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DISSOCIATION OF RESPIRATORY SINUS ARRHYTHMIA AND HIGH FREQUENCY HEART RATE VARIABILITY FOLLOWING EXERCISE

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Abstract

Introduction: Heart rate variability during recovery from exercise may show a decrease in the high frequency (HF) range, indicative of a decrease in vagal modulation of the heart rate. However, a post-exercise clustering of heart beats during inspiration (respiratory sinus arrhythmia, RSA) potentially improves ventilatory efficiency by matching pulmonary perfusion to alveolar ventilation.

Aim: The purpose of this study was to investigate HRV following a single bout of exercise in trained endurance athletes, while independently quantifying respiratory sinus arrhythmia using simultaneous measures of ventilatory flow and ECG.

Materials and methods: Therefore, in 12 endurance trained males (mean $\dot{V}O_2$max 66 ml Kg^{-1} min^{-1}), both HF and RSA were measured following a single bout of exercise, consisting of 70 min continuous cycle ergometry.

Results: Mean tidal volume was elevated following exercise (0.96 vs. 1.47 L), with no change in breathing frequency. An elevated heart rate following exercise (56 ± 2 vs. 70 ± 8 beats min^{-1}) had reduced variability in the time domain and in total spectral power, but showed no change in frequency distribution (HF: 30.53 ± 16.43 vs. 29.86 ± 17.67; low frequency 65.35 ± 19.82 vs. 61.66 ± 17.63). Before exercise, mean heart rates during inspiration and expiration were 33 ± 4 and 24 ± 3 beats min^{-1} respectively. After exercise, mean heart rates during inspiration and expiration were 42 ± 6 and 28 ± 5 beats min^{-1} respectively.

Conclusions: A feature of the post-exercise tachycardia was increased clustering of heart beats during inspiration, a finding consistent with an increased respiratory sinus arrhythmia. This may serve to increase the efficiency of ventilation during recovery from exercise.

Key words: *cardiac, autonomic nervous system, heart rate, recovery, ventilation*

Introduction

In humans, studies which examine cardiac autonomic control during recovery from exercise have consistently shown a reduced vagal activity, with a shift toward increased sympathetic dominance. This autonomic contribution to cardio-deceleration following exercise has previously been studied in both trained and untrained subjects [1-5], often with the use of heart rate variability (HRV) measures to quantify the contribution of both vagal and sympathetic influences on cardiac rhythm.

In these aforementioned studies, the high frequency (0.15 – 0.4 Hz) component of HRV has been used as a measure of vagal modulation of the heart rate. However, the high frequency component of HRV, which includes normal respiratory frequencies, has also been used to quantify respiratory sinus arrhythmia [6-10]. Respiratory sinus arrhythmia describes a clustering of heart beats during inspiration which may serve to improve gas exchange efficiency by matching alveolar ventilation with pulmonary perfusion throughout the ventilatory cycle (11-14). However, it is unknown if the commonly reported decrease in the HF component of HRV following exercise also indicates a decrease in respiratory sinus arrhythmia.

A reduced respiratory sinus arrhythmia following exercise may decrease the efficiency of the lung by failing to optimally match pulmonary perfusion to alveolar ventilation throughout the ventilatory cycle. Alternatively, an increased heart rate, attributable to either vagal withdrawal or increased sympathetic activity, may allow further clustering of heart beats during inspiration and increase ventilatory efficiency – this would also demonstrate a dissociation between the measures of high frequency HRV and respiratory sinus arrhythmia. Therefore, the aim of this study was to investigate HRV following a single bout of exercise in trained endurance athletes, while independently quantifying respiratory sinus arrhythmia using simultaneous measures of ventilatory flow and ECG. It was hypothesized that:

1) a reduction in the high frequency component of HRV (indicative of vagal withdrawal) will occur following exercise; and
2) This reduction reflects a decrease in respiratory sinus arrhythmia.

Materials and Methods

Twelve endurance trained male subjects, (mean ± SD age 42.1 ± 3.3 yr; mass 75.1 ± 18.0 Kg) gave written informed consent, and completed a medical screening questionnaire. Procedures used in this study have local Ethics Committee approval. All subjects had participated in regular training for endurance (events longer than 1 hour) cycling events for more than 5 years, and participated in at least two sessions per week of cycling exercise. Subjects reported to the laboratory in a 4 hour post-prandial state, and were asked to not consume drinks containing caffeine for at least 4 hours prior to testing. Subjects were asked to refrain from strenuous exercise for at least 24 hours before the test. Subjects were excluded if they were on any prescribed medication, had experienced illness (e.g. stomach upset, cold or flu like symptoms) in the preceding week, or had a musculo-skeletal injury which affected their normal routine training. Water was provided ad-libitum throughout the test.

Pre- exercise measures were taken during the last 6 min of a 20 min seated rest period before the initial warm-up. Post-exercise measures were taken during final 6 min of a 20 min seated rest after all exercise, including the 10 min 'active' recovery.

Exercise protocol

The exercise protocol consisted of a single laboratory session on a cycle ergometer, lasting approximately 70 min, during which subjects continuously exercised. The session had two components: firstly a continuous incremental ramp test with 5 min stages (duration 35–45 min); and secondly, a ramp test (1 min stages) to volitional exhaustion (duration 4–6 min). An active recovery for 10 min was allowed between components. This protocol was chosen as it can be used to determine multiple performance parameters (e.g. anaerobic threshold estimation, power and heart rate relations, and maximal oxygen uptake) in a single session, and is well tolerated by endurance cyclists.

Initially, subjects performed a 10 minute warm up on an electronically braked cycle ergometer (Lode Excalibur, Lode BV, Groningen, Netherlands). Subjects were asked to ride at their normal cadence, and the ergometer was set up to replicate their normal riding position. Subjects performed a continuous incremental test with approximately 7-9 stages, where each stage was 5 min, during which a capillary blood sample was collected from a finger tip after 4 min and used for measurement of blood lactate concentration [La^-] (Lactate Pro, Arkray, Kyoto, Japan). Heart rate was continuously monitored throughout the test (S610, Polar Electro, Finland). Power output was increased by 20 Watts at the start of each stage, and initial power output was based on the ability of the subject (in the region of 80–120 Watts for all subjects). When a lactate value greater than 4.5 mmol L^{-1} was achieved, exercise intensity was reduced and subjects were given a 10 min active recovery at a power output approximately 50% below their estimated power at 4mmol L^{-1} [La^-]. Subjects then performed an incremental test to volitional exhaustion, starting at a power output equivalent to that achieved at a [La^-] of approximately 4.5mmol L^{-1}, during which power output was increased by 20W min^{-1}. Throughout each minute, exhaled air was collected and analysed for oxygen (Servomex O_2 1440, East Sussex, UK) and carbon dioxide (Servomex CO_2 1440, East Sussex, UK) content, and total volume (Dry Gas Meter, Harvard Apparatus, Kent, UK). The $\dot{V}O_2$max test was terminated when the subject could no longer continue (volitional exhaustion), and all subjects had reached a plateau in their heart rate response to the exercise at test termination. The maximum heart rate of all subjects was within 10% of their age predicted maximum (220 – Age), and analysis of the exhaled gases indicated that all subjects achieved a respiratory exchange ratio of >1.1 at test termination. Previous unpublished work using this $\dot{V}O_2$max protocol indicated that intra-subject reproducibility was approximately 5 %.

Heart rate and heart rate variability analysis

Resting electrocardiogram (ECG) recordings (limb lead 2, sampled at 1KHz, AD Instruments, Dunedin, NZ) were taken from subjects seated quietly in an air conditioned laboratory. The last 6 min of a 20 min recording period before exercise was used to represent pre-exercise. Following a 10 min low intensity active recovery from exercise, subjects were again seated for 20 min while ECG was recorded. The last 6 min of the 20 min recording period after exercise was used to represent post-exercise.

Heart rate and HRV were determined using commercially available software (HRV Module for Chart 5 Pro, AD Instruments). Heart rate was calculated by expressing R - R intervals as beats/min. Intervals between adjacent R waves were detected using a threshold detection of between 0.5 and 1.0 mV, and classified as artifact (< 5 ms and > 2000 ms), ectopic (5 ms to 500 ms, and 1600 ms to 2000 ms), and normal (500 ms to 1600 ms). HRV data were analysed in the time domain using:

1. The mean R-R interval;
2. The standard deviation of the normal mean R-R interval (SDRR);
3. The standard deviation of the mean difference between successive R-R intervals (SD Δ RR).

In the frequency domain, HRV data were analysed with a 1024 Fast Fourier Transform using a Welch averaged periodogram method (histogram bin size 10ms), and banded as Very Low Frequency (VLF: 0 – 0.04 Hz), Low Frequency (LF: 0.04 – 0.15 Hz), and High Frequency (HF: 0.15 – 0.4 Hz). Total power for each

spectrum was defined as the area under the spectrum from 0 to 0.5 Hz, and normalised units (NU) for the LF and HF components (which take into account any changes in total spectrum power) were used to calculate the low frequency : high frequency ratio (LF:HF).

Ventilation before and after exercise

During the periods of seated rest before and after exercise, subjects breathed orally (nasal occlusion) through a sterile mouthpiece connected to a pneumotach (MLT1000L Respiratory Flow Head, AD Instruments) connected to a spirometer (ML141, AD Instruments). Ventilatory flow was integrated to determine volume, and this signal was calibarated using a 3L Hans Rudolph calibration syringe. Before each recording, the pneumotach signal was reset to zero when disconnected from the subject. Values for tidal volume (V_T), duration times for inspiration (T_I) and expiration (T_E), and breathing frequency (B_f) were taken from the ventilatory flow and volume signals for all breaths taken during the final 6 min period of seated rest, to coincide with the ECG trace used for HRV analysis. Short periods of breath holding (eg. swallowing) and obvious excursions from "normal" (e.g. coughing), were rarely observed and were excluded from the analysis. Minute ventilation was determined as the product of B_f and V_T. All experimentation was carried out in a large, well ventilated fit-for-purpose human performance laboratory, at an ambient temperature of between 19 and 21°C. The temperature of expired air on a breath-by-breath basis could not be measured, and therefore there is no correction for small changes in expired volumes induced by body temperature.

Values of flow were negative for inspiration and positive for expiration. For all ECG R waves recorded throughout each 6 min epoch, the corresponding value of the ventilatory flow signal was recorded and sorted according to value. For each subject under each condition, the total number of all positive, and of all negative values, was used to determine the average heart rates during exhalation and inhalation respectively.

Statistical analysis

Throughout, paired data (pre-exercise *vs*. post-exercise) were compared with an equal variance, two tail Student *t*-test, where significance was set at $P<0.05$. HRV data in the frequency domain were transformed using a natural logarithmic function before statistical analysis.

Results

All subjects completed the tests, and no ectopic beats were observed in any subject either before or after exercise. Mean (SD) $\dot{V}O_2$max was 66 (7.3) ml

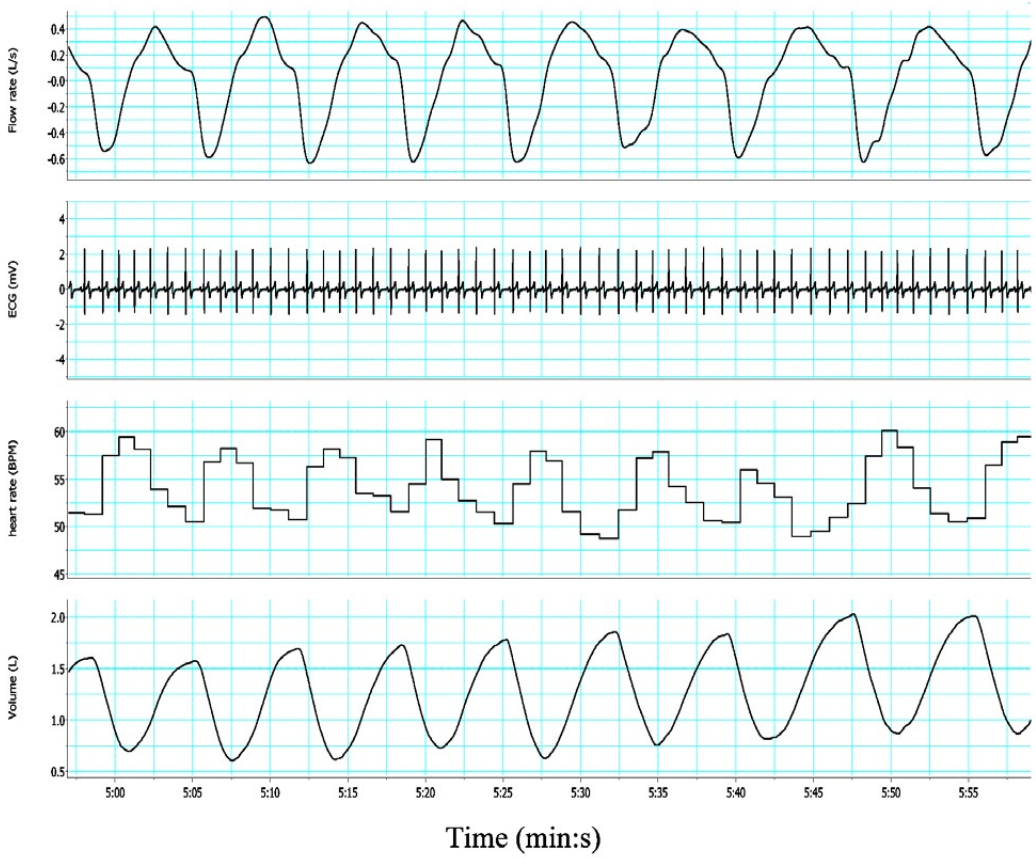

Figure 1. *Representative 1 min recording of ventilatory flow, limb lead II ECG, beat-to-beat heart rate, and tidal volume. Cardio-acceleration occurs during inspiration (negative flow rate), and cardio-deceleration occurs during expiration (positive flow rate). In this example, average tidal volume is 1.09 L, and breathing frequency is 9 breaths min^{-1} (0.15 Hz).*

Table 1. *Heart rate (HR) and measures of heart rate variability in time and frequency domains, before and after exercise in 12 endurance trained athletes. TP: total power; LFNU: normalised low frequency power; HFNU: normalised high frequency power.*

	Time domain				Frequency Domain		
	HR beats min^{-1}	mean RR ms	SDRR ms	SD Δ RR ms	TP ms^2	LF_{NU} (0.04-0.15 Hz)	HF_{NU} (0.15-0.4 Hz)
Pre-exercise	56	1066	82.42	55.24	8398	65.35	30.53
	(2)	(26)	(35.61)	(29.84)	(7222)	(19.82)	(16.43)
Post-exercise	74	755	46.16	31.72	2880	61.66	29.86
	(8)	(80)	(31.81)	(27.61)	(3289)	(17.63)	(17.67)
P value	**0.003**	**0.002**	**0.006**	**0.040**	**0.039**	0.763	0.956

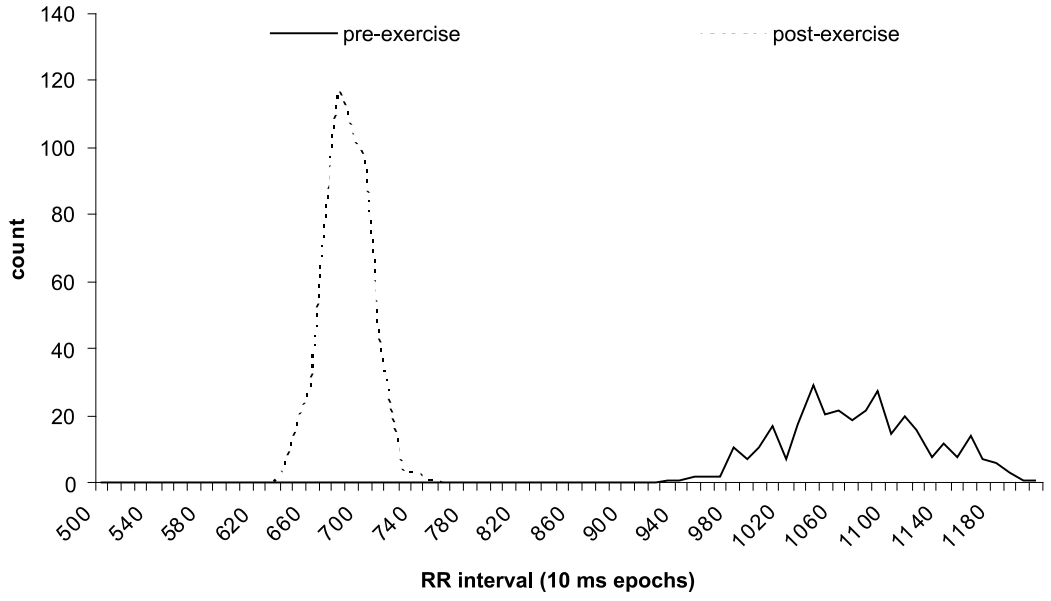

Figure 2. *Representative R-R interval histogram for a 6 min ECG recording before and after exercise*

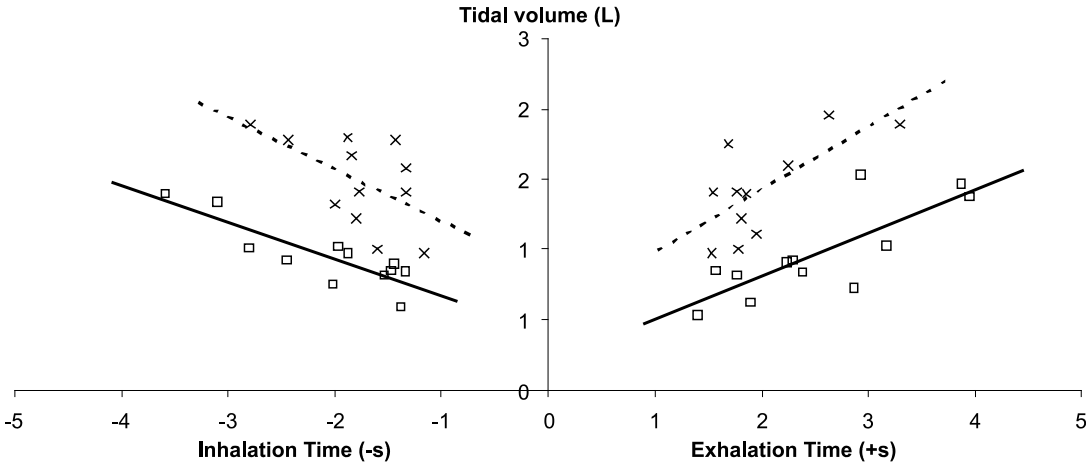

Figure 3. *Variability in tidal volume, inspiration duration time, and expiration duration time, before (squares) and after (crosses) exercise. Each data point represents the mean of 10 successive breaths for each subject.*

Table 2. *Mean (SD) tidal volume (VT) and inspiration (TI) and expiration (TE) times before and after exercise in 12 endurance trained athletes*

	Inspiration		Expiration	
Pre-exercise	VT (litres)	0.96 (0.23)	VT (litres)	0.97 (0.32)
	TI (s)	2.08 (0.75)	TE (s)	2.53 (0.84)
Post - exercise	VT (litres)	1.49 (0.31)	VT (litres)	1.45 (0.33)
	TI (s)	1.78 (0.47)	TE (s)	2.02* (0.51)

Statistical comparisons (paired samples *t*-test):
Pre-exercise TI *vs.* TE: *P*=0.287 post-exercise TI *vs.* TE: *P*=0.276
TI pre- *vs.* TI post-exercise: *P*=0.103 TE pre- *vs.* TI post-exercise: ***P*=0.011**

Table 3. *Calculated heart rates during phases of ventilation, derived from the number of beats occurring during either inspiration or expiration throughout a 6 min period*

	Heart rate (inspiration) (beats min^{-1})	Heart rate (expiration) (beats min^{-1})
Pre-exercise	33 (4)	24 (3)
Post - exercise	42 (6)	28 (5)
P value	**0.004**	**0.076**

P value from a paired samples Student *t*-test.

Kg^{-1} min^{-1}, and mean peak HR was 185 (12) beats min^{-1}. At $\dot{V}O_2$max, all subjects had a respiratory exchange ratio >1.1, and all tests were terminated at volitional exhaustion. Mean (SD) power at $\dot{V}O_2$max was 405 (35) Watts, and the mean relative power at the estimated anaerobic threshold was 78% of $\dot{V}O_2$max power. A representative 1 minute recording of ECG and ventilation is shown in figure 1 – the respiratory sinus arrhythmia is clear, with heart rates consistently higher during inspiration.

There were significant differences in heart rate and measures of heart rate variability in the time domain between the pre- and post-exercise periods (see Table 1). A representative example of the changes in the R-R interval between the pre- and post-exercise periods is shown in figure 2 – the shift toward a lower mean interval (higher heart rate) and reduced range of interval values typifies the differences in the post-exercise ECG. There were significant differences in measures of ventilation between the pre- and post-exercise periods (see Figure 3), with increases in both VT and $\dot{V}_E$ following exercise with no change in Bf. Table 2 shows the descriptive statistics for VT, and the inspiration (TI) and expiration (TE) duration times. The only statistical difference was between TE, where pre-exercise mean $\pm$ SD were 2.53 $\pm$ 0.84 s and post-exercise was 2.02 $\pm$ 0.51 s.

There was a significant difference in the number of heart beats recorded during inspiration in the post-exercise period compared to the pre-exercise period. However, there were no changes occurring in the number of heart beats recorded during expiration. These have been expressed as rates (beats min^{-1}), and are shown in table 3.

Discussion

This study uniquely reports a 'clustering' of heart beats during inspiration in the period following exercise. This study also shows that clustering occurs when the heart rate is elevated above normal resting values. A further unique finding is that clustering of heart beats during inspiration is occurring without any obvious changes in the frequency characteristics of heart rate variability, thus indicating dissociation between respiratory sinus arrhythmia and the HF component of HRV.

This study reports evidence of decreased HRV in the time domain following exercise, with significant reductions in SDRR and SD Δ RR. This finding is consistent with an elevated mean resting heart rate and partly reflects the reduced vagal modulation of cardiac rhythm. However, there were no significant changes in heart rate variability following exercise when explored in the frequency domain – a finding which is not consistent with previous literature [4, 5, 15, 16]. Subjects in these aforementioned studies were all trained, with a typical mean $\dot{V}O_2$max 55 ml Kg^{-1} min^{-1} – somewhat lower than the mean $\dot{V}O_2$max of subjects in the present study (66 ml Kg^{-1} min^{-1}). It is likely that restoration of normal resting autonomic control of the heart rate is faster in trained subjects, and one could speculate that the 'fitter' the subject, the faster the recovery.

The exercise protocol used in the present study differed somewhat from previous studies, and it is acknowledged that short duration protocols [21] or longer interval sessions [4] may have different influences on cardiac autonomic control following exercise. Breathing is closely matched to metabolism such that

the magnitude of metabolic acidosis incurred during exercise is a strong determinant of ventilation. Equally, muscle perfusion is closely matched to metabolic demand during exercise. Therefore it is likely that the cardio-respiratory response to exercise protocols with different metabolic demands (high intensity – short duration, or low intensity – long duration) will be different.

In the present study, the LF component (in ms^2) was lower or when normalized for changes in total power, unchanged, following exercise – consistent with the findings of others [1, 4, 20, 21]. Variability in the heart rate at these lower frequencies reflects multiple mechanisms of cardiac autonomic control [17, 18], for example, an exercise-induced increase in baroreceptor sensitivity [19] may contribute to an increased LF component when recovering from exercise [2].

There are previous reports of a dissociation of HF heart rate variability and respiratory sinus arrhythmia, for example, during exposure to normoxic hypercapnia [22] and during a head-up tilt procedure [6]. The elevated heart rate following exercise is partly a result of an increased sympathetic drive, coupled to a reduced vagal tone, and this is typically associated with a reduced HF cardiac variability. However, in the present study, there was no evidence of a reduced HFNU component of HRV, but evidence of inspiration induced cardio-acceleration. The increase in heart rate during inspiration post-exercise may indicate a cardiac reflex independent of the vagus nerve whereby an inspiration-induced increase in venous return may initiate more rapid SA node depolarisation [23].

When pre-exercise was compared to post-exercise, there was a small reduction in the time spent during expiration, and this reduced the time available for clustering of heart beats during expiration. However, in the present study, the number of heart beats occurring during expiration was not different between pre-exercise and post-exercise, such that the only significant change in heart beat clustering occurred during inspiration. The ventilatory changes which occur following exercise, particularly the increased V_T, will affect venous return and therefore cardiac output. The clustering of heart beats during inspiration may be a mechanism by which pulmonary perfusion is more closely matched to alveolar ventilation – potentially improving ventilatory efficiency [24, 25].

In summary, this study uniquely reports dissociation between the high frequency component of HRV and respiratory sinus arrhythmia. A feature of the post-exercise tachycardia was an increase in the number of heart beats occurring during inspiration, a finding consistent with an elevated respiratory sinus arrhythmia. This may potentially improve pulmonary perfusion during alveolar ventilation and may improve the efficiency of the lungs.

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