagal reactivation, during the

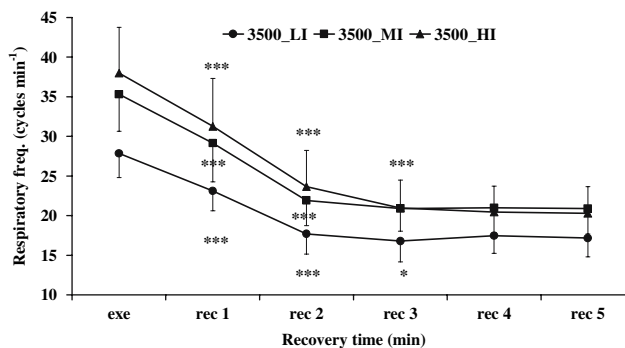


Fig. 4 The effect of exercise intensity on respiratory frequency at the end of the exercise and during the first 5 min of the recovery after 3,500_{LI}, 3,500_{MI} and 3,500_{HI}. 3,500_{LI} = 3,500 m at 50% vVO_{2max} , 3,500_{MI} = 3,500 m at ~63% vVO_{2max} , 3,500_{HI} = 3,500 m at ~74% vVO_{2max} . Significant differences within exercises when compared to the preceding minute. * $P < 0.05$, *** $P < 0.001$. The graph of 7,000_{LI} and 7,000_{MI} are not presented, since they were similar to 3,500_{LI} and 3,500_{MI}

first 5 min of the recovery after five different endurance exercises. We used STFT method to be able to analyze HRV during the immediate recovery, where rapid changes occur in cardiac function. More specifically, we wanted to find out the effects of increased intensity as well as effects of prolonged running distance on post-exercise HRV.

In the present study, we found HFP and TP to be significantly higher at the end of the low-intensity (50% vVO_{2max}) exercises when compared to moderate- (~63%) and high-intensity (~74%) exercises. This finding supports the findings of e.g. Perini et al. (1990), Casadei et al. (1995) and Tulppo et al. (1998), who have found HRV almost to disappear at the exercise intensities exceeding 50–60% VO_{2max} . Since the moderate- and high-intensity exercises were performed at higher intensities than 60% VO_{2max} , it was expected that there was barely any HRV left at the end of these exercises.

The main findings of the present study were that HRV started to increase during the first 5 min of the recovery and that the increased intensity of the exercise (from 50→63→74% vVO_{2max}) caused slower HFP recovery and also lower HFP and TP levels during the first 5 min of the recovery when compared to the low-intensity exercise. The doubled running distance (from 3,500 to 7,000 m) at the intensities of 50 and 63% vVO_{2max} had no influence on HRV recovery.

Immediate recovery of HRV

As it is well known, fast changes occur in cardiac function during the immediate recovery after exercise. Possible mechanisms inducing the immediate changes in cardiac function include e.g., fast changes in cardiac pre-load, after-load and contractility of the heart (Miles et al. 1984; Plotnick et al. 1986). Combined with the loss of central command and baroreflex activation, these mechanisms contribute to fast vagal reactivation after exercise cessation (e.g., Arai et al. 1989; O'Leary 1993; Oida et al. 1997). An increase in vagal activation as well as a decrease in sympathetic activation has been detected already during the first minutes of the recovery e.g., in the blockade study of Savin et al. (1982), in which the recovery of heart rate after peak exercise was investigated.

We found a significant decrease in HR and an increase in TP during the first minute of the recovery after each of the five exercises. Instead, HFP increase during the first minute was detected only after the two low-intensity exercises, whereas the increase after the moderate-intensity exercises was observed after the fourth (3,500_{MI}) and the fifth (7,000_{MI}) minute of the recovery. Previous studies investigating the immediate recovery after exercise have found parallel results to ours, although the exact

comparison of the results cannot be made because of different analysis methods. In the study of Martinmäki and Rusko (2007), the intensity of the exercises affected significantly to the immediate HFP recovery dynamics, as well as to the level of HFP, similarly to our study. Also Casties et al. (2006) found slightly increased HFP during the first 10 min of the recovery after a graded, high-intensity exercise (up to 90% vVO_{2max}). However, the difference in HFP was not significant because they compared the average of the first 10 min to the value at the end of exercise. Since we used averages of 1 min, the results of Casties et al. (2006) cannot be directly compared to ours. The results of the present study are in agreement with the blockade study of Goldberger et al. (2006), who investigated HRV immediately after a maximal bout of exercise. They found increasing time-domain parameters of HRV, root mean square successive difference of the R–R intervals (RMSSD) and root mean square residual (RMS), during the first 5 min of the recovery. Based on their blockade findings, Goldberger and colleagues concluded that these HRV parameters indicated parasympathetic reactivation after exercise.

The results of our previous study (Kaikkonen et al. 2007) on athletes showed no recovery of HFP during the first 5 min of the recovery, when the exercise intensity exceeded 80% vVO_{2max} . HFP dynamics after the moderate- and high-intensity exercises of the present study on untrained are similar to those, since we found only minor recovery of HFP during the first 5 min of the recovery after 3,500_{MI} and no recovery after 3,500_{HI}. According to the present results, after exercises exceeding 50–60% vVO_{2max} , the recovery of cardiac autonomic modulation is significantly slower when compared to exercises performed at the intensity of 50% vVO_{2max} . This suggests that when HRV decreases close to zero during the exercise, the recovery pattern is also delayed.

In the present study, the results of HFP and TP were different, suggesting that the origins of these parameters are slightly different. The recovery pattern of HFP separated the exercises performed at different intensities, but this kind of differentiation was not detected in TP recovery. While HFP has been found to indicate changes in vagal modulation (Akselrod et al. 1981; Berntson et al. 1993; Cacioppo et al. 1994, Goldberger et al. 2006, Martinmäki et al. 2006a), the origin of TP is more controversial. Although TP has been suggested to indicate mainly the changes in vagal modulation at rest (Task Force 1996), the contribution of the LFP must be taken into account when discussing TP results. One plausible explanation for the different results is that during recovery from high-intensity exercise, TP consists also of low frequency variation and therefore indicates overall changes in autonomic modulation rather than changes in vagal modulation only.

According to the blockade study of Goldberger et al (2006), the traditional (RMSSD) and also the new (RMS) index of HRV indicate changes in vagal activity during the immediate recovery after exercise. Since the time domain parameter RMSSD correlates strongly with HFP (Task Force 1996), we believe that HFP of the present study indicates changes in vagal modulation better than TP.

We also measured RF dynamics during the immediate 5-min recovery, making it possible to compare the recovery of HFP and RF, since breathing frequency may have an impact on HFP (Grossman et al. 1991; Keselbrener and Akselrod 1996). We found different recovery patterns for HFP and RF during the immediate recovery period, since the fast decrease of RF was found during the first 3 min, similarly after each exercise, but the recovery of HFP was different after low-, moderate- and high-intensity exercises. This suggests that the changes in HFP during the recovery represent true changes in vagal modulation rather than are caused by changes in RF.

Effects of increased intensity and prolonged distance

One of our interests was to compare the effects of increased exercise intensity and prolonged duration on post-exercise HRV recovery. The effect of exercise intensity has been investigated to some extent, but no studies concerning the effects of prolonged duration of the exercise on HRV recovery have been published. We found decreased HRV after moderate- and high-intensity exercises when compared to low-intensity exercises. The difference between exercises was notable during the whole 30-min recovery, both in HFP and in TP. Similar results have been reported by e.g., Parekh and Lee (2005), who found decreased HFP and TP after high-intensity (HI = 80% VO_2 reserve) exercise when compared to low-intensity (LO = 50% VO_2 reserve) exercise. Similarly to our results, they found the difference between LO and HI exercises to be more obvious in $\ln\text{HF}$ than in $\ln\text{TP}$. Also Terziotti et al. (2001) have found lower HFP during recovery after high-intensity exercise (80% of anaerobic threshold) when compared to low-intensity exercise (50% of anaerobic threshold). The results of the present study suggest that the increased intensity of exercise resulted in a disturbance of autonomic functions, even though the total work load (= running distance) of the exercises at different intensities was the same.

An interesting finding was that the doubled running distance of the low- and moderate-intensity exercises did not have an influence on HRV recovery. This finding could be partly expected based on the results of blood lactate, oxygen uptake and RPE, which were quite similar between the exercises performed at the same intensity. However, we

do not know the effects of even more prolonged exercises on HRV, during which the levels of stress hormones may increase.

Limitations

The lack of control of the breathing could be seen as a limitation of this study. However, although both tidal volume and breathing frequency may have an impact on HFP at rest (Grossman et al. 1991; Keselbrener and Akselrod 1996). Bartels et al. (2004) concluded that HFP during exercise represents true cardiovascular autonomic modulation, since they found no differences in HFP at rest at the RF between 15 and 32 cycles/min, but during the exercise at the same breathing frequencies, HFP decreased with increasing intensity and RF. In addition, the main focus of the present study was to clarify HRV dynamics during recovery, when RF is lower than during exercise. Controlled breathing could have disturbed the real autonomic modulation during the recovery. Therefore, taking also into account the different recovery dynamics of RF and HFP of the present study, we believe that HFP recovery during the immediate recovery is not dependent on RF. The subjects of the present study were women, in contrast to most of the studies of HRV. However, since the 30-min HRV recovery was comparable with previous studies on men, we expect that the present findings on the fast HRV recovery are similar in men.

Conclusions

The purpose of the present study was to find out HRV dynamics, especially vagal reactivation, during the immediate recovery after different constant-load exercises. We found increased HFP during the first 5 min of the recovery after each exercise, except for 3,500_{HI}, suggesting fast vagal reactivation after the exercise cessation. The recovery of HFP was faster after low-intensity exercises. We also found that the exercise intensity affected significantly the post-exercise HRV level, but no influence of the doubled running distance of the exercise on the HRV recovery was observed.

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