

ANALYSIS OF HEART RATE VARIABILITY DURING ACUTE EXPOSURE TO MODERATE ALTITUDE AND ROWING EXERCISE

Ching-Feng Cheng¹, Hui-Mei Lin², Hsin-Chieh Tsai¹, Chia-Hua Chu¹, Jung-Charng Lin¹

¹*Department of Physical Education, College of Sports and Recreation,
National Taiwan Normal University, Taipei, TAIWAN*

²*Department of Sport Science-Aquatic, Taipei Physical Education College, Taipei, TAIWAN*

The present study investigated the effects of acute exposure to moderate altitude and rowing exercises on heart rate variability (HRV). Eleven elite male rowers volunteered to participate in this study (age: 20.7 ± 2.0 yrs; height: 177.5 ± 3.5 cm; weight: 77.0 ± 6.9 kg). Each subject completed one resting measurement in the upright sitting position and two submaximal steady-state exercises (30% and 60% maximal oxygen consumption per minute, $\dot{V}O_{2max}$) in random balanced order on a rowing ergometer before, during and seven days after exposure to moderate altitude (2,200–2,600 m). Beat-to-beat HRV was measured continuously during the tests, and the 10 minutes of HRV data were recorded for subsequent analysis. Time and frequency domain analyses of HRV were performed to determine the effects of altitude and exercise. The results demonstrated that the standard deviation of all RR intervals (SDNN) and the square root of the mean squared successive differences between adjacent RR intervals (RMSSD) in sitting position at altitude were significantly lower than before exposure to altitude. There were no significant differences on the time and frequency domain indices in submaximal exercises between altitude and sea level. There were significant differences, however, on the time and frequency domain indices between resting and two submaximal exercises. These results showed that the time domain analysis of HRV could discriminate between the altitude effects on the modulation of the autonomic nervous system, but only in the sitting position and particularly the SDNN and RMSSD indices. HRV analysis could only distinguish the changes of cardiac autonomic modulation between sitting position and exercise.

Keywords: frequency domain analysis, heart rate variability, hypoxia, time domain analysis

Introduction

The cardiovascular system is mostly controlled by autonomic regulation through the activity of sympathetic

(SNS) and parasympathetic (PNS) pathways of the autonomic nervous system. Analysis of heart rate variability (HRV) permits insight into this control mechanism (Aubert et al. 2003; Task Force of European Society of Cardiology and North American Society of Pacing and Electrophysiology 1996). Various methods for the analysis of the electrocardiogram (ECG) tachogram have been applied since the late 1960s, as with the statistical, geometric and power spectral density methods (Task Force of European Society of Cardiology and North American

Corresponding Author

Ching-Feng Cheng, Department of Physical Education, College of Sports and Recreation, National Taiwan Normal University, Taipei, Taiwan

E-mail: andes_cheng@vip.pchome.com.tw

Society of Pacing and Electrophysiology 1996). Tachogram analysis can easily be determined from ECG recordings, resulting in a time series or a time duration between two consecutive R waves of the ECG (RR intervals) that are usually analyzed in time and frequency domains. As a first approach, it can be assumed that power in different frequencies band to activity of SNS and PNS nerves [low frequency (LF), 0.04–0.15 Hz; high frequency (HF), 0.15–0.40 Hz, respectively] (Aubert et al. 2003).

Altitude conditions, particularly hypoxia, exert an additional impact on the stress response to exercise. During hypoxia, endurance exercise at a given workload leads to a more pronounced increase of circulating stress hormones when compared with normal conditions (Niess et al. 2003). Sevre et al. (2001) found that a transient reduction in SNS and PNS activity (significantly decreased LF and HF power) was demonstrated during stepwise exposure to simulated high altitude (4,500 m) over three days. Yamamoto et al. (1996) reported that acute exposure to simulated altitude (> 2,500 m) affected the frequency domain indices of HRV (increased SNS and decreased PNS activity) during exercise. Liu et al. (2001) suggested that HRV analyzed with both time domain and frequency domain methods could predict the tolerance to hypoxia, which was simulated by inhalation of low oxygen gas mixture. Although the results of the simulated altitude indicated that changes of HRV could reflect the modulation of the autonomic nervous system, there were limited data to show whether the HRV indices would be affected by the real altitude environment. The real altitude conditions include hypoxia, hypothermia, hypoglycemia and hypohydration (Brooks et al. 2000). Therefore, one purpose of this study was to investigate if the time and frequency domain indices of HRV would be altered in the real moderate altitude.

It has been shown that during dynamic exercise, heart rate increases due to both a PNS withdrawal and an augmented SNS activity (Aubert et al. 2003; Bernardi & Piepoli 2001). Interestingly, Kamath et al. (1990) found that steady-state exercise (50% maximal oxygen consumption per minute ($\dot{V}O_{2max}$)) caused a significant suppression of both LF and HF components. Perini et al. (1990) performed power spectral analysis during steady-state exercise at different intensities, and found that 30% $\dot{V}O_{2max}$ represents a threshold in the cardio-

vascular adjustment to exercise. However, there were limited data to support the hypothesis that time domain analysis of HRV could detect the changes during steady-state exercise. Therefore, another purpose of this study was to investigate the effects of dynamic exercises on the time and frequency domain indices of HRV.

Methods

Subjects

Eleven elite male rowers with no smoking history from the rowing team of the Taipei Physical Education College participated in this study after providing their written, informed consent. Each subject was medically screened and one subject was excluded due to abnormal ECG signals. All subjects were asked to refrain from taking any drugs and from drinking beverages containing alcohol or caffeine during the experimentation days. Physical characteristics of the subjects are presented in Table 1.

Table 1. Physical characteristics of subjects* (n = 10)

Age (yrs)	20.7 ± 2.0
Height (cm)	177.5 ± 3.5
Weight (kg)	77.0 ± 6.9
$\dot{V}O_{2max}$ (ml · kg ⁻¹ · min ⁻¹)	57.0 ± 3.6
HR _{max} (beats · min ⁻¹)	187.8 ± 6.3

* Values are mean ± SD (standard deviation), $\dot{V}O_{2max}$ = maximum oxygen consumption, HR_{max} = maximum heart rate.

Experimental design

A Concept II Model C rowing ergometer (Concept II, Morrisville, VT, USA) was used for all tests. Each subject completed one resting measurement in the upright sitting position and two submaximal steady-state exercises (30% and 60% $\dot{V}O_{2max}$), in random balanced order on the rowing ergometer before, during and seven days after a sojourn at 2,200–2,600 m above sea level (ambient temperature, 3 ~ 12°C; humidity, 50 ~ 70%). Beat-to-beat heart rate was measured continuously using a heart rate monitor (Polar S810i™, Polar Electro Inc, Finland), and the 10 minutes of heart rate data were recorded for the subsequent analysis. Figure 1 shows the overall experimental design and testing schedule.

$\dot{V}O_{2max}$ test

Participants performed a continuous incremental rowing test developed by Cosgrove et al. (1999) to volitional exhaustion. The target duration of the test was eight to 14 minutes. The test began with each participant exercising at a 500 meter split-time of two minutes 30 seconds. Thereafter, the split-time was decreased by five seconds each minute, until the participant reached volitional exhaustion. A warm-up of six minutes, at a 500 meter split-time of two minutes 30 seconds, was performed by all participants before the $\dot{V}O_{2max}$ test. During the $\dot{V}O_{2max}$ test, respiratory and metabolic measures were recorded every 10 seconds, using a semi-automated open-circuit spirometry system (Sensormedics Vmax 29, The CardioPulmonary Care Company™, USA). Maximal effort was confirmed by attainment of at least three criteria: a respiratory exchange ratio of greater than 1:2, a heart rate of greater than 90% of age-predicted maximum, or a plateau of $\dot{V}O_2$ defined as no change ($< 150 \text{ ml} \cdot \text{min}^{-1}$) in $\dot{V}O_2$ from the previous test stage. The highest $\dot{V}O_2$ (averaged over 10 seconds) measured during the test was recorded as $\dot{V}O_{2max}$. The regression line of the 500 m split time versus $\dot{V}O_2$ relationship was used to estimate the intensities set to elicit 30% (mild) and 60% (moderate) of $\dot{V}O_{2max}$.

HRV analysis

The following time domain indices of HRV were assessed: RR mean, standard deviation of all RR intervals (SDNN), the square root of the mean squared successive differences between adjacent RR intervals (RMSSD), and the standard deviation of the one minute mean RR interval (SDANN). Power spectral density analysis of HRV was also performed to classify the LF between 0.05 and 0.15 Hz, and the HF greater than 0.15 Hz to 0.40 Hz. The ratio between the powers in LF and HF bands was calculated.

Before processing, the heart rate signals were automatically corrected by the Polar Precision Performance

SW 3.0 package software for ectopic and missed beats. Non-stationary signals or periods with more than 15% correction were excluded. One subject's heart rate data during 60% $\dot{V}O_{2max}$ rowing exercise were excluded for ectopic beats of more than 15%. Thereafter, the heart rate data were analyzed in the time domain using the HRV analytic software (Nevrokard HRV analysis, version 6.4.0, Medistar, Ljubljana, Slovenia). The frequency domain analysis was also performed by Fast-Fourier Transform using the HRV analytic software.

Statistical analysis

All data were expressed as means \pm SD (standard deviation). The significance of the biologic data was tested by repeated measurement of one-way analysis of variance (ANOVA). In the presence of a significant F value, *post hoc* comparisons of means were provided by Tukey's range test. Statistical significance was denoted by a *p* value of less than 0.05.

Results

Effects of altitude on heart rate

Heart rates in either sitting position or submaximal rowing exercises at moderate altitude were significantly higher than at sea level (Figure 2).

Effects of altitude on HRV

The means of RR intervals in the three different conditions at altitude were also significantly lower than at sea level (Table 2). The SDNN and RMSSD in sitting position at altitude were significantly lower than before exposure to altitude, and returned to sea level values by the seventh day at altitude. However, there were no significant differences on the SDNN, RMSSD and SDANN in submaximal exercises between altitude and sea level. The SDANN in sitting position at altitude was also not different than at sea level. The changes of time domain indi-

Experimental test	SL1	SL2	AL1	AL2	AL3	SL3
Environment	Sea level		Altitude		Sea level	
Testing schedule	Day 1	Day 3	Day 10	Day 12	Day 16	Day 22

Fig. 1 Experimental design and testing schedule: sea level (SL) heart rate variability (HRV) tests at sea level, altitude (AL) HRV tests at altitude.

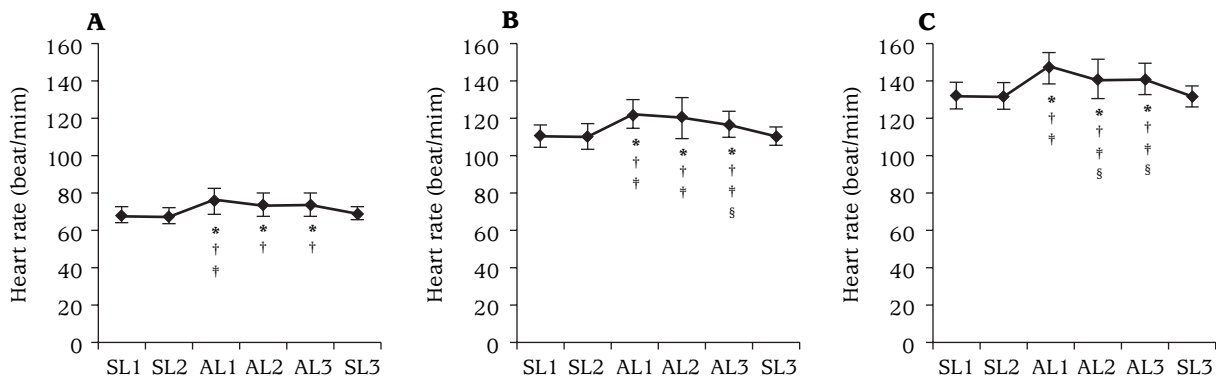


Fig. 2 Heart rates in different situations at sea level (SL) and at altitude (AL): A, sitting position; B, 30% $\dot{V}O_{2max}$; C, 60% $\dot{V}O_{2max}$; *Significantly different from SL1 ($p < 0.05$), †Significantly different from SL2 ($p < 0.05$), ‡Significantly different from SL3 ($p < 0.05$), §Significantly different from AL1 ($p < 0.05$).

Table 2. Time duration between two consecutive R waves of the electrocardiogram (RR intervals) means in sitting position, or submaximal exercises at sea level or altitude.

	SL1 (ms)	SL2 (ms)	AL1 (ms)	AL2 (ms)	AL3 (ms)	SL3 (ms)
Sitting	857.9 ± 57.8	869.8 ± 50.9	781.0 ± 69.2*†‡	802.8 ± 64.8*†	801.1 ± 62.2*†	853.1 ± 42.9
30% $\dot{V}O_{2max}$	543.0 ± 30.1	544.5 ± 31.9	490.1 ± 31.1*†‡	502.0 ± 43.9*†‡	513.4 ± 30.8*†‡	544.9 ± 24.6
60% $\dot{V}O_{2max}$	456.5 ± 24.6†	459.2 ± 25.2†	487.2 ± 31.4	497.2 ± 43.7*†‡	508.3 ± 27.8*†‡	529.6 ± 26.3

Values are mean ± standard deviation. *Significantly different from SL1 ($p < 0.05$). †Significantly different from SL2 ($p < 0.05$). ‡Significantly different from SL3 ($p < 0.05$).

AL = altitude, SL = sea level, $\dot{V}O_{2max}$ = maximal oxygen consumption per minute.

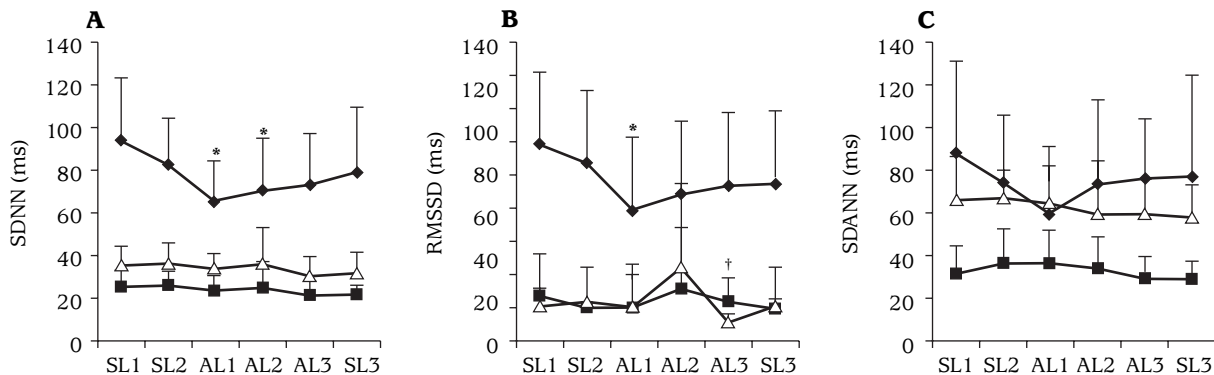


Fig. 3 The changes of time domain indices of heart rate variability (HRV) in the different exercising situations at sea level (SL) and altitude (AL). ◆, sitting position; ■, 30% $\dot{V}O_{2max}$; △, 60% $\dot{V}O_{2max}$. RMSSD = square root of the mean squared successive differences between adjacent RR intervals, RR = R waves intervals of the electrocardiogram, SDANN = standard deviation of the one minute mean RR interval, SDNN = standard deviation of all RR intervals.

*Significant difference between AL1 and SL1 values in sitting position ($p < 0.05$), †Significant difference between AL2 and AL3 values during 60% $\dot{V}O_{2max}$ exercise ($p < 0.05$).

ces of HRV in the different exercising situations at sea level or altitude are shown (Figure 3).

The LF power in sitting position measured in the frequency domain was significantly lower in AL2 than SL1, but there were no significant differences in the time

course on the HF and LF : HF ratio during resting (Figures 4 and 5). During the mild and moderate exercises, in addition to the LF : HF ratio in the third day after exposure to altitude being significantly lower than the first day at sea level, there were no changes in the LF and HF

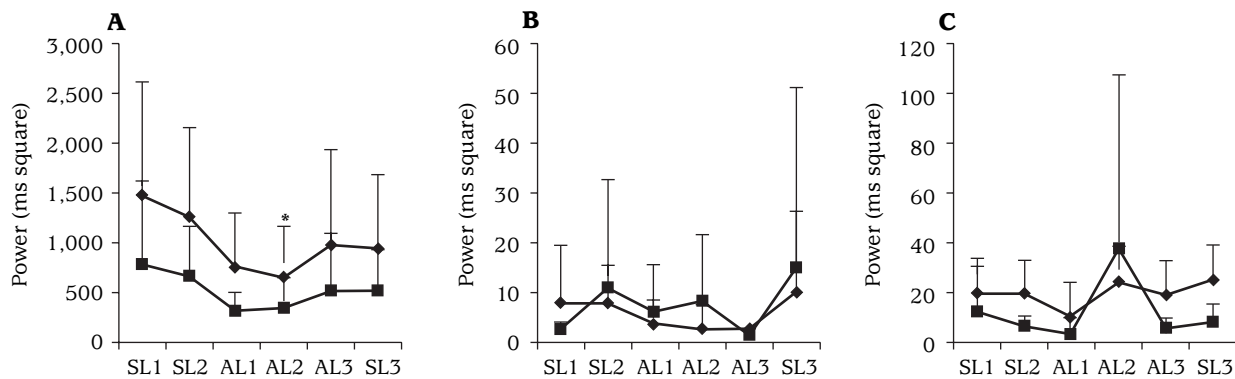


Fig. 4 Changes of frequency domain indices of heart rate variability (HRV) in the different exercising situations at sea level (SL) or altitude (AL). A, sitting position; B, 30% $\dot{V}O_{2max}$; C, 60% $\dot{V}O_{2max}$. ◆, Low frequency (LF) power; ■, High frequency (HF) power.

*Significant difference between AL2 and SL1 values in LF power.

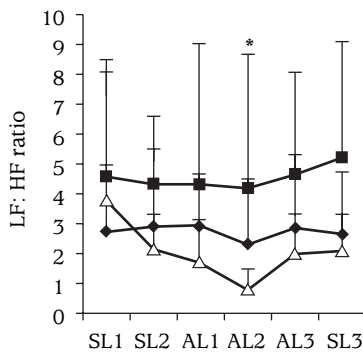


Fig. 5 The changes of LF : HF ratio in the different exercising situations at sea level or altitude. ◆, sitting position; ■, 30% $\dot{V}O_{2max}$; △, 60% $\dot{V}O_{2max}$.

*Significant difference between AL2 and SL1 values in 60% $\dot{V}O_{2max}$.

powers and the LF : HF ratios after ascending to the moderate altitude (Figures 4 and 5).

Effects of rowing exercise on HRV

The changes of HRV during mild and moderate rowing exercise in the first day at sea level are illustrated in Table

Table 3. Changes of HRV during rowing exercises at sea level.

	HR (beats · min ⁻¹)	RR Mean (ms)	SDNN (ms)	RMSSD (ms)	SDANN (ms)	LF (ms ²)	HF (ms ²)	LF : HF
Sitting	70.3 ± 4.8	857.9 ± 57.8	94.9 ± 28.6	59.8 ± 25.7	44.5 ± 20.8	1483.4 ± 1120.6	782.8 ± 840.7	2.77 ± 2.19
30% $\dot{V}O_{2max}$	110.8 ± 5.9*	543.0 ± 30.1*	25.3 ± 8.6*	12.9 ± 13.0*	15.8 ± 6.5*	19.4 ± 11.3*	11.7 ± 18.2*	4.88 ± 3.97
60% $\dot{V}O_{2max}$	131.3 ± 6.8*†	457.7 ± 23.5*†	37.2 ± 7.9*	11.4 ± 5.4*	34.3 ± 9.6†	9.0 ± 11.2*	4.7 ± 6.8*	3.51 ± 4.15

Values are mean ± standard deviation.

*Significantly different from sitting ($p < 0.05$), †Significantly different from 30% $\dot{V}O_{2max}$ ($p < 0.05$).

HR = Heart Rate, HF = high frequency, LF = low frequency, RMSSD = square root of the mean squared successive differences between adjacent RR intervals, RR = R waves intervals of the electrocardiogram, SDANN = standard deviation of the one minute mean RR interval, SDNN = standard deviation of all RR intervals.

3. There were significant differences on heart rate and RR mean among sitting and submaximal rowing exercises. The SDNN and RMSSD at the intensities of 30% and 60% $\dot{V}O_{2max}$ rowing exercises were significantly lower than in sitting position. However, there were no significant differences between mild and moderate rowing exercises. The SDANN during the 30% $\dot{V}O_{2max}$ exercise was significantly lower than in sitting position and rowing at 60% $\dot{V}O_{2max}$.

Both the LF and HF powers during 30% and 60% $\dot{V}O_{2max}$ exercises were significantly lower than in sitting position, but there were no differences in LF and HF powers between the two exercises; there also were no significant differences on the LF : HF ratios among resting, mild and moderate exercises (Table 3).

Discussion

The primary results of this study indicated that although the elite rowers exposed to altitude would significantly increase resting, mild and moderate exercising heart

rates, and would reduce the means of RR intervals, the moderate altitude environment only observably affected the SDNN and RMSSD in the sitting position. Furthermore, the frequency domain analysis may have been insensitive to the effects of moderate altitude. Interestingly, both time and frequency domain analysis in this study could not distinguish the effects of mild and moderate exercises on the cardiovascular adjustment. On the other hand, there were significant differences on the time and frequency domain indices of HRV between rest in sitting position and the two submaximal exercises.

Several studies have demonstrated that the SNS is up-regulated at rest and during exercise in response to acute and chronic exposure to 4,300 m, as evidenced by increases in plasma epinephrine and norepinephrine (Mazzeo et al. 2001, 1998, 1994, 1991). A recent study has shown similar increases in plasma epinephrine and norepinephrine in male and female distance runners, following a high-intensity interval session (10 × 1,000 m, 2-min recovery) done at moderate altitude (1,800 m), as compared with the same workout at sea level (Niess et al. 2003). The present study found that, in addition to the SDNN and RMSSD in sitting position, there were no significant differences on the time domain indices of HRV during mild or moderate exercises between at-sea-level and at-altitude. Liu et al. (2001) also found that the RMSSD at rest decreased markedly during inhalation of low oxygen gas mixture to simulate acute exposure to hypoxia.

Previous studies reported that the power spectral analysis of HRV showed that altitude increased SNS and reduced PNS indices in sitting or supine positions (Sevre et al. 2001; Bernardi et al. 1998; Perini et al. 1996). Contrary to this, in addition to finding that the LF power in sitting position in the third day and at altitude was significantly lower than at sea level, we did not find any differences in the sitting position, as HF and LF : HF ratio values at altitude were close to those observed at sea level.

Yamamoto et al. (1996) found no effects on HRV at rest when the subjects were in acute exposure to moderate levels of simulated altitude. Perini et al. (1996) reported that there were no effects on the LF and HF powers in sitting position during 35 days of acclimatization at high altitude, but they did observe that the HF in the supine position was significantly reduced and the LF : HF ratio in the supine position was 2.5 times greater than at sea level. The increase in heart rate found in the

sitting position at altitude, therefore, was not associated with modifications in spectrum components or indices of autonomic nerve activities (Perini et al. 1996). It seems that the time domain analysis of HRV discriminates with the altitude effects on the modulation of the autonomic nervous system, but only in the sitting position and particularly with the SDNN and RMSSD indices. The frequency domain indices of HRV, however, failed to demonstrate changes in autonomic regulation of the heart.

The results of this study also indicated that the lower values of SDNN and RMSSD in the sitting position lasted about seven days at altitude; the frequency domain indices of HRV at altitude, however, were not different than those at sea level. Wilber (2004) reported that the hypoxia-induced increase in epinephrine was more acute than for norepinephrine, occurring within the first hours of altitude exposure but returning to sea level values by the fifth day at altitude. It seems that the time domain analysis of HRV in sitting position is more sensitive than frequency domain analysis, and allowed us to distinguish the effects of moderate altitude acclimatization on the autonomic nervous system.

Sevre et al. (2001) demonstrated a transient reduction in LF and HF powers during stepwise exposure to high altitude (4,500 m). Yamamoto et al. (1996) reported that altitude (3,500 m) effects significantly increased heart rate and SNS indicator, and decreased the PNS indicator during 25% and 50% of the estimated maximal work rate exercises. Our study found that both time and frequency domain indices of HRV during mild and moderate exercises were unchanged at moderate altitude (2,247 m). These differences might be ascribed to factors such as the differences in levels of altitude, exercise intensity, and the period of exposure (acute or 1-week).

During dynamic exercise, heart rate increases due to both a parasympathetic withdrawal and an augmented sympathetic activity (Aubert et al. 2003; Bernardi & Piepoli 2001). Numerous studies have shown that LF and HF powers during exercise do not reflect the modifications in vagal and sympathetic activities occurring as loads are increased (Perini et al. 2000; Perini et al. 1990; Arai et al. 1989). Perini et al. (1990) suggested that above 30% maximal oxygen uptake, additional mechanisms were involved in cardiovascular adjustment. Tulppo et al. (1996) showed that the vagal modulation of heart rate is nonexistent at the ventilatory threshold level. González-

Camarena et al. (2000) reported that when cycling at 60% $\dot{V}O_{2max}$, there is a clear decline in relation with rest values on the SDNN and RMSSD indices. However, they also found that the two time domain indices during 30% $\dot{V}O_{2max}$ cycling exercise tended to remain unchanged. The results of this study indicated that the time and frequency domain analysis of HRV could only evaluate the cardiac autonomic modulation between resting and exercise; however, there were no distinctions between 30% and 60% $\dot{V}O_{2max}$ exercises.

In conclusion, the time domain analysis of HRV could discriminate between the altitude effects on the modulation of autonomic nervous system, but only in the sitting position. This is true particularly of the SDNN and RMSSD indices, but the frequency domain indices of HRV failed to demonstrate any changes in autonomic regulation of the heart. The time and frequency domain analyses of HRV could only distinguish cardiac autonomic modulation when the exercise intensity was below 30% $\dot{V}O_{2max}$.

Most previous studies have investigated the effects of simulated altitude on the HRV—e.g. hypobaric hypoxic chamber (Sevre et al. 2001; Yamamoto et al. 1996) and inhalation of low oxygen gas (Liu et al. 2001)—but the present study was the first to find that the time domain indices of HRV might be more suitable for reflecting the modulation of autonomic nervous system in a real altitude environment. However, further studies must clarify whether the Fast-Fourier Transformation or the other analysis methods, such as autoregressive modeling (Bartoli et al. 1985) or wavelet decomposition (Verlinde et al. 2001), are appropriate for detecting the effects of real altitude environment. In addition, past researchers suggested that the analysis of HRV did not adequately reflect the autonomic changes that occurred during exercise (Casadei et al. 1995; Kamath et al. 1991; Perini et al. 1990). Finally, the analysis of HRV does seem to enable comparisons within the sympatho-vagal balance among posture changes (Perini et al. 1996; Farinelli et al. 1994).

The present study also suggests that the analysis of HRV cannot distinguish cardiac autonomic modulation during mild and moderate exercise. We note that more studies are needed to investigate the application of the analysis of HRV during submaximal exercise. In addition, future studies which apply the analysis of HRV to reflect

the sympatho-vagal balance should focus on the effects of posture changes at resting condition.

References

- Arai Y, Saul JP, Albrecht P, Hartley LH, Lilly LS, Cohen RJ, Colucci WS (1989). Modulation of cardiac autonomic activity during and immediately after exercise. *Am J Physiol* 256 (1 Pt 2):H132–41.
- Aubert AE, Beckers F, Ramaekers D (2001). Short-term heart rate variability in young athletes. *J Cardiol* 37:85–8.
- Aubert AE, Seps B, Beckers F (2003). Heart rate variability in athletes. *Sports Med* 33:889–919.
- Bartoli F, Baselli G, Cerutti S (1985). AR identification and spectral estimate applied to the R-R interval measurements. *Int J Biomed Comput* 16 (3–4):201–15.
- Bernardi L, Passino C, Spadacini G, Calciati A, Robergs R, Greene R, Martignoni E, Anand I, Appenzeller O (1998). Cardiovascular autonomic modulation and activity of carotid baroreceptors at altitude. *Clin Sci (Lond)* 95:565–73.
- Brooks GA, Fahey TD, White TP, Baldwin KM (2000). Exercise physiology: Human bioenergetics and its applications (3rd ed). MacMillan Publishing Company, California.
- Bernardi L, Piepoli M (2001). Autonomic nervous system adaptation during physical exercise. *Ital Heart J* 2:831–9.
- Casadei B, Cochrane S, Johnston J, Conway J (1995). Pitfalls in the interpretation of spectral analysis of the heart rate variability during exercise in humans. *Acta Physiol Scand* 153:125–31.
- Cosgrove MJ, Wilson J, Watt D, Grant SF (1999). The relationship between selected physiological variables of rowers and rowing performance as determined by a 2000 m ergometer test. *J Sports Sci* 17:845–52.
- Farinelli CCJ, Kayser B, Binzoni T, Cerretelli P, Girardier L (1994). Autonomic nervous control of heart rate at altitude (5050 m). *Eur J Appl Physiol Occup Physiol* 69:502–7.
- González-Camarena R, Carrasco-Sosa S, Román-Ramos R, Gaitán-González MJ, Medina-Bañuelos V, Azpiroz-Leehan J (2000). Effect of static and dynamic exercise on heart rate and blood pressure variabilities. *Med Sci Sports Exerc* 32:1719–28.
- Ground A, Krause H, Kraus M, Siewers M, Rieckert H, Müller MJ (2001). Association between different attributes of physical activity and fat mass in untrained, endurance- and resistance-trained men. *Eur J Appl Physiol Occup Physiol* 84:310–20.
- Liu XX, Lu LL, Zhong CF, Cheng ZH, Yuan Q, Ren HR (2001). Analysis of heart rate variability during acute exposure to hypoxia. *Space Med Med Eng (Beijing)* 14:328–31.
- Mazzeo RS, Bender PR, Brooks GA, Butterfield GE, Groves BM, Sutton JR, Wolfel EE, Reeves JT (1991). Arterial catecholamine responses during exercise with acute and chronic high altitude exposure. *Am J Physiol: Endocrinol Metabol* 261:E419–24.
- Mazzeo RS, Carroll JD, Butterfield GE, Braun B, Rock PB, Wolfel EE, Zamudio S, Moore LG (2001). Catecholamine response to β -adrenergic blockade during exercise in women acutely exposed

-
- to altitude. *J Appl Physiol* 90:121–6.
- Mazzeo RS, Child A, Butterfield GE, Mawson JT, Zamudio S, Moore LG (1998). Catecholamine response during 12 days of high altitude exposure (4,300 m) in women. *J Appl Physiol* 84:1151–7.
- Mazzeo RS, Wolfel EE, Butterfield GE, Reeves JT (1994). Sympathetic responses during 21 days at high altitude (4,300 m) as determined by urinary and arterial catecholamines. *Metabolism* 43:1226–32.
- Niess AM, Fehrenbach E, Strobel G, Roecker K, Schneider EM, Buergler J, Fuss S, Lehmann R, Northoff H, Dickhuth H-H (2003). Evaluation of stress responses to interval training at low and moderate altitudes. *Med Sci Sports Exerc* 35:263–9.
- Perini R, Milesi S, Biancardi L, Veicstenas A (1996). Effects of high altitude acclimatization on heart rate variability in resting humans. *Eur J Appl Physiol Occup Physiol* 73:521–8.
- Perini R, Milesi S, Fisher NM, Pendergast DR, Veicsteinas A (2000). Heart rate variability during dynamic exercise in elderly males and females. *Eur J Appl Physiol Occup Physiol* 82:8–15.
- Perini R, Orizio C, Baselli G, Cerutti S, Veicstenas A (1990). The influence of exercise intensity on the power spectrum of heart rate variability. *Eur J Appl Physiol Occup Physiol* 61:143–8.
- Sevre K, Bendz B, Hankø E, Nakstad AR, Hauge A, Kåsin JI, Lefrandt JD, Smit AJ, Eide I, Rostrup M (2001). Reduced autonomic activity during stepwise exposure to high altitude. *Acta Physiol Scand* 173:409–17.
- Task Force of European Society of Cardiology and North American Society of Pacing and Electrophysiology (1996). Heart rate variability. Standards of measurement, physiological interpretation and clinical use. *Circulation* 93:1046–65.
- Tulppo MP, Mäkikallio TH, Takala TES, Seppänen T, Huikuri HV (1996). Quantitative beat-to-beat analysis of heart rate dynamics during exercise. *Am J Physiol* 271(1 Pt 2):H244–52.
- Verlinda D, Beckers F, Ramaekers D, Aubert AE (2001). Wavelet decomposition analysis of heart rate variability in aerobic athletes. *Auton Neurosci* 90(1–2):138–41.
- Wilber RL (2004). *Altitude Training and Athletic Performance*. Human Kinetics Publishers, Inc. Champaign IL.
- Yamamoto Y, Hoshikawa Y, Miyashita M (1996). Effects of acute exposure to simulated altitude on heart rate variability during exercise. *J Appl Physiol* 81:1223–9.