Impact of High-intensity Intermittent and Moderate-intensity Continuous Exercise on Autonomic Modulation in Young Men

Abstract

The aim of this study was to compare heart rate variability (HRV) recovery after two iso-volume (5 km) exercises performed at different intensities. 14 subjects volunteered (25.17 ± 5.08 years; 74.7 ± 6.28 kg; 175 ± 0.05 cm; 59.56 ± 5.15 mL·kg⁻¹·min⁻¹) and after determination of peak oxygen uptake (VO$_{2}$Peak) and the speed associated with VO$_{2}$Peak (sVO$_{2}$Peak), the subjects completed 2 random experimental trials: high-intensity exercise (HIE – 1:1 at 100% sVO$_{2}$Peak) and moderate-intensity continuous exercise (MIE – 70% sVO$_{2}$Peak). HRV and RR intervals were monitored before, during and after the exercise sessions together with the HRV analysis in the frequency domains (high-frequency – HF: 0.15 to 0.4 Hz and low-frequency – LF: 0.04 to 0.15 Hz components) and the ratio between them (LF/HF). Statistical analysis comparisons between moments and between HIE and MIE were performed using a mixed model. Both exercise sessions modified LFlog, HFlog, and LF/HF (F = 16.54, F = 19.32 and F = 5.17, p < 0.05, respectively). A group effect was also found for LFlog (F = 23.91, p < 0.05), and HFlog (F = 57.55, p < 0.05). LF/HF returned to resting value 15 min after MIE exercise and 20 min after HIE exercise. This means that the heavy domain (aerobic and anaerobic threshold) induces dissimilar autonomic modification in physically active subjects. Both HIE and MIE modify HRV, and generally HIE delays parasympathetic autonomic modulation recovery after iso-volume exercise.

Introduction

Heart rate variability (HRV) is considered a non-invasive and well-accepted method for measuring cardiovascular autonomic modulation [20]. The HRV recovery has been widely studied, since its reduction during rest and exercise are identified as a predictor of mortality and the risk of sudden death [13, 20]. More recently, HRV recovery has also been considered an important tool to evaluate stress after exercise sessions of different intensities [10, 24]. Seiler et al. [26] compared HRV recovery after different exercises in trained athletes. They found that iso-volume (i.e., 60 min) exercise performed between the aerobic and anaerobic thresholds, and above the anaerobic threshold, resulted in similar HRV recovery, indicating that high-intensity exercise (HIE) does not promote higher autonomic stress regarding return to baseline conditions compared to moderate-intensity exercise (MIE). According to Seiler et al. [26], this finding may explain why athletes prefer to train at a higher intensity, triggering greater physical capacity adjustments, with an autonomic modulation recovery similar to that observed in moderate-intensity training [9, 26]. Recently, studies have verified the effects of high-intensity compared to moderate-intensity training on body composition, physical fitness and molecular alterations; high-intensity training is usually more effective in improving these variables [4, 5, 12]. However, data concerning whether HIE and MIE exert different HRV recovery in physically active subjects is conflicting, because HIE intensities are usually too low and the volume is not matched [6, 16, 20, 23]. Thus, in contrast to highly trained athletes [8, 26], physically active subjects exhibit a different autonomic balance after iso-volume HIE and MIE, which is an important factor when selecting training distribution for this population.

Moreover, high-intensity training is often selected as it is time-efficient compared with moderate-intensity training [4, 12, 26]; however, the superiority of high-intensity training over...
moderate-intensity training could be even higher if an iso-
volume HIE is performed. On the other hand, this could lead to
higher autonomic stress in HIE compared to MIE, both during
exercise and recovery (i.e., retarding autonomic modulation
recovery), as both intensity and session time are greater, reduc-
ing the applicability of HIE for every-day training. However, this
hypothesis remains to be tested. Thus, the aim of the present
study.

Methods

Subjects
14 male subjects volunteered for the present study. They pre-
sented a health and neuromuscular status that demonstrated
their ability to complete the study protocol. All procedures per-
formed in the study were in accordance with the ethical stan-
dards of the University Research Ethics Committee for studies
involving human participants and met the ethical standards of
this journal described for Harriss and Atkinson [14]. Written
informed consent was obtained from all subjects after they had
been informed about the purpose and risks of the study.

Experimental design
Subjects completed 3 experimental trials at the laboratory. The
first visit aimed to determine peak oxygen uptake (VO2peak) and
the speed associated with VO2peak (sVO2peak). During the remain-
ing 2 visits, all participants were submitted to 2 protocols of
5 km of treadmill running in randomized sequence [28]: high-
intensity exercise (HIE), or moderate-intensity exercise (MIE),
separated by at least 72 h. Due to the influence of time of day on
HRV, all tests took place at the same time of the day, between
1:00 p.m. and 6:00 p.m., at a average temperature of between
20°C and 24°C [1]. The subjects were instructed to abstain from
strenuous exercise for at least 24 h prior to each exercise session
and were encouraged to maintain their usual nutritional and
hydration routines. Moreover, they were also requested not to
ingest stimulants (tea, coffee, soda, chocolate, chocolate powder)
or alcoholic beverages during this period.

Incremental test
The participants were subjected to an incremental test on a
treadmill (Inbramed MASTER CI, Inbrasport®, Porto Alegre, Bra-
zil). The initial speed was set at 8 km · h⁻¹, increasing by 1 km · h⁻¹
every 2 min until volitional exhaustion. Strong verbal encour-
agement was given during the test. The oxygen uptake was mea-
sured (Quark PFT, Cosmed®, Rome, Italy) throughout the test
and the average of the last 30 s as defined as VO2peak. The sVO2peak
was assumed as the final incremental test speed. When the sub-
ject was unable to complete a stage, the speed was expressed
according to the time in the final stage, determined as follows:
sVO2peak = speed of final complete stage + (time, in seconds,
remaining at the final incomplete stage / 120s) * 1 km.h⁻¹ [18].

High and moderate-intensity exercise sessions
For both exercise trials, the subjects performed a warm-up con-
sisting of running at 50% of sVO2peak for 5 min at 1% incline. The
HIE was performed intermittently with subjects running on a
treadmill for 1 min at 100% of sVO2peak [23], interspersed by
1 min of passive recovery (without exercise) until they had com-
pleted 5 km. The MIE consisted of a continuous 5-km run on the
treadmill at 70% of sVO2peak [23].

Heart rate variability recovery
Heart rate variability was monitored before, during and after
each exercise session. A recording strap was placed on the indi-
vidual’s chest at the sternal angle and at the Polar S8110i heart
rate receiver (Polar Electro, Finland) on their wrist. After place-
ment of the strap and monitor, the individuals remained at rest
in the supine position, breathing spontaneously for 20 min. After
this period, the individuals performed the exercise protocol
before returning to the supine position, at rest, breathing spa-
taneously, for 60 min. For HRV analysis, the behavior pattern
was recorded beat-to-beat throughout the experimental protocol,
with a sampling rate of 1000 Hz.

In order to evaluate HRV, data on the intervals between heart
beats (RR intervals) were sent to a microcomputer, from the
pulse receiver’s data transmission port to Polar Precision Per-
formance software (Polar Electro, Finland), using an infrared signal
interface. Only series with less than 5% errors were included in
the study. The RR interval series passed initially through filter-
ing using standard filter Polar Precision Performance software
(Polar Electro, Finland) [17], with a moderate filter, after which
visual inspection was performed of the temporal series of RR
intervals on the computer monitor to ensure the absence of arti-
facts that could interfere with HRV analysis. The HRV indices
were calculated in the time and frequency domains using Kubios
HRV software (Kubios, Biosignal Analysis and Medical Image
Group, Department of Physics, University of Kuopio, Finland)
[22].

The RR interval series were analyzed at the following moments:
M1 (after 5 min at rest), M2 (immediately after the exercise until
the 5th min of recovery), after the 5th min, the recovery period
was divided into 11 excerpts (M3 to M13) of 5 min each. All the
excerpts obtained contain at least 256 consecutive RR intervals.
In the time domain, the following indices were used for HRV
analysis: RMSSD and SDNN. The RMSSD index is defined as the
root mean square of successive differences between adjacent
normal RR intervals in a given time interval in milliseconds, and
the SDNN represents the standard deviation of normal to normal
RR intervals in milliseconds [30].

For the HRV analysis in the frequency domain, the high-
frequency (HF, 0.15 to 0.4 Hz) and low-frequency components (LF,
0.04 to 0.15 Hz), as well as the ratio between them (LF/HF), were
analyzed. The spectral analysis was calculated using the Fourier
Transform algorithm [30] after which the HRV parameters were
logarithmically transformed (Log) to control for skewed distri-
butions.

Statistical analysis
Statistical analysis was performed using SPSS 17.0. Prior to the
analysis, the normality of data was tested and confirmed using
the Shapiro-Wilk test. Comparison of HRV recovery between
moments, and between HIE and MIE was performed using a
mixed model. Exercise and moment were specified as fixed fac-
tors, and the subjects as a repeated factor. If a significant main
effect or interaction existed, this was further explored through
multiple comparison analyses with the Sidak adjustment for
multiple comparisons. The comparison between both exercise
characteristics was assessed using Student’s t test, with signif-
ificance level set at 5%.
Results

The subjects’ characteristics, anthropometry measures, summary of the incremental test and baseline HRV are shown in Table 1. The exercise characteristics are presented in Table 2. Significant differences were found between speed, total session time and total exercise time.

Resting HRV was similar between each exercise session. Both exercise sessions significantly modified SDNN and RMSSD (F = 18.45, F = 14.42, p < 0.05; respectively; Fig. 1), as well as LF log, HF log and LF/HF (F = 16.54, F = 19.32, and F = 5.17, p < 0.05; respectively, Fig. 2). A group effect was also found for SDNN (F = 12.16, p < 0.05), RMSSD (F = 43.18, p < 0.05), LF log (F = 23.91, p < 0.05) and HF log (F = 57.55, p < 0.05). Nevertheless, no interaction (group vs. moment) was observed.

For MIE, the SDNN returned to rest values after 20 min of recovery, while the RMSSD returned after 30 min. Both the LF log and HF log returned to rest values at the 20th min of recovery. After the HIE, the SDNN returned to the rest values only after 45 min, while the RMSSD did not recover over the 60 min. Moreover, the LF log and HF log recovered after 20 and 35 min, respectively. The LF/HF returned to rest values 15 min after MIE exercise and 20 min after HIE exercise.

Discussion

The aim of the present study was to compare HRV recovery after 2 iso-volume (5 km) exercises performed at different intensities. The main finding of the present study was that both iso-volume HIE and MIE modified HRV, and that HIE delayed HRV recovery compared with MIE. These results mean that from the heavy domain (i.e., between the aerobic and anaerobic threshold) onward, iso-volume exercise induces dissimilar autonomic modification in physically active subjects.

During exercise, general HRV decreases (i.e., parasympathetic withdraw and sympathetic excitation) with the aim of increasing heart rate [13]. After exercise, the return of HRV to rest level delays according to exercise intensity, duration and modality [6,20,26]. Furthermore, HRV recovery is also used to characterize the acute stress response to training sessions in trained individuals [3,26], which contributes to training prescription. Thus, since HIE and MIE training were compared in physically active subjects [12,28], understanding the behavior of HRV recovery after these training sessions may also contribute to training prescriptions for physically active subjects or for optimizing exercise training sessions for individuals who present cardiovascular disease. Stewart et al. [29] have verified that HRV recovery is delayed after another range of exercise intensity (at anaerobic threshold), and duration (2 h), evidencing the applicability of HRV recovery to differentiate autonomic recovery after different stimuli. Indeed, HRV obtained by both cardiac monitor and electrocardiography is also suggested to be used in different viewpoints [19].

It is important to note that SDNN increased immediately after both exercises. SDNN index, which represents the standard deviation of normal to normal RR intervals and reflects the overall variability [30], had a significant increase immediately after exercise until the 5th min of recovery compared to baseline. During the recovery from the exercise period, the initial return of heart rate to baseline occurs primarily due to parasympathetic reactivation [7,15]. With cessation of exercise, there is loss of central command, and the baroreflex activation and other mechanisms contribute to the parasympathetic activity increase [15]. This parasympathetic reactivation promotes increase in RR intervals variation, which may be associated with SDNN index increase immediately after exercise.

Table 1 Mean, standard deviation and 95% of confidence interval (95% CI) for subjects characteristics and heart rate variability indices.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Subjects (n = 14)</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>25.17 ± 5.08</td>
<td>22.50–27.83</td>
</tr>
<tr>
<td>Body Mass (kg)</td>
<td>74.70 ± 6.28</td>
<td>71.41–77.98</td>
</tr>
<tr>
<td>Height (m)</td>
<td>1.75 ± 0.05</td>
<td>1.72–1.77</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>24.42 ± 1.91</td>
<td>23.41–25.42</td>
</tr>
<tr>
<td>VO₂peak (ml · kg⁻¹ · min⁻¹)</td>
<td>59.56 ± 5.15</td>
<td>56.86–62.25</td>
</tr>
<tr>
<td>Rest HR (bpm)</td>
<td>61.50 ± 8.07</td>
<td>57.27–65.72</td>
</tr>
<tr>
<td>RMSSD (ms)</td>
<td>58.15 ± 18.92</td>
<td>48.23–68.06</td>
</tr>
<tr>
<td>SDNN (ms)</td>
<td>68.50 ± 16.10</td>
<td>60.06–76.93</td>
</tr>
<tr>
<td>LF Log</td>
<td>3.04 ± 0.30</td>
<td>2.88–3.19</td>
</tr>
<tr>
<td>HF Log</td>
<td>2.99 ± 0.29</td>
<td>2.83–3.14</td>
</tr>
<tr>
<td>LF:HF (a.u.)</td>
<td>1.45 ± 1.03</td>
<td>0.91–1.98</td>
</tr>
</tbody>
</table>

Values are mean ± standard deviation. BMI = body mass index; VO₂peak = peak oxygen uptake; HR = heart rate; RMSSD = root mean square of successive differences; SDNN = standard deviation of NN intervals; LF = low frequency; HF = high frequency; LF:HF = LF/HF ratio

Table 2 Mean and standard deviation (SD) of high-intensity exercise (HIE) and moderate-intensity exercise (MIE) characteristics (n = 14).

<table>
<thead>
<tr>
<th>Variable</th>
<th>HIE</th>
<th>MIE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Speed (km·h⁻¹)</td>
<td>14.51 ± 1.19</td>
<td>10.16 ± 0.83 *</td>
</tr>
<tr>
<td>Total session duration (minutes)</td>
<td>40.72 ± 3.39</td>
<td>29.72 ± 2.42 *</td>
</tr>
<tr>
<td>Total exercise duration (minutes)</td>
<td>20.86 ± 1.70</td>
<td>20.86 ± 1.70</td>
</tr>
</tbody>
</table>

* significantly different from HIE

Fig. 1 Heart rate variability recovery of time domain indices (a SDNN and b RMSSD) after MIE (●), and HIE (■). * significantly different from rest value for MIE; ϕ significantly different from rest value for HIE.
Martinmäki and Rusko analyzed short-term HRV recovery (10 min) after high-intensity and low-intensity exercise and found that the intensity delayed parasympathetic recovery after high-intensity exercise [20]. However, the high-intensity exercise was performed at 61% of maximal power, which is lower than the MIE in the present study. Mourot et al. compared a constant and interval exercise with similar physical workloads [21]. They found slower parasympathetic recovery (i.e., HF) up to 60 min after the interval exercise. Compared with the present study, these studies presented either lower intensity of exercise or session volume [21]. Aiming to understand the effect of both intensity and volume, Kaikkonen et al. compared HRV recovery after 3 iso-volume different constant-speed exercises: low, moderate and high-intensity [16]. The authors found no difference in HRV recovery after moderate and high-intensity exercise; however, the Kaikkonen et al. study also presented an intensity lower than the present study (74% vs. 100% of sVO2peak) [16].

When comparing the effects of HIE and MIE, HIE seems to induce better or similar physical fitness adaptations than MIE, with significantly lower volume [5,12]. Therefore, HIE training would induce even higher adaptation, if performed with the same MIE volume; however, this would likely induce greater autonomic stress after the HIE. The present study demonstrates that both iso-volume HIE and MIE likely reduced parasympathetic control (RMSSD) and increased sympathetic predominance (high LF/HF ratio) [3]. However, the group effects for all variables indicate that, in general, HIE delays parasympathetic autonomic modulation recovery when compared with iso-volume MIE. These results contradict Seiler et al.’s study, in which similar HRV responses were verified after 2 exercise sessions performed at and above anaerobic threshold intensity with the same total session time [26]. 2 factors may contribute to the differences: (i) the subject’s training status, since they evaluated highly trained athletes; and (ii) we equalized exercise session volume by distance covered instead of session time.

In contrast to highly trained athletes [8,25], physically active subjects do exhibit different HRV recovery after iso-volume HIE and MIE; this is important for selecting training distributions for this population. Given the increasing number of investigations comparing different aspects of high and moderate-intensity training (physiological, morphological, and performance), it would also be interesting to understand whether higher autonomic stress after HIE leads to different HRV adaptations. Moreover, it is not known whether HIE accumulation in training interferes with training commitment.

It is important to highlight that even though the exercise volume was the same, the training load was not. Banister et al. developed the concept of training impulse – based on heart rate response to training – as a method for integrating training variables [2], and Foster et al. proposed the calculation of training impulse through the rating of perceived effort (RPE) [11]. Both methods help to understand external loads (volume: minutes, kilometers, and repetitions) and internal loads (heart rate, lactate, and rating of perceived effort) [3].

It is likely that the HIE training impulse is higher than MIE, which would influence HRV response. This issue is a limitation of the present study. Moreover, we did not control breathing frequency during the HRV analysis, which influences HFlog values [25]; however, even after 60 min of recovery, we found modifications in HRV related to the parasympathetic branch.

In summary, both HIE and MIE modify HRV, and generally HIE delays parasympathetic autonomic modulation recovery after an iso-volume exercise. This delay may be taken into account when prescribing training for physically active subjects, especially since RMSSD – indicating parasympathetic control – did not recover after 1 h. Future studies should verify whether iso-training loads HIE and MIE also present different HRV recovery values.

Acknowledgements
Eduardo Zapatero Campos thanks CNPq for their support (401676/2014-5). Fabio Santos Lira thanks Fapesp for their support (2013/25310-2).

Conflict of interest: None.

Affiliations
1 Exercise and Immunometabolism Group, Physical Education Department, Universidade Estadual Paulista, Presidente Prudente, Brazil
2 Physiotherapy Graduate Program, Physiotherapy Department, Universidade Estadual Paulista, Presidente Prudente, Brazil
3 Faculty of Physical Education, University of Campinas, Campinas, Brazil

References
30 Vanderlei LCM, Pastre CM, Hoshi RA, Carvalho TD, Godoy MF. Basic notions of heart rate variability and its clinical applicability. Rev Bras Cir Cardiovasc 2009; 24: 205–221