Original Article

Resting and exercise-related heart rate responses to high intensity interval training in women: A pilot study

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Abstract

Purpose: We aimed to 1) explore associations between changes in supine and standing heart rate variability (HRV) and submaximal exercising HR (HRe_x) and post-exercise HR recovery (HRR) variables 1 day post-high intensity interval training (HIIT), and 2) determine how baseline characteristics (e.g., aerobic fitness, HRV, etc.) associate with HRV responses in women. **Methods:** After baseline assessment of maximal oxygen uptake ($\dot{V}O_{2max}$) and post-waking supine and standing HRV (5 days natural logarithm of the root-mean square of successive differences, LnRMSSD), healthy women ($n = 10$, age = 23.3 ± 3.4 years, height = 163.1 ± 8.9 cm, weight = 61.4 ± 9.7 kg) performed HIIT on a treadmill (7×3 min at 90% of the velocity attained at $\dot{V}O_{2max}$, interspersed with 2min recovery at $4 \text{ km} \cdot \text{h}^{-1}$). LnRMSSD was obtained the subsequent morning. Immediately before and 24 h post-HIIT, a submaximal exercise test was performed to obtain HRe_x at 60, 70, 80, and 90% of velocity attained at $\dot{V}O_{2max}$ and post-test HRR. Changes (Δ) from pre- to 1 day post-HIIT were calculated. **Results:** A large but non-significant relationship was observed between Δ LnRMSSD standing and Δ HRe_x at 60% ($r = -0.55$, $p = 0.10$). All other associations between Δ LnRMSSD, Δ HRe_x and Δ HRR ranged from *trivial-moderate* (all $p > 0.05$). $\dot{V}O_{2max}$, HR recovery at 2 min post- $\dot{V}O_{2max}$, baseline LnRMSSD (supine and standing), and standing LnRMSSD coefficient of variation were each associated with Δ LnRMSSD standing (all *large*, $p < 0.05$). **Conclusions:** These findings suggest that Δ LnRMSSD does not predict Δ HRe_x in healthy women at moderate-high intensities. Women with lower and less stable baseline LnRMSSD may require a longer recovery duration from HIIT, impacting training intensity and frequency prescription considerations.

Key Words: cardiovascular, parasympathetic, autonomic, fitness, recovery

Introduction

Tracking changes in heart rate variability (HRV) has become increasingly popular for guiding exercise prescription (Javaloyes et al., 2018) and assessing training adaptations in competitive (Flatt et al., 2020; Korobeynikov et al. 2018) and recreational (Weippert et al., 2018) athletes. HRV refers to the variation in time between heart beats and reflects centrally-mediated cardiac control (Shaffer & Ginsberg, 2017). Vagally-mediated HRV is suppressed for up to 48h post-high intensity exercise (HIE) in women (Kiviniemi et al., 2010) and its return to baseline is thought to reflect cardiovascular recovery (Stanley et al., 2013). Implementation of HIE on days when HRV is within or above baseline yields the same performance improvement from fewer HIE sessions (Kiviniemi et al., 2010). Conversely, attenuated HRV following HIE may be associated with acute exercise responses suggestive of inadequate cardiovascular recovery, such as increased exercising heart rate (HRe_x) and decreased post-exercise HR recovery (HRR) (Lamberts et al., 2004). Indeed, endurance-trained women exhibit higher resting HRV, lower HRe_x at submaximal intensities and a faster HRR when compared to untrained women (Du et al., 2005; Hartung et al., 1995). However, associations among these variables as they change on a day-to-day basis in response to HIE requires investigation.

Baseline cardiac-autonomic characteristics have been associated with chronic adaptations to endurance exercise among men and women. For example, individuals with higher pre-intervention HRV tend to experience greater improvements in maximal oxygen uptake ($\dot{V}O_{2max}$) relative to those with lower HRV (Hedelin et al., 2001). In addition, individuals with higher baseline cardiac-parasympathetic activity (i.e., higher HRV) show greater aerobic fitness improvements following chronic HIE, whereas individuals with lower baseline HRV appear to respond better to low-intensity exercise (Vesterinen et al., 2016). Thus, baseline characteristics may also influence shorter-term (e.g., 1 day) responses to a single bout of HIE in women. However, this hypothesis has received little investigation.

While both supine (Vesterinen et al., 2016) and standing (Kiviniemi et al., 2010) HRV has been used to guide endurance training and reflect adaptations in women (Flatt & Esco, 2016), the optimal position remains to be elucidated (Schneider et al., 2019). Supine measures reflect the resting state while standing measures reflect

autonomic responsiveness to orthostasis that may be exaggerated in fatigued women (Uusitalo et al., 2000). Determining which measurement position is most sensitive to the effects of training is needed to guide HRV recording methodology in the field. Therefore, the aims of this study were 1) to explore associations between changes in supine and standing HRV, HRR, and HRe_x 1 day following a bout of high intensity interval training (HIIT) and 2) determine how baseline characteristics (e.g., $\dot{V}O_{2\max}$, Baseline LnRMSSD, etc.) associate with HRV responses among women.

Materials & Methods

Participants

Ten healthy and recreationally active women were recruited for this study (age = 23.3 ± 3.4 years, height = 163.1 ± 8.9 cm, weight = 61.4 ± 9.7 kg, $\dot{V}O_{2\max} = 40.1 \pm 7.9$ ml·kg⁻¹·min⁻¹). Volunteers indicated that they were free from cardiovascular, metabolic, and orthopedic disorders, and not taking heart rate-altering prescription medications. All participants were informed of the benefits and risks of the investigation prior to signing an informed consent document that was approved by the institutional review board.

Protocol

This was a prospective, pre vs. post observational study involving a single cohort of healthy women. Following assessment of baseline fitness and post-waking supine and standing HRV, participants performed HIIT on a treadmill. HRV was obtained again on the subsequent morning for comparison to baseline. Immediately before and 24h post-HIIT, a submaximal exercise test (SET) was performed to obtain HRe_x and HRR variables. Differences and associations among variables were assessed.

HRV

At their first laboratory visit, participants were provided with a clinically-validated ECG sensor (Bittium Faros 180, 1000 Hz, Oulu, Finland) (Vandecasteele et al., 2017) and instructed how to perform 3-min R-R recordings in the supine and standing position. Participants were asked to perform HRV measurements at home, after waking and urination for 5 days leading up to and 1 day following HIIT. HRV measurements were subsequently downloaded from devices and evaluated with Kubios™ software (Kuopio, Finland). The natural logarithm of the root-mean square of successive differences (LnRMSSD) was computed as the vagally-mediated HRV parameter for analysis, obtained from the second minute of each recording (Flatt & Esco, 2016; Nakamura et al., 2017). The 5-day LnRMSSD average and coefficient of variation (LnRMSSDcv) were calculated to represent baseline cardiac-autonomic characteristics (Flatt & Esco, 2015).

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

Following HRV familiarization, $\dot{V}O_{2\max}$ was assessed via indirect calorimetry (Parvo Medics™, Sandy, Utah, USA) using a standardized graded maximal exercise test (GXT) on a motorized treadmill (4Front, Woodway, Waukesha, WI). After a 3-min warm-up (5 km·h⁻¹, 0.5% fixed grade), the test started at 8 km·h⁻¹ and increased 1.6 km·h⁻¹ every 3 min until volitional exhaustion with continual HR monitoring (H10, Polar Electro Oy, Kempele, Finland). HR at 1 and 2 min post-test were obtained and subtracted from peak HR (HR_{peak}) while participants walked at 4 km·h⁻¹ to derive GXT HRR1 and HRR2. The highest 30-s mean oxygen uptake was determined as $\dot{V}O_{2\max}$.

Submaximal Exercise Test

Participants reported to the laboratory for their second visit between the hours of 0900-1100 following a 2-hour fast and having refrained from vigorous activity for 48 hours. After a 3-min warm-up (5 km·h⁻¹), a SET consisting of 3 min stages at 60, 70, 80, and 90% of the velocity attained at $\dot{V}O_{2\max}$ ($v\dot{V}O_{2\max}$) was administered to obtain HRe_x. Ratings of perceived exertion (RPE) were recorded at the end of each stage (Borg, 1973). Following completion of the test, subjects walked at 4 km·h⁻¹ for 2 min to determine SET HRR1 and HRR2. On the third laboratory visit (one day post-visit 2) SET was repeated to assess changes in HRe_x and HRR variables.

HIIT Session

Immediately following SET during laboratory visit 2, participants performed a bout of HIIT on the treadmill. The protocol consisted of 7×3 min at 90% of $v\dot{V}O_{2\max}$, interspersed with 2 min recovery at 4 km·h⁻¹ (Kaikkonen et al., 2008). HRe_x and RPE responses were recorded at the end of each interval (Table 1).

Table 1. Exercising heart rate and rating of perceived exertion acquired at the end of each interval.

Interval	HRe _x (b·min ⁻¹)	Relative HRe _x (%)	RPE
1	187.3 ± 10.6	95.0 ± 2.6	15.9 ± 2.3
2	186.1 ± 10.6	94.4 ± 2.4	15.1 ± 1.5
3	187.9 ± 11.3	95.3 ± 2.5	16.7 ± 1.9
4	188.7 ± 10.5	95.7 ± 2.2	17.0 ± 1.9
5	188.8 ± 10.7	95.7 ± 2.1	17.7 ± 2.2
6	189.0 ± 10.0	95.8 ± 2.1	18.1 ± 2.0
7	189.5 ± 10.1	96.1 ± 2.3	18.2 ± 2.0

HRe_x = exercising heart rate; RPE = rating of perceived exertion on the 6-20 scale.

Statistical Analysis

Paired t-tests and effect sizes (ES) (Lininger & Riemann, 2016) were used to assess differences in supine and standing LnRMSSD, HRex at each intensity and HRR from before and 1 day post-HIIT. ES were interpreted qualitatively as: $<0.2 = \text{trivial}$; $<0.6 = \text{small}$; $<1.20 = \text{moderate}$; $<2.0 = \text{large}$ (Hopkins et al., 2009). Changes (Δ) were calculated for all HR measures. Associations between Δ -variables were quantified via Pearson's correlations and were interpreted qualitatively as $<0.1 = \text{trivial}$, $<0.3 = \text{small}$, $<0.5 = \text{moderate}$, $<0.8 = \text{large}$, $\geq 0.9 = \text{nearly perfect}$ (Hopkins et al., 2009). $P \leq 0.05$ was statistically significant. Procedures were carried out using JMP 13 (Carry, North Carolina, USA).

Results

There were no differences ($P > 0.05$) for supine or standing LnRMSSD, HRex at 60-80% of $\dot{V}O_{2\max}$ or HRR1. HRex at 90% of $\dot{V}O_{2\max}$ and HRR2 were each greater 1 day post-HIIT (Table 2).

Table 2. Comparison of values from pre (or baseline) to 1 day post-high intensity interval training.

Variable	Pre/Baseline	Post	P	Effect Size
Supine LnRMSSD	4.12 ± 0.52	4.17 ± 0.67	0.692	0.08
Standing LnRMSSD	3.26 ± 0.61	3.23 ± 0.78	0.716	-0.04
HRex 60% $\dot{V}O_{2\max}$ ($\text{b} \cdot \text{min}^{-1}$)	147.6 ± 12.2	151.2 ± 12.5	0.237	0.28
HRex 70% $\dot{V}O_{2\max}$ ($\text{b} \cdot \text{min}^{-1}$)	167.0 ± 11.2	167.8 ± 10.5	0.581	0.07
HRex 80% $\dot{V}O_{2\max}$ ($\text{b} \cdot \text{min}^{-1}$)	178.9 ± 10.5	179.2 ± 10.7	0.766	0.03
HRex 90% $\dot{V}O_{2\max}$ ($\text{b} \cdot \text{min}^{-1}$)	187.3 ± 10.6	189.4 ± 9.9	0.029*	0.20
RPE 60% $\dot{V}O_{2\max}$ (au)	8.2 ± 2.1	7.9 ± 1.6	0.278	-0.15
RPE 70% $\dot{V}O_{2\max}$ (au)	11.0 ± 1.6	10.3 ± 2.1	0.111	-0.36
RPE 80% $\dot{V}O_{2\max}$ (au)	13.7 ± 1.9	13.8 ± 2.7	0.798	0.04
RPE 90% $\dot{V}O_{2\max}$ (au)	16.1 ± 2.2	16.7 ± 2.5	0.193	0.24
HRR1 ($\text{b} \cdot \text{min}^{-1}$)	35.1 ± 8.3	37.3 ± 11.6	0.201	0.21
HRR2 ($\text{b} \cdot \text{min}^{-1}$)	55.0 ± 13.2	59.1 ± 16.4	0.041*	0.26

LnRMSSD = natural logarithm of the root-mean square of successive differences; HRex = exercising heart rate; $\dot{V}O_{2\max}$ = the velocity obtained at maximal oxygen uptake; RPE = rating of perceived exertion on the 6-20 scale; HRR = heart rate recovery.

* = statistically significant.

Correlation coefficients between Δ LnRMSSD, HRex responses, and baseline participant characteristics are presented in Table 3. A *large* but non-significant relationship ($P = 0.10$) was observed between Δ LnRMSSD standing and Δ HRex at 60% of $\dot{V}O_{2\max}$. All other associations between Δ LnRMSSD, Δ HRex and Δ HRR ranged from *trivial*–*moderate* and were non-significant ($P > 0.05$). $\dot{V}O_{2\max}$, GXT HRR2, baseline LnRMSSD mean (supine and standing) and standing LnRMSSDcv were significantly ($p < 0.05$) associated with Δ LnRMSSD standing.

Table 3. Correlation coefficients between heart rate and fitness variables.

Variable	Δ LnRMSSD supine	Δ LnRMSSD standing
<i>Submaximal Exercise Responses</i>		
Δ HRex 60% $\dot{V}O_{2\max}$ ($\text{b} \cdot \text{min}^{-1}$)	0.189	-0.552
Δ HRex 70% $\dot{V}O_{2\max}$ ($\text{b} \cdot \text{min}^{-1}$)	-0.098	-0.209
Δ HRex 80% $\dot{V}O_{2\max}$ ($\text{b} \cdot \text{min}^{-1}$)	0.020	-0.137
Δ HRex 90% $\dot{V}O_{2\max}$ ($\text{b} \cdot \text{min}^{-1}$)	0.307	0.123
Δ HRR1 ($\text{b} \cdot \text{min}^{-1}$)	-0.226	-0.023
Δ HRR2 ($\text{b} \cdot \text{min}^{-1}$)	-0.016	-0.046
<i>Baseline Characteristics</i>		
$\dot{V}O_{2\max}$ ($\text{ml} \cdot \text{kg} \cdot \text{min}^{-1}$)	-0.141	0.660*
GXT HRR1 ($\text{b} \cdot \text{min}^{-1}$)	0.028	0.450
GXT HRR2 ($\text{b} \cdot \text{min}^{-1}$)	0.193	0.648*
Baseline LnRMSSD Supine	-0.070	0.748*
LnRMSSDcv Supine	-0.175	-0.228
Baseline LnRMSSD Standing	-0.240	0.654*
LnRMSSDcv Standing	0.061	-0.700*

*indicates statistical significance ($p < 0.05$)

Δ HRex = changes in exercising heart rate; Δ HRR = changes in heart rate recovery; $\dot{V}O_{2\max}$ = maximal oxygen uptake; $\dot{V}O_{2\max}$ = velocity obtained at $\dot{V}O_{2\max}$; GXT = graded exercise test; LnRMSSD = natural logarithm of the root-mean square of successive differences; LnRMSSDcv = coefficient of variation of baseline LnRMSSD; Δ LnRMSSD = changes in LnRMSSD.

Discussion

We aimed to 1) explore associations between changes in HRV, HRex and HRR variables 1 day post-HIIT and 2) determine how baseline characteristics (e.g., $\dot{V}O_{2\max}$, Baseline LnRMSSD, etc.) associated with HRV responses in women. A *large* but non-significant relationship was observed between Δ LnRMSSD standing and Δ HRex at 60% of $\dot{V}O_{2\max}$, and Δ LnRMSSD standing was associated with numerous baseline characteristics.

There were no group changes in LnRMSSD 1 day post-HIIT, although considerable inter-individual variation was observed (Δ LnRMSSD ranged by 1.0 and 0.6 in the supine and standing positions, respectively). Contrary to our findings, reductions in standing HRV among women have been reported 1 day following a 40 min HIE session at 85% of HRpeak (Kiviniemi et al., 2010). This discrepancy may be explained by the greater total HIE duration from the previous study, which was 13 min longer than the current protocol.

We observed no change or only *small* group changes in HRex 1 day post-HIIT, with considerable inter-individual variation in responses that diminished with increasing intensity (Δ HRex at 60, 70, 80 and 90% of $\dot{V}O_{2max}$ ranged by 22, 12, 8 and 6 $b \cdot min^{-1}$, respectively). HRex and HRR have been shown to be less variable as intensity increases, with ~90% of HRpeak yielding the least variation in HRex (Lamberts et al., 2004). Evidence of a potential association ($r=-0.55$) between Δ LnRMSSD and Δ HRex was observed only with standing values and only at the lowest submaximal intensity (60% $\dot{V}O_{2max}$), and correlation coefficients among Δ LnRMSSD standing and Δ HRex weakened as intensity increased. This may be explained by progressive vagal withdrawal via central command and greater sympathetic activation with increasing intensity from higher anaerobic demands and metaboreflex stimulation, resulting in less vagal influence over HR during and immediately post-exercise. It has been proposed that Δ LnRMSSD may only associate with Δ HRex at intensities below the ventilatory threshold (Blain et al., 2005; Buchheit, 2014). Our lowest submaximal intensity corresponded to 147.6 $b \cdot min^{-1}$, which is well-above VT in both non-habitual (128.2 $b \cdot min^{-1}$) and habitually-exercising women (141.6 $b \cdot min^{-1}$) (Swaine et al., 1995). Whether Δ LnRMSSD may predict Δ HRex at intensities $< VT$ is worthy of future investigation.

Aerobic fitness is an established predictor of cardiac-parasympathetic reactivation following exercise (Stanley et al., 2013). For example, women with high $\dot{V}O_{2max}$ exhibit significantly greater basal HRV and accelerated short-term (≤ 30 min) post-exercise autonomic recovery (Du et al., 2005). Our results extend these associations in women to intermediate-term recovery (1 day), as standing Δ LnRMSSD was significantly associated with $\dot{V}O_{2max}$, baseline LnRMSSD, LnRMSSDcv standing, and GXT HRR2. However, these relationships may be a result of multicollinearity as both HRR (Molina et al., 2016) and LnRMSSDcv (Flatt et al., 2017) have been associated with baseline LnRMSSD and may all be considered markers of cardiac-parasympathetic activity.

Conclusions

This study was limited by sample size and lack of intensities $< 60\% \dot{V}O_{2max}$. Our results indicate that Δ LnRMSSD in response to HIIT does not predict Δ HRex in women at moderate-high intensities, but may explain some of the variation in Δ HRex at intensities $< 60\%$ of $\dot{V}O_{2max}$. As nearly 75% of total training volume is accumulated below the VT in endurance athletes (Seiler & Kjerland, 2006), future research to examine whether daily changes in LnRMSSD predict HRex during lower intensity (i.e., $< VT$) exercise is of interest. Thus, an important next step will be to modify the current protocol such that intensities of the SET are performed relative to and below the VT. Women that were more aerobically fit and displayed higher and more stable baseline cardiac-parasympathetic activity demonstrated greater standing LnRMSSD recovery 1 day post-HIIT. Baseline standing LnRMSSD characteristics may therefore provide useful training prescription information regarding recovery from and responsiveness to HIIT in women. For example, individuals with lower LnRMSSD may not tolerate frequent use of HIIT within a training week due to slower cardiac-autonomic recovery between sessions.

Conflicts of interest

The authors have no conflicts to declare.

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