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Cardiac Autonomic Control Immediately after Exercise in Female Distance Runners

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Abstract The purpose of this study was to examine the timing of recovery of the high-frequency (HF) component of heart rate variability (HRV) after exercise in female distance runners using frequency analysis of HRV. Twenty-two female young (n=11; YG) and middle-aged (n=11; MG) distance runners participated in this study. The two groups performed incremental cycle exercise with progressive intensity until exhaustion. The R–R intervals were processed by the maximum entropy method for determination of HF power on successive 7-second segments of 70 seconds of the recovery period. In the YG, the HF power of the second 7-sec segment showed significantly higher values than the 7 sec before cessation of exercise ($p<0.005$), whereas the MG exhibited significantly higher values in the third segment ($p<0.005$). The YG indicated significantly higher HF power than the MG in the fifth segment ($p<0.0045$). These findings suggested the occurrence of parasympathetic reactivation at an earlier period compared to the previous findings. Multiple influences of various factors including the subjects' characteristics to HF recovery were suggested. However, the detection of the timing of HF recovery despite the duration of sharp change in HR indicated that HRV was an effective evaluation technique for determination of autonomic control immediately after exercise.
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Introduction

The heart rate (HR) is antagonistically controlled by the sympathetic activity of the autonomic nervous system and the parasympathetic branch of the vagus nerve. The increase in HR during exercise is brought about by the inhibition of the vagus nerve and the activation of the sympathetic nervous system. In contrast, HR recovery after exercise is linked to the inhibition of sympathetic nerve activity and parasympathetic reactivation (Arai et al., 1989; Imai et al., 1994; Javorka et al., 2003; Kannankeril et al., 2004; Pierpont et al., 2000). Imai et al. (1994) reported that the HR recovery 30 seconds immediately after exercise was strongly influenced by the vagus nerve activity, not by the decrease in sympathetic nerve activity. Kannankeril et al. (2004) also reported that cardiac parasympathetic nerve activity increased one minute after exercise. The results of these previous studies (Imai et al., 1994; Kannankeril et al., 2004) suggest that reactivation of parasympathetic nervous activity after exercise occurs to a considerable degree during the early recovery period immediately after exercise. However, it does not seem that the timing has been fully examined.

Heart rate variability (HRV) is widely used as an index to assess cardiac autonomic nervous activity noninvasively. Many researchers have examined the autonomic nervous activity during and after exercise using frequency analysis of HRV (Arai et al., 1989; Perini et al., 1990; Yamamoto et al., 2001). Since conventional methods, such as the fast Fourier transform (FFT) and autoregressive (AR) techniques, need stationary data, they cannot be applied to the duration of sharp change in HR immediately after exercise (Goldberger et al., 2006). Recently, newer methods, such as the maximum entropy method (Macor et al., 1996; Murasato et al., 1998; Sumi et al., 2006) and the complex demodulation method (Hayano et al., 1994), which do not depend on stationary data, have been developed. These newer methods make it possible to assess autonomic nervous activity based on a short-term R–R interval. Since the lower frequency limit of the high-frequency (HF) component of HRV, which is the index of cardiac parasympathetic nervous activity (Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology, 1996), is 0.15 Hz, the required shortest time-series data length to assess HF power is approximately 7 seconds (1 sec/0.15). By analyzing a 7-second segment using the newer methods, it is possible to evaluate HF

power during sharp change in HR.

Exercise training increases parasympathetic tone (Levy et al., 1998), and high aerobic capacity is associated with fast HR recovery after exercise (Darr et al., 1988). Distance running is one of the typical endurance exercises that improve aerobic capacity. Nevertheless, HRV immediately after exercise in distance runners is not well understood. In addition, very little is known about the autonomic control of post-exercise heart rate in trained female subjects (Du et al., 2005). The possible factors related to post-exercise HR recovery are age and athletic level (Buchheit and Gindre, 2006; Darr et al., 1988; Lipsitz et al., 1990; Shannon et al., 1987; Yamasaki et al., 1996). Darr et al. (1988) reported that trained subjects, irrespective of age, demonstrated a significantly faster HR recovery than untrained subjects. Increasing age resulted in a slower decline of heart rate after exercise (Kostis et al., 1982). Therefore, post-exercise HR recovery of a young group with high athletic levels is influenced by the status of age and athletic level. It is expected that the post-exercise HR will recover earlier than that of older runners. The significance of this study is to find the timing of parasympathetic reactivation after exercise using a newer method and to obtain newer findings on the cardiac autonomic modulation in female distance runners.

This study aims to assess the cardiac autonomic nervous activity immediately after exercise in female distance runners by using the newer method of frequency analysis of HRV. It is hypothesized that the timing of recovery of HF power can be distinguished by analyzing a shorter time segment than was done in previous studies (Imai et al., 1994; Kannankeril et al., 2004), even in the change in HR with time immediately after exercise. Furthermore, the timing of recovery of HF power in highly trained young female runners is faster than in well-trained middle-aged female runners.

Methods

1. *Subjects*

Twenty-two healthy young (n=11) and middle-aged (n=11) female subjects participated in this study. The young group (YG) was composed of highly trained collegiate long distance runners. The middle-aged group (MG) included well-trained regional runners.

No subjects were on any regular medication, including hormone replacement therapy. No one was diagnosed with diabetes and hypertension, and none were smokers. The subjects were asked to refrain from strenuous exercise and caffeine consumption for at least 48 hours and 24 hours before the test, respectively. The subjects were informed about the aim of the study and its possible risks, and all gave their written informed consent before entering the study. This study protocol was approved by the Human Subjects Committee at Nagoya City Rehabilitation & Sports Center.

2. *Exercise protocol*

An incremental cycle exercise was carried out in a quiet room maintained at constant temperature (22–24°C). The subjects performed the test in upright position on an electronically braked ergometer (Well Bike BE-360, Fukuda Denshi, Tokyo, Japan). At first, the subjects rested in a supine position for 20 min. Then, the subjects rode on the ergometer. Seat and handlebar heights were set for each subject and kept constant during the test. Following a 3-min rest, the subjects performed a 2-min warm-up pedaling at 0 W, and then exercised with progressive intensity until a subject could no longer maintain the pedaling rate (volitional exhaustion). The work load was increased by $20\,W \cdot min^{-1}$. The pedaling frequency was set at $50\,rev \cdot min^{-1}$. Immediately after cessation of exercise, the subjects were instructed to stop pedaling and to stay on the ergometer for 2 min. No attempt was made to control breathing frequency during and after the test. All tests were conducted in a controlled environment with proper experimental design. During the tests, all subjects were mentally and physically fit.

Oxygen uptake was obtained using an Oxycon-α automatic online breath-by-breath system (Jaeger, Mijnhard bv, Netherlands). The system is adapted for expired and inspired volumes. Breath-by-breath data were averaged to provide one data point for each 30-sec period. This was subjected to a three-way calibration process, involving a flow volume sensor, gas analyzer, and delay time calibration. The flow volume sensor calibration ensures that a measuring system of the Oxycon (consisting of the amplifier, Triple V, and pressure transducer) is functioning correctly. A calibrated 3-L syringe connected to the Triple V assembly was used. A series of six complete pumps of the syringe was repeated until the percent difference between the current and previous volume calibration was less than 1%. The gas analyzer and delay time calibration involved an automated calibration procedure (Carter and Jeukendrup, 2002). The criteria for achievement of maximal oxygen uptake ($\dot{V}O_{2max}$) included all the following measures: 1) Leveling off of oxygen uptake despite a further progression of the exercise load (<150 ml/min increase in oxygen uptake), 2) final respiratory exchange ratio of >1.1, and 3) visible exhaustion. In this study, considering the difficulty of estimating maximal HR (Gellish et al., 2007), the age-predicted maximal HR was not adopted as a criterion for $\dot{V}O_{2max}$. Breathing rate (BR) was measured continuously. Throughout the test, determination of 12-lead electrocardiogram (ECG) was continuously digitized at 4 kHz (ML-5000, Fukuda Denshi, Tokyo, Japan). HR was measured and recorded with ECG monitoring. Cuff blood pressures were obtained every minute with an indirect automatic manometer (STBF-680F, Collin Denshi, Aichi, Japan).

3. *Measurement of cardiac autonomic activity*

In order to obtain the R–R interval of each subject, ECG (CM5) was recorded (LRR-03, GMS, Tokyo, Japan) over a period extending from the supine rest up to the end of

the recovery. The measured R–R interval time series data were then transferred by an A/D converter (AD12-8(PM), CONTEC, Osaka, Japan) from the ECG to a personal computer (VAIO VGN-T91PSY, SONY, Tokyo, Japan) with a sampling rate at 1 kHz. Based on the R–R interval time series data, a time series analysis system (MemCalc, Suwa Trust, Tokyo, Japan) was then used to obtain the power spectral distribution of HRV.

MemCalc is a computer program used to calculate spectrum based on the maximum entropy method. The procedure overcomes the disadvantage of the conventional spectral analysis in the frequency domain, such as poor resolution and insufficiency for estimating short time-series data in FFT and AR methods (Ohtomo et al., 1994; Radoski et al., 1976). MemCalc can also detect proper frequency even from data of the length corresponding to only one period (Ohtomo et al., 1994; Ohtomo et al.,1995; Tsuchida et al., 1998).

In order to filter ectopic beats in this system, we extracted the R waves. First, we recognized the wave patterns, and then removed the values, which were not adequate as human R–R intervals (less than 272 msec, more than 3000 msec). If the ectopic beats were mixed in between normal R waves, they were removed and made primary R–R intervals. In this study, the total numbers of R–R intervals removed from each subject's original data were 22.5±18.82 in the YG and 6.5±7.61 in the MG. The outliers of the original data were 0.9±0.72% in the YG and 0.2±0.24% in the MG. Since the R–R interval time series data were unequally spaced, they were made even. The function of MemCalc is to interpolate data to even it out. That is, it connects anteroposterior data dots linearly and sets the height of the dot to where the straight line crosses the vertical line. The latter was taken at the observed time and observed value. We resampled the data at even intervals from the R–R interval time series data, which became a continuous function. The resampling frequency at rest was 1 Hz, and the data during exercise and recovery was 3.33 Hz.

The resting value was calculated as the average value taken over a 3-min period in sitting position on the cycle ergometer prior to exercise. To elucidate the detailed structure of HRV during recovery, segment time series analysis was carried out. Previous studies described the increase in parasympathetic nervous activity between 30 sec and one minute immediately after exercise (Goldberger et al., 2006; Imai et al., 1994; Kannankeril et al., 2004). Therefore, we divided the original time series from 7 sec before cessation of exercise to 70 sec of the recovery period into a sub-series of ten 7 sec segments.

The HF power spectrum (ms^2) was set as the sum of the power from 0.15 to 0.4 Hz at rest. The HF power from the peak of exercise to the end of the recovery period was set between 0.15 and 1.0 Hz, depending on the subjects. The upper limit of the frequency range was altered by respiratory frequency because HF power was influenced by respiratory activity (Hirsh and Bishop, 1981). During high-intensity exercise, the central frequency responded to respiratory frequency even if total power was low (Sumi et al., 2006). In this study, we considered that total power immediately after exercise was as low as that during exercise and confirmed the central frequency in the power spectrum. Furthermore, BR during the test was measured by automatic an online breath-by-breath system. The peak frequency range of HF power was determined based on the results of these calculations.

4. *Statistical analysis*

No subjects were eliminated from the analysis by virtue of the irregularities in the ECG during the recovery period. HR was calculated using the average R–R intervals in each segment. Characteristics, HR, and BR were expressed as mean±SD. HF was expressed as median ($1^{st}-3^{rd}$ quartile). A Student's unpaired t-test was used for the comparison of characteristics of subjects. Serial changes in HR and BR were evaluated by 2 (groups)×11 (time points) repeated measures ANOVA. Analyses that did not meet Mauchley's sphericity criteria were interpreted using the Greenhouse-Geisser correction for the inflated risk of a type I error (Ludbrook, 1994). Post-hoc testing using a Bonferroni adjustment was used to assess specific differences between 7 sec before cessation of exercise and 70 sec of the recovery period, and between-group comparisons at the same time points. The results of Lilliefors test revealed that no normality concerning HRV parameters was obtained, so that non-parametric tests were used. As regards resting value and each segment of HF power between 7 sec before cessation of exercise and 70 sec of the recovery period, the comparison between both groups was performed using a Mann-Whitney U test. A Wilcoxon signed-rank test with Bonferroni correction was employed in the pair-wise comparisons between 7 sec before cessation of exercise and each successive time point in each group. The significant difference between the proportions of the tenth 7 sec segment of HF (HF_{63-70}) to the resting value in both groups was assessed using a chi-square test for independence. The level of significance was set at $p<0.05$. Data were analyzed using StatView J5.0 and SPSS 15.0J for Windows.

Results

The characteristics of the subjects are presented in Table 1. The subjects of the YG were significantly younger than the MG subjects ($p<0.0001$). $\dot{V}O_{2max}$ was significantly higher in the YG ($p<0.001$). HR at rest was significantly lower in the YG ($p<0.01$). HF power at rest of the YG was significantly higher than for the MG ($p<0.01$). Training experience was significantly longer in the MG ($p<0.0001$), but current training status was significantly higher in the YG ($p<0.0001$).

There was a significant groups×time points interaction for HR [F (2.390, 47.794)=26.100; $p<0.001$; Greenhouse-Geisser correction applied; Fig. 1]. The HR in both groups decreased immediately after exercise. In the YG, HR of the second 7-sec segment (HR_{7-14}) indicated a significantly lower level than HR_{peak} ($p<0.005$), whereas, in the MG, HR_{14-21} demonstrated a significantly lower level than HR_{peak} ($p<0.005$). HR_{peak} was

Table 1. Characteristics of subjects

	YG (n=11)	MG (n=11)
Age (yrs)	19.8±0.87****	57.1±7.17
Mass (kg)	47.3±6.24	47.0±4.07
Height (cm)	160.9±5.18**	153.0±5.28
$\dot{V}O_{2max}$ (ml/min/kg)	57.6±4.91***	42.0±11.20
HR at rest (bpm)	56.5±7.31**	66.5±9.00
HF power at sitting rest (ms^2)	650.8 (300.92−750.81)**	150.6 (100.83−250.50)
Training experience (yrs)	7.8±0.87	23.3±4.10****
Training frequency (sessions/week)	6.9±0.30****	4.0±1.10
Training time (min/session)	155.5±32.36****	58.2±26.77

Values are means±SD or median (1st quartile–3rd quartile); YG, young group; MG, middle-aged group; $\dot{V}O_{2max}$, maximal oxygen uptake; HR, heart rate; HF, high-frequency component of heart rate variability. *p<0.05, **p<0.01, ***p<0.001, ****p<0.0001 significant differences between two groups.

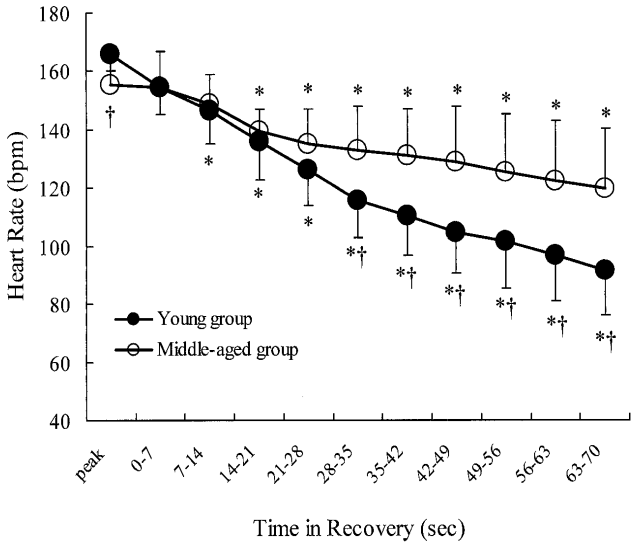

Fig. 1. Mean and standard deviation of heart rate at peak exercise and first 70 seconds of recovery for the young group (n=11) and middle-aged group (n=11). The results from the repeated-measures two-way ANOVA for the heart rate data demonstrate that there was a significant group×time interaction (F (2.390, 47.794)=26.100; p<0.001; Greenhouse-Geisser correction applied). Alpha levels were adjusted by the Bonferroni technique for comparisons at ten time points for specific difference between peak and 70 sec of the recovery period (i.e., *p<0.05/10=0.005) and eleven time points for between-group differences (i.e., †p<0.05/11=0.0045).

significantly higher in YG (p<0.0045). A significant difference between groups was revealed in HR_{28-35} (p<0.0045). Thereafter, the YG similarly demonstrated significantly lower values (p<0.0045). HR_{63-70} demonstrated 91.5±15.34 (bpm) in the YG and 119.5±20.83 (bpm) in the MG. The ratio of HR_{63-70} to HR_{peak} was 55.1±8.48% in the YG and 76.6±9.74% in the MG.

The change with time in HF power is shown in Table 2. In the YG, the HF power of the second 7-sec segment (HF_{7-14}) showed a significantly higher value than 7 sec before cessation of exercise (p<0.005). Thereafter, significantly higher values were similarly demonstrated (p<0.005). The MG exhibited significantly higher values after HF_{14-21} (p<0.005). The proportions of HF_{63-70} to the resting value were 76.3% (496.5/650.8) in the YG and 5.8% (8.7/150.6) in the MG. This difference was statistically significant (χ^2=84.001, p<0.001). In the comparison between both groups, the YG indicated significantly higher values after HF_{28-35} (p<0.0045).

There was a significant groups×time points interaction for BR [F (2.949, 58.979)=29.244; p<0.001; Greenhouse-Geisser correction applied; Table 3]. BR after exercise in both groups demonstrated similar values to HF. In the YG, BR of the second 7-sec segment (BR_{7-14}) showed a significantly lower value than BR_{peak} (p<0.005). Subsequently, the reduction was continued (p<0.005). In the MG, BR_{14-21} showed a significantly lower value than BR_{peak} (p<0.005). Subsequently, the reduction was continued as well (p<0.005). In the comparison between both groups, there was no significant difference in BR_{peak}. The YG indicated significantly lower values in BR_{7-14} (p<0.0045). Thereafter, lower values were similarly demonstrated until BR_{63-70} (p<0.0045).

Discussion

The major findings of this study are as follows: 1) We were able to comprehend clearly the change in HF power during the duration of the sharp change in HR immediately after exercise using the newer frequency analysis of HRV based on the maximum entropy method and 2) the timing of the recovery of HF power immediately after exercise in the YG was faster than in the MG. As far as we know, this study reveals for the first time the detection of HRV recovery immediately after exercise by analyzing the shortest segment of time.

HRV indices immediately after exercise

In this study, HF power in the YG started to rise between 7 and 14 seconds after exercise. On the other hand, HF power in the MG increased between 14 and 21 seconds. These results suggested the occurrence of parasympathetic reactivation at an earlier period compared to the 30 seconds or one minute after exercise described in previous studies (Goldberger et al., 2006; Imai et al., 1994; Kannankeril et al., 2004). Immediately after exercise, the autonomic nervous activity begins to change (Kannankeril and Goldberger, 2002). That is, during exercise

Table 2. High-frequency component of heart rate variability immediately after exercise

Time segment (sec)	HF power (ms^2)		*p* value		
	YG (n=11)	MG (n=11)	vs. Peak (YG)	vs. Peak (MG)	YG vs. MG
Peak	1.8 (1.35–4.44)	1.5 (0.70–6.00)	—	—	0.467
0–7	4.1 (2.47–5.06)	1.8 (0.87–6.05)	0.269	0.080	0.166
7–14	6.4 (2.64–9.56)	1.7 (1.35–6.75)	0.001*	0.189	0.053
14–21	7.4 (3.81–20.37)	2.3 (1.54–6.01)	0.001*	0.002§	0.018
21–28	21.9 (5.15–93.64)	2.8 (1.54–6.77)	0.002*	0.001§	0.027
28–35	78.5 (8.64–146.85)	4.9 (1.93–8.60)	0.001*	0.001§	0.003†
35–42	272.3 (12.09–340.10)	5.4 (1.90–15.03)	0.001*	0.001§	0.002†
42–49	271.4 (20.98–666.84)	6.4 (1.58–10.05)	0.001*	0.001§	<0.001†
49–56	264.7 (46.10–664.70)	6.9 (4.87–14.90)	0.001*	0.001§	<0.001†
56–63	352.3 (121.21–671.84)	7.0 (2.49–16.29)	0.001*	0.001§	<0.001†
63–70	496.5 (172.98–800.68)	8.7 (2.30–13.95)	0.001*	0.001§	<0.001†

Values are median (1st quartile–3rd quartile); HF, high-frequency component of heart rate variability; YG, young group; MG, middle-aged group. *p* values are from Wilcoxon signed-rank test with Bonferroni correction for the pairwise comparisons between 7 sec before cessation of exercise (Peak) and each successive time point and Mann-Whitney U test with Bonferroni correction for the comparison between both groups.* Significant difference from Peak in the young group ($p<0.05/10=0.005$). § Significant difference from Peak in the middle-aged group ($p<0.05/10=0.005$). † Significant difference between two groups ($p<0.05/11=0.0045$).

Table 3. Breathing rate immediately after exercise

Time segment (sec)	Breathing rate (times/min)		*p* value		
	YG (n=11)	MG (n=11)	vs. Peak (YG)	vs. Peak (MG)	YG vs. MG
Peak	52.6±5.95	47.6±8.14	—	—	0.118
0–7	48.8±5.04	46.9±6.03	0.602	1.000	0.417
7–14	29.1±3.13	46.5±7.61	<0.001*	0.744	<0.001†
14–21	27.7±2.82	37.9±3.75	<0.001*	<0.001§	<0.001†
21–28	26.3±4.07	35.8±3.37	<0.001*	<0.001§	<0.001†
28–35	24.9±3.13	33.8±3.37	<0.001*	<0.001§	<0.001†
35–42	24.6±3.45	32.2±3.40	<0.001*	<0.001§	<0.001†
42–49	24.1±2.82	31.4±3.37	<0.001*	<0.001§	<0.001†
49–56	23.8±2.51	30.5±3.24	<0.001*	<0.001§	<0.001†
56–63	23.2±1.88	29.7±3.31	<0.001*	<0.001§	<0.001†
63–70	23.0±1.68	28.7±3.13	<0.001*	<0.001§	<0.001†

Values are means±SD; YG, young group; MG, middle-aged group. The results from the repeated-measures two-way ANOVA for the breathing rate data demonstrated that there was a significant group×time interaction (F (2.949, 58.979)=29.244; $p<0.001$; Greenhouse-Geisser correction applied). Alpha levels were adjusted by the Bonferroni technique for comparisons at ten time points for specific difference between 7 sec before cessation of exercise (Peak) and 70 sec of the recovery period in the young group (i.e., *$p<0.05/10=0.005$) and middle-aged group (i.e., §$p<0.05/10=0.005$), and eleven time points for between-group differences (i.e., †$p<0.05/11=0.0045$).

the decrease in parasympathetic nervous activity and the increase in sympathetic nerve activity result in the rise of HR. In contrast, after exercise, HR is decreased by interaction between the inhibition of the sympathetic nerve activity and parasympathetic reactivation (Goldberger et al., 2006; Pierpont and Voth, 2004). However, there is no agreement of findings concerning the timing of change of the autonomic nervous system. The previous studies stated that vagus nerve activity was reactivated immediately after exercise (Arai et al., 1989; Imai et al., 1994) and was associated with the exponential decline of cardiac activity (Nishime et al., 2000; Perini et al., 1989). On the other hand, Savin et al. (1982) reported that the reduction in sympathetic nerve activity contributed greatly to HR recovery immediately after exercise and parasympathetic nervous system reactivation appeared after HR reached lower levels.

We think that the methods and segment of analysis are different from the findings of these studies. The previous studies examined a segment data setting after exercise between 15 and 60 seconds (Goldberger et al., 2006; Imai et al., 1994). In the current study, HR decreased up to 55.1% of HR_{max} in only 70 seconds after exercise in the YG and 76.6% in the MG. Therefore, in order to assess the cardiac autonomic modulation mechanism, which changes from moment to moment, data length for analysis should be as short as possible. The 7-sec segment used in our study is the shortest data length to detect HF using frequency analysis. Heretofore, HR recovery has been a clinically important index to evaluate possible mortality

caused by myocardial ischemia (Cole et al., 2000; Nishime et al., 2000). However, HR recovery cannot be used as an index of the assessment of parasympathetic reactivation because it is influenced by sympathetic and parasympathetic nervous activity (Kannankeril et al., 2004). On the other hand, since the conventional techniques such as FFT and AR need stationary data, they could not be used during recovery after exercise. Therefore, validity is doubtful in the assessment of cardiac autonomic modulation mechanisms using these techniques. Recently, Kaikkonen et al. (2007), and Martinmäki and Rusko (2008) investigated the acute recovery of HRV by analyzing minute-by-minute values immediately after different intensity exercises using the Short-time Fourier transform, which is an extension of the FFT. However, spectral powers were averaged for each successive 60-sec period in the methods of the aforementioned study. We still believe that a precise description of HRV dynamics immediately after exercise cannot be provided through such a long time segment (i.e., 1 min). Goldberger et al. (2006) recently examined HRV using the time-domain method and revealed that the effect of the parasympathetic nervous system appeared 30 seconds after exercise. We proved that HF power increases within 30 seconds after exercise in both groups. It indicates that frequency analysis of HRV using the maximum entropy method can also detect aspects of change in autonomic nervous activity after exercise.

In this study, HRV was observed for 70 seconds after exercise, which is a shorter time compared to previous studies, e.g., Martinmäki and Rusko (2008), which observed HRV for 10 minutes after exercise and Kaikkonen et al. (2007) and Goldberger et al. (2006), which observed HRV for 5 minutes after exercise. This is why this study aims to find the mutation point when HF power begins to increase, which is demonstrated at a near-zero level during exercise. HF recovery of both groups for 70 seconds shows a small value. HF after 70 seconds demonstrates 76.3% at rest in the YG and only 5.8% in the MG. However, the discovery of the mutation points of both groups will provide a basis to predict modulation of HR recovery after exercise. Early recovery of HF immediately after exercise could be observed because well-trained subjects were examined in this study. The characteristics of training and age of the subjects require a longer analysis time.

The time point that indicated a significant increase or decrease in each peak of HR and HF after exercise in both groups was the same. (Reference to Fig. 1 and Table 2). The strong association of parasympathetic nervous activity with HR recovery immediately after exercise is suggested. This result supports the previous study (Imai et al., 1994; Kannankeril et al., 2004). In addition, the time point that indicated significant increase or decrease in each peak of HF and BR in both groups was also the same. This result supports the idea that HF is synchronous with a change in tidal volume or breathing frequency (Hirsh and Bishop, 1981). It is confirmed that the main driver of HRV even immediately after exercise is respiration.

Factors influencing the timing of HF recovery

In this study, HF power in YG started to recover earlier than in MG. The result suggests that factors such as age, physical fitness, and training status influence the timing of HF recovery in female distance runners. Previous studies showed the influence of age on parasympathetic modulation of HR by estimating HF power (Stratton et al., 2003). Darr et al. (1988) indicated that trained subjects demonstrated a significantly faster HR recovery than untrained subjects, which was particularly marked during fast-phase recovery. Buchheit and Gindre (2006) reported a significant relationship between HR recovery and training load estimated by the Baecke sport score. Therefore, multiple influences of these factors to HF recovery after exercise are suggested. A future examination is required to examine the factor which reflects most strongly the timing of HF recovery or the presence of other factors.

The validity of the method used in our study was verified by our investigation of possible differentiation of HF recovery in the subject groups with different properties. With respect to the occurrence of parasympathetic reactivation after exercise, it remains unclear because the time varies wildly from 30 seconds (Imai et al., 1994) to one minute (Kannankeril et al., 2004). In this study, we were able to detect the timing of reactivation with more detailed scales by measuring HF recovery as one segment for 7 seconds compared to previous studies. In spite of the differences in age and athletic level among the subjects, the difference in timing of HF recovery was just one segment (YG: HF_{7-14}, MG: HF_{14-21}). However, this is not a small difference because HF is influenced by respiration (Hirsh and Bishop, 1981). The BR of YG was approximately 3.4 times (29.1/60*7) for the 7-sec segment of HF_{7-14}, while that of MG was approximately 4.4 times (37.9/60*7) for the 7-sec segment of HF_{14-21}. Considering the results, a difference up to 14 second in time point when the cardiac vagus nerve activity of both groups started to reactivate is possible. Therefore, the detection of further detailed differences remains to be solved. However, the method used in this study showed validity in evaluating cardiac autonomic nervous activity because we could detect more detailed timing of the reactivation and the difference between two groups with different characteristics.

Methodological limitations

This study has discovered three methodological limitations. First, HRV indicates variability of the R–R interval, not an index for determination of cardiac autonomic nervous activity quantitatively. Parasympathetic blockade by using atropine is a standard method to evaluate parasympathetic activity (Berntson et al., 1997; Vukajlovic et al., 2006). However, this study does not clarify the influence of medical agents. Therefore, as regards this context, we have to wait for future physiological verification for the HRV index in autonomic nervous activity immediately after exercise, including the influence of medical agents.

Second, reflex cardiac rate regulation immediately after

exercise was not examined. Tahara et al. (2005) indicated a reperfusion reflex immediately after exercise. It is also indicated that direct stimulation of the central chemoreceptor by CO_2 increases the amplitude of respiratory arrhythmia (Péronnet and Aguilaniu, 2006). Therefore, this study indicates that HF power is influenced not only by vagus nerve activity but also by reflex enhancement.

Third, the LF (sum of the power from 0.04 to 0.15 Hz)/HF ratio reflects the balance of sympathetic and parasympathetic nervous activity (Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology, 1996). In the present study, a 7-sec segment is analyzed in order to examine parasympathetic reactivation. The segment data of 25 sec (1 sec/0.04) at the earliest is needed to estimate LF power. Therefore, the influences of sympathetic nerve activity and sympathovagal balance immediately after exercise are not clear.

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