

Analysis of post-exercise recovery of heart rate variability using maximum entropy method

Katsuhiko Sumi¹
Shigehiro Suzuki²
Naota Sogo¹
Hiroshi Kadoma³
Yoshiro Ando¹
Michitaka Matsubara⁴

Abstract

The purpose of this study was to examine the timing of recovery of high-frequency (HF) component of heart rate variability (HRV) after exercise using frequency analysis of HRV.

Eighteen endurance-trained female volunteers participated in this study and were assigned to two groups of different characteristics (G1 and G2). The two groups performed incremental cycle exercise with progressive intensity until exhaustion. The R-R intervals were processed by maximum entropy method for determination of HF power on successive 7-second segments of 70 seconds of the recovery period. In the G1 (n = 8), HF power of the second 7-sec segment showed significantly higher values than the 7-sec before cessation of exercise ($p < 0.05$); whereas, the G2 (n = 10) exhibited significantly higher values in the third segment ($p < 0.05$). The G1 indicated significantly higher HF power than the G2 in the fifth segment ($p < 0.05$).

These findings suggested that the timing of HF recovery can be influenced by various factors, including the subjects' characteristics. However, the detection of timing of HF recovery despite the duration of sharp change in HR indicated that HRV was an effective evaluation technique for determination of autonomic nervous activity immediately after exercise.

Key words: heart rate recovery, autonomic nervous system, parasympathetic reactivation, MEM.

1 School of International Liberal Studies, Chukyo University.

2 Faculty of Science & Technology, Meijo University.

3 Aichi Shukutoku University

4 Department of Neurological Therapy, Nagoya City Rehabilitation & Sports Center.

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; Pierpont et al., 2000; Javorka et al., 2003; Kannankeril et al., 2004). 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 considerably during the early recovery period immediately after exercise. However, it does not seem that the timing is fully examined.

Heart rate variability (HRV) is widely used as an index to assess non-invasively the cardiac autonomic nervous activity. 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 maximum entropy method (Macor et al., 1996; Murasato et al., 1998; Sumi et al., 2006) and 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 the autonomic nervous activity based on a short-term R-R interval. Since the lower frequency limit of 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 ($1\text{sec}/0.15$). By analyzing a 7-second segment using the newer methods, it is possible to evaluate HF power during sharp change in HR.

This study aims to assess the cardiac autonomic nervous activity immediately after exercise by using the newer method of frequency analysis of HRV. It is hypothesized that the timing of recovery of HF power can be distinguished even in the change in HR with time immediately after exercise.

Methods

Subjects

Eighteen healthy female subjects of this study were divided into groups. The group 1 (G1, $n = 8$) was composed of highly trained younger long distance runners. The group 2 (G2, $n = 10$) included well-trained middle-aged volunteers, who performed regular endurance training (running, swimming, cycling, etc).

In order to confirm the validity of the method used in this study such grouping was conducted because the possible factors related to post-exercise HR recovery are age and athletic levels (Buchheit and Gindre, 2006; Darr et al., 1998; Shannon et al., 1987; Lipsitz et al., 1990; Yoshimitsu et al., 1996). Therefore, dependent on the characteristics of each group, it was necessary to verify if discrimination of the difference in the timing of post-exercise recovery of HRV is possible.

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 Center.

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·min⁻¹. The pedaling frequency was set at 50 rev·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.

Oxygen uptake was obtained using an Oxycon- α automatic on-line 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 to estimate maximal HR (Gellish, et al. 2007), the age-predicted HRmax was not adopted as a criterion for $\dot{V}O_{2max}$. Breathing rate (BR) was measured continuously. Throughout the test, 12 leads of the electrocardiogram (ECG) were 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).

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 (Radoski et al., 1976; Ohtomo et al., 1994). 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 data removed from each subject's original data were 20.3 ± 14.31 in the

G1 and 7.2 ± 8.20 in the G2. The outliers of the original data were $0.8 \pm 0.58\%$ in the G1 and $0.2 \pm 0.25\%$ in the G2. Since the R-R interval time series data were unequally-spaced, they were made even. MemCalc interpolates 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 stated the increase in parasympathetic nervous activity between 30-sec and one minute immediately after exercise (Imai et al., 1994; Kannankeril et al., 2004; Goldberger et al., 2006). Therefore, we divided the original time series from 7-sec before cessation of exercise to 70-sec of recovery period into ten 7-sec segments of sub-series.

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 recovery period was set between 0.15 and 1.0 Hz, depending on the subjects. The upper limit of 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 power spectrum. Furthermore, BR during the test was measured by automatic on-line breath by breath system. The peak frequency range of HF power was determined based on the results of these calculations.

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. Values were expressed as mean \pm SD. Student's unpaired t-test were used for the comparison of characteristics of subjects. Serial changes in HR and BR were evaluated by 2 (groups) \times 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 difference between 7-sec before cessation of exercise and 70-sec of 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 each segment of HF power between 7-sec before cessation of exercise and 70-sec of recovery period, the comparison between both groups was performed by Mann-Whitney U test. Wilcoxon signed rank test with Bonferroni correction was employed in the pairwise comparisons between 7-sec before cessation of exercise and each successive time point in each group. A 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 G1 were significantly younger than the G2 subjects ($p < 0.0001$). $\dot{V}O_{2max}$ was significantly higher in the G1 ($p < 0.05$). HR at rest was significantly lower in the G1 ($p < 0.01$). HF power at rest of the G1 was significantly higher than the G2 ($p < 0.05$). Training status was significantly higher in the G1 ($p < 0.0001$).

The change in HR is shown in Table 2. The HR in both groups decreased immediately after exercise. In the G1, HR of the second 7-sec segment (HR_{7-14}) indicated the significant lower level than HR_{peak} ($p < 0.05$), whereas in the G2, HR_{14-21} demonstrated significant lower level than HR_{peak} ($p < 0.05$). HR_{peak} was significantly higher in G1 ($p < 0.05$). Significant difference between groups was revealed in HR_{28-35} ($p < 0.05$). Thereafter, the G1 similarly demonstrated lower values ($p < 0.05$, $p < 0.01$). HR_{63-70} demonstrated 90.1 ± 19.2 in the G1 and 121.9 ± 20.28 in the G2 ($p < 0.01$). The ratio of HR_{63-70} to HR_{peak} was $55.7 \pm 11.50\%$ in the G1 and $75.1 \pm 13.46\%$ in the G2.

The change with time in HF power and BR are shown in Table 3. In the G1, HF power of the second 7-sec segment (HF_{7-14}) showed significantly higher value than 7-sec before cessa-

Table 1. Characteristics of subjects

	Group 1 (n=8)	Group 2 (n=10)
Age (yrs)	19.6 \pm 0.92****	57.5 \pm 7.41
Mass (kg)	48.6 \pm 6.52	47.5 \pm 3.92
Height (cm)	161.7 \pm 5.65**	153.4 \pm 5.39
$\dot{V}O_{2max}$ (ml/min/kg)	58.2 \pm 4.21*	41.4 \pm 11.62
HR at rest (bpm)	54.9 \pm 5.40**	66.1 \pm 9.41
HF power at sitting rest (ms^2)	659.9 \pm 382.09*	225.0 \pm 202.68
Training experiences (yrs)	7.6 \pm 0.92****	23.0 \pm 4.22
Training frequency (sessions/week)	6.9 \pm 0.35****	3.9 \pm 1.10
Training time (min/session)	153.8 \pm 33.78****	61.0 \pm 26.44

Values are means \pm SD; $\dot{V}O_{2max}$, maximal oxygen consumption; HR, heart rate, HF, high-frequency component of heart rate variability. * $p < 0.05$, ** $p < 0.01$, **** $p < 0.0001$ significant differences between two groups.

Table 2. Mean \pm SD of heart rate at peak exercise and first 70 second of recovery for group 1 and group 2.

Time in Recovery (sec)	Group 1 (n=8)	Group 2 (n=10)
peak	161.8 \pm 6.80	155.9 \pm 11.33 †
0- 7	152.8 \pm 9.99	155.7 \pm 12.40
7-14	146.4 \pm 11.20*	151.9 \pm 13.37
14-21	139.0 \pm 13.06**	147.5 \pm 14.23*
21-28	130.4 \pm 16.62***	143.1 \pm 14.46**
28-35	121.1 \pm 19.70***	138.4 \pm 15.89*** †
35-42	113.4 \pm 20.60***	134.6 \pm 17.19*** †
42-49	107.3 \pm 20.67***	131.4 \pm 18.21*** †
49-56	103.0 \pm 22.15***	127.9 \pm 18.99*** †
56-63	96.3 \pm 20.19***	12.50 \pm 19.61*** † †
63-70	90.1 \pm 19.20***	121.9 \pm 20.28*** † †

*p < 0.05, **p < 0.01, ***p < 0.001 significant differences vs. peak values.

† p < 0.05, † † p < 0.01 significant differences between two groups.

Table 3. High-frequency component of heart rate variability and breathing rate immediately after exercise

Time segment (sec)	HF power (ms ²)		Breathing rate (times/min)	
	Group 1 (n=8)	Group 2 (n=10)	Group 1 (n=8)	Group 2 (n=10)
Peak	2.8 \pm 1.79	3.3 \pm 1.80	53.1 \pm 6.09	47.2 \pm 8.46
0- 7	4.4 \pm 3.09	3.5 \pm 3.79	49.1 \pm 5.06	46.1 \pm 5.84
7-14	6.9 \pm 6.23*	3.6 \pm 3.44	29.4 \pm 3.21 ^{a, c}	42.3 \pm 6.89
14-21	11.3 \pm 15.03*	4.4 \pm 4.58 §	27.9 \pm 2.89 ^{a, c}	37.8 \pm 3.93 ^b
21-28	37.3 \pm 58.58*	5.5 \pm 6.90 §	26.7 \pm 4.17 ^{a, c}	35.8 \pm 3.55 ^b
28-35	59.4 \pm 70.72* †	9.1 \pm 11.52 §	25.2 \pm 3.21 ^{a, c}	33.5 \pm 3.47 ^b
35-42	159.7 \pm 215.79* †	16.8 \pm 27.51 §	24.9 \pm 3.53 ^a	32.0 \pm 3.47 ^b
42-49	287.7 \pm 443.41* †	22.3 \pm 40.03 §	24.3 \pm 2.89 ^a	31.1 \pm 3.42 ^b
49-56	264.3 \pm 472.69* †	24.7 \pm 43.94 §	24.0 \pm 2.57 ^a	30.2 \pm 3.30 ^b
56-63	369.7 \pm 738.76* †	24.6 \pm 46.23 §	23.4 \pm 1.92 ^a	29.5 \pm 3.41 ^b
63-70	486.3 \pm 754.43* †	29.1 \pm 51.58 §	23.1 \pm 1.67 ^a	28.6 \pm 3.24 ^b

Values are means \pm SD; HF, high-frequency component of heart rate variability. *p < 0.05 significant differences vs. peak values in the group 1. § p < 0.05 significant differences vs. peak values in the group 2. † p < 0.05 significant differences between two groups. ^ap < 0.05 significant differences vs. peak values in the group 1. ^bp < 0.05 significant differences vs. peak values in the group 2. ^cp < 0.05 significant differences between two groups.

tion of exercise ($p < 0.05$). Thereafter, higher values were similarly demonstrated ($p < 0.05$). The G2 exhibited significantly higher value in HF₁₄₋₂₁ ($p < 0.05$). Thereafter, a higher level was similarly demonstrated ($p < 0.05$). The ratio of HF₆₃₋₇₀ to the resting value was 63.5 \pm 50.95% in the G1 and 17.1 \pm 26.21% in the G2. In the comparison between both groups, the G1 indicated significantly higher values in HF₂₈₋₃₅ ($p < 0.05$). Thereafter, higher values were similarly demonstrated ($p < 0.05$).

BR after exercise in both groups demonstrated similar values to HF as shown in Table 3. In the G1, BR of the second 7-sec segment (BR_{7-14}) showed significantly lower value than BR_{peak} ($p < 0.05$). Subsequently, the reduction was continued ($p < 0.05$). In the G2, BR_{14-21} showed significantly lower value than BR_{peak} ($p < 0.05$). Subsequently, the reduction was continued, as well ($p < 0.05$). In the comparison between both groups, there was no significant difference in BR_{peak} . The G1 indicated significantly lower values in BR_{7-14} ($p < 0.05$). Thereafter, lower values were similarly demonstrated until BR_{28-35} ($p < 0.05$).

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 maximum entropy method and 2) there was the time difference in the recovery of HF power immediately after exercise in both groups.

HRV indices immediately after exercise

In this study, HF power in the G1 started to rise between 7 and 14 seconds after exercise. On the other hand, HF power in the G2 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 (Imai et al., 1994; Kannankeril et al., 2004; Goldberger et al., 2006). Immediately after exercise, the autonomic nervous activity begins to change (Kannankeril and Goldberger, 2002). That is, during exercise 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 (Pierpont and Voth, 2004; Goldberger et al., 2006). 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 (Perini et al., 1989; Nishime et al., 2000). 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 (Imai et al., 1994; Goldberger et al., 2006). In the current study, HR de-

creased up to 55.7% of HR_{max} in only 70 seconds after exercise in the G1 and 75.1% in the G2. Therefore, in order to assess cardiac autonomic modulation mechanism, which changes from moment to moment, data length for analysis should be as short as possible. A 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 activities (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, the validity is doubtful in the assessment of cardiac autonomic modulation mechanisms using these techniques. Recently, Kaikkonen et al. (2007) and Martinmäki et al. (2008) investigated 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 the 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 time-domain method and revealed that the effect of 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 maximum entropy method can also detect aspect of change in the 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. 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 63.5% at rest in the G1 and only 17.1% in the G2. However, the discovery of the mutation points of both groups will provide a basis to predict modulation of HR recovery after exercise.

HR and HF show similar variation (Reference to Table 2 and Table 3). This supports the previous study (Imai et al., 1994; Kannankeril et al., 2004). The strong association of parasympathetic nervous activity with HR recovery immediately after exercise is suggested. There is a parallel variation in HF and BR in both groups. This result supports 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

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. By finding the difference in the timing of HF recovery of both groups, the validity of our method used in our study was proved. Simultaneously, the results of this study suggest that factors, such as age, physical fitness, and training status influence the timing of HF recovery. 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, irrespective of age, demonstrated a significantly faster HR recovery than untrained subjects, which was particularly marked during fast-phase recovery. Buchheit and Gindre (2006) reported significant relationship between HR recovery and training load estimated by 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 to the timing of HF recovery or the presence of other factors.

Methodological limitations

This study has discovered three methodological limitations. First, HRV indicates variability of the R-R interval, not an index which shows 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 agent.

Second, cardiac rate regulation by reflex immediately after exercise is not examined. Tahara et al. (2005) indicated reperfusion reflex immediately after exercise. It is also indicated that direct stimulation to the central chemoreceptor by CO₂ increases the amplitude of respiratory arrhythmia (Peronnet and Aguilaniu, 2006). Therefore, this study indicates that HF power is influenced not only by the 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 activities (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 (1sec/0.04) at the earliest is needed to estimate LF power. Therefore, the influence of sympathetic nerve activity and sympathovagal balance immediately after exercise are not clear.

Perspectives

Frequent occurrence of cardiovascular reaction disorder, such as arrhythmia immediately after exercise, indicates the importance of HRV analysis after exercise. The reduced HF component recorded post-exercise may indicate that parasympathetic withdrawal is the key part of the autonomic control of the post-exercise tachycardia (Brown, 2007). However, it is hardly investigated owing to the limitation of the analysis method. In this study, we indicated that HF component can be analyzed in detail using maximum entropy method. This does not only lead to further understanding of cardiac autonomic nervous activity after exercise but it will be also helpful in planning out a safe cool-down exercise for athletes as well as sport lovers.

Acknowledgements

This study was partly supported by the Grant-in Aid for Scientific Research (C) of Japan Society for the Promotion of Science (No 17500486).

References

- Arai Y, Saul JP, Albrecht P, Hartley LH, Lilly LS, Cohen RJ, Colucci WS. Modulation of cardiac autonomic activity during and immediately after exercise. *Am J Physiol* 1989; 256: H132-H141.
- Berntson GG, Bigger JT Jr, Eckberg DL, Grossman P, Kaufmann PG, Malik M, Nagaraja HN, Porges SW, Saul JP, Stone PH, van der Molen MW. Heart rate variability: origins, methods, and interpretive caveats. *Psychophysiology* 1997; 34: 623-648.
- Brown SJ, Brown JA. Resting and postexercise cardiac autonomic control in trained master athletes. *J Physiol Sci* 2007; 57: 23-29.
- Buchheit M, Gindre C. Cardiac parasympathetic regulation: respective associations with cardiorespiratory fitness and training load. *Am J Physiol Heart Circ Physiol* 2006; 291: H451-H458.
- Carter J, Jeukendrup AE. Validity and reliability of three commercially available breath-by-breath respiratory systems. *Eur J Appl Physiol* 2002; 86: 435-441.
- Cole C, Foody J, Blackstone E, and Lauer M Heart rate recovery after submaximal exercise testing as a predictor of mortality in a cardiovascularly healthy cohort. *Ann Intern Med* 2000; 132: 552-555.
- Darr KC, Bassett DR, Morgan BJ, Thomas DP. Effects of age and training status on heart rate recovery after peak exercise. *Am J Physiol* 1998; 254: H340-H343.
- Gellish RL, Goslin BR, Olson RE, McDonald A, Russi GD, Moudgil VK. Longitudinal modeling of the relationship between age and maximal heart rate. *Med Sci Sports Exerc* 2007; 39: 822-829.
- Goldberger JJ, Le FK, Lahiri M, Kannankeril PJ, Ng J, Kadish AH. Assessment of Parasympathetic Reactivation After Exercise. *Am J Physiol Heart Circ Physiol* 2006; 290: H2446-H2452.
- Hayano J, Taylor JA, Mukai S, Okada A, Watanabe Y, Takata K, Fujinami T. Assessment of

- frequency shifts in R-R interval variability and respiration with complex demodulation. *J Appl Physiol* 1994; 77: 2879-2888.
- Hirsh J, Bishop B. Respiratory sinus arrhythmia in humans: how breathing pattern modulates heart rate. *Am J Physiol* 1981; 241: H620-H629.
- Imai K, Sato H, Hori M, Kusuoka H, Ozaki H, Yokoyama H, Takeda H, Inoue M, Kamada T. Vagally mediated heart rate recovery after exercise is accelerated in athletes but blunted in patients with chronic heart failure. *J Am Coll Cardiol* 1994; 24: 1529-1535.
- Javorka M, Zila I, Balharek T, Javorka K. On- and off-responses of heart rate to exercise - relations to heart rate variability. *Clin Physiol Funct Imaging* 2003; 23: 1-8.
- Kaikkonen P, Nummela A, Rusko H. Heart rate variability dynamics during early recovery after different endurance exercises. *Eur J Appl Physiol* 2007; 102: 79. 86.
- Kannankeril PJ, Goldberger JJ. Parasympathetic effects on cardiac electrophysiology during exercise and recovery. *Am J Physiol Heart Circ Physiol* 2002; 286: H2091-H2098.
- Kannankeril PJ, Le FK, Kadish AH, Goldberger JJ. Parasympathetic effects on heart rate recovery after exercise. *J Investig Med* 2004; 52: 394-401.
- Lipsitz LA, Mietus J, Moody GB, Goldberger AL. Spectral characteristics of heart rate variability before and during postural tilt. Relations to aging and risk of syncope. *Circulation* 1990; 81: 1803-1810.
- Ludbrook J. Repeated measurements and multiple comparisons in cardiovascular research. *Cardiovasc Res* 1994; 28: 303-311.
- Macor F, Fagard R, Amery A. Power spectral analysis of RR interval and blood pressure short-term variability at rest and during dynamic exercise: comparison between cyclists and controls. *Int J Sports Med* 1996; 17: 175-181.
- Martinmaki K, Rusko H. Time-frequency analysis of heart rate variability during immediate recovery from low and high intensity exercise. *Eur J Appl Physiol* 2008; 102: 353-360.
- Murasato Y, Hirakawa H, Harada Y, Nakamura T, Hayashida Y. Effects of systemic hypoxia on R-R interval and blood pressure variabilities in conscious rats. *Am J Physiol Heart Circ Physiol* 1998; 275: 797-804.
- Nishime E, Cole C, Blackstone E, Pashkow F, and Lauer M. Heart rate recovery and treadmill exercise score as predictors of mortality in patients referred for exercise ECG. *JAMA* 2000; 284: 1392-1398.
- Ohtomo N, Terachi S, Tanaka Y, Tokiwano K, Kaneko N. New method of time series analysis and its application to Wolf's sunspot number data. *Jpn J Appl Phys* 1994; 33: 2821. 2831.
- Ohtomo N, Tokiwano K, Tanaka Y, Sumi A, Terachi S, Konno H. Exponential Characteristics of Power Spectral Densities Caused by Chaotic Phenomena. *J Phys Soc Jpn* 1995; 64: 1104-1113.
- Perini R, Orizio C, Baselli G, Cerutti S, Veicsteinas A. The influence of exercise intensity on the power spectrum of heart rate variability. *Eur J Appl Physiol* 1990; 61: 143-148.
- Perini R, Orizio C, Comandè A, Castellano M, Beschi M, Veicsteinas A. Plasma norepinephrine and heart rate dynamics during recovery from submaximal exercise in man. *Eur J Appl Physiol* 1989; 58: 879. 883.
- Peronnet F, Aguilaniu B. Lactic acid buffering, nonmetabolic CO₂ and exercise hyperventilation: a critical reappraisal. *Respir Physiol Neurobiol* 2006; 150: 4-18.
- Pierpont GL, Stolpman DR, Gornick CC. Heart rate recovery post-exercise as an index of parasympathetic activity. *J Auton Nerv Syst* 2000; 80: 169-174.
- Pierpont GL, Voth EJ. Assessing autonomic function by analysis of heart rate recovery from exercise

- in healthy subjects. *Am J Cardiol* 2004; 94: 64-68.
- Radoski HR, Zawalick EJ, Fougere PF. The superiority of maximum entropy power spectrum techniques applied to geomagnetic micropulsations. *Phys Earth Planet Interiors* 1976; 12: 208-216.
- Savin WM, Davidson DM, Haskell WL. Autonomic contribution to heart rate recovery from exercise in humans. *J Appl Physiol* 1982; 53: 1572-1575.
- Shannon DC, Carley DW, Benson H. Aging of modulation of heart rate. *Am J Physiol* 1987; 253: H874-H877.
- Stratton JR, Levy WC, Caldwell JH, Jacobson A, May J, Matsuoka D, Madden K. Effects of aging on cardiovascular responses to parasympathetic withdrawal. *J Am Coll Cardiol* 2003; 41: 2077-2083.
- Sumi K, Suzuki S, Matsubara M, Ando Y, Kobayashi F. Heart rate variability during high-intensity field exercise in female distance runners. *Scand J Med Sci Sports* 2006; 16: 314-320.
- Tahara N, Takaki H, Taguchi A, Suyama K, Kurita T, Shimizu W, Miyazaki S, Kawada T, Sunagawa K. Pronounced HR variability after exercise in inferior ischemia: evidence that the cardioinhibitory vagal reflex is invoked by exercise-induced inferior ischemia. *Am J Physiol Heart Circ Physiol* 2005; 288: H1179-H1185.
- Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology. Heart rate variability: standards of measurement, physiological interpretation, and clinical use. *Circulation* 1996; 93: 1043-1065.
- Tsuchida M, Tokiwano K, Hosoi H, Sueoka K, Ohtomo N, Tanaka Y, Mukasa K. Restoration of images obtained from the scanning tunneling microscope. *Jpn J Appl Phys* 1998; 37: 3500-3505.
- Vukajlovic DD, Guettler N, Miric M, Pitschner HF. Effects of atropine and pirenzepine on heart rate turbulence. *Ann Noninvasive Electrocardiol* 2006; 11: 34-37.
- Yamamoto K, Miyachi M, Saitoh T, Yoshioka A, Onodera S. Effects of endurance training on resting and post-exercise cardiac autonomic control. *Med Sci Sports Exerc* 2001; 33: 1496-1502.
- Yoshimitsu Y, Kodama M, Matsuhisa M, Kishimoto M, Ozaki H, Tani A, Ueda N, Ishida Y, Kamada T. Diurnal heart rate variability in healthy subjects: effects aging and sex difference. *Am J Physiol* 1996; 271: H303-H310.