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ABSTRACT

Heart rate variability (HRV) is widely considered as a non-invasive method of evaluating autonomic influence on the cardiac rhythm. Following exercise, through HRV assessment, acute manipulations of autonomic influence have been demonstrated, but the influence of exercise intensity remains unclear. **Objective:** The aim of the present study was to investigate the influence of exercise of two different intensities on HRV response following exercise. **Design:** Sixteen runners completed two distance-matched conditions; moderate (90% of the speed at gas exchange threshold, MOD) and severe (1 km.h^{-1} below the speed at maximal oxygen uptake, SEV) intensity interval training sessions (6 x 800m runs with 3 min recovery intervals) to study post exercise HRV. At one hour prior to (-1 h) and at +1 h, +24 h, +48 h and +72 h following each exercise session participants sat quietly for 20 min whilst breathing at 0.20 Hz. Resting HRV data was collected over the final 5 min of each 20 min period, followed by measurement of arterial blood pressure. **Results:** Time domain indices and high frequency component showed a significant decrease ($p<0.001$) between -1 h and +1 h in SEV and low frequency component in normalised units significantly increased ($p<0.01$). Systolic blood pressure significantly decreased ($p = 0.001$) for SEV at +1 h compared with -1 h. No changes were demonstrated at +24 h, +48 h, +72 h for any outcome or any condition compared with -1 h. **Conclusions:** These findings suggest that severe intensity exercise is required to elicit a change in HRV outcomes post exercise, resulting in a reduction in parasympathetic influence on the heart at +1 h, with values returning to baseline by +24 h.

Key words:

Autonomic nervous system; parasympathetic; sympathetic.

Introduction

Heart rate variability (HRV) has been established as a useful non-invasive method of evaluating autonomic influence on the cardiac rhythm.[1] It is widely accepted that regular exercise training induces adaptations in HRV (i.e., the oscillation in the interval between consecutive heartbeats) outcomes with a shift of autonomic balance towards higher parasympathetic activity, consistent with improved cardiac health.[1]

Less attention has been paid to the HRV response following a single bout of exercise, but there is a growing body of evidence based on studies of trained athletes, [2-4] trained students, [5] moderately trained, [6,7] detrained [8] and untrained active participants. [3,9-12] Findings indicate that single bout of exercise may result in an increase in sympathetic influence at one hour post exercise cessation [2,3,5,7,9,10,12] and that by 24 h post exercise sympathetic influence returns to baseline levels. [2,7,12] Such findings are of interest, since an elevated sympathetic influence has been associated with increased risk of cardiac events and compromised health. [13-15]

Following an initial suppression of parasympathetic influence at one hour post exercise cessation, elevated parasympathetic influence has been observed at 48 h following prolonged exercise. [4] In contrast, in one study, moderate intensity exercise has been associated with elevations in parasympathetic influence within an hour after exercise cessation. [11] This is an interesting finding, since such changes are known to have a cardio-protective effect. [16]

Even though different studies have examined HRV responses at different intensities, [7,10,12,17] to the best of our knowledge only one study by Mourot et al has directly explored the impact of exercise intensity over 48 h following exercise cessation. [7] In this study, both the moderate and high intensity exercise resulted in reduced parasympathetic influence at one hour following exercise cessation, and there appeared to be no notable differences between exercise intensity condition in the time course of the response up to 48 h post exercise. However, the study by Mourot et al [7] included a potential confounding variable, since participants undertook continuous exercise for the moderate intensity condition and interval exercise for the severe intensity condition. It is therefore difficult to be certain whether the findings were a result of the exercise intensity or exercise type (*i.e.*, continuous versus interval). To our knowledge no previous studies have examined the effect of exercise intensity, using a fixed exercise type, on HRV response.

Therefore, the aim of the present study was to investigate the influence of exercise of two differing intensities (moderate and severe) on the HRV response post-exercise. Both exercise conditions were applied as interval exercise, and the distance covered was consistent between conditions. We were interested in the nature of the response over 72 h following cessation of exercise, and the contrast in the response following the differing exercise intensities.

Methods

Participants

Sixteen (14 male; 2 female) experienced (8.8 ± 5.3 years of training) runners involved in regular endurance training sessions (3.7 ± 1.1 session.wk^{-1}) volunteered to participate (Table 1). All participants completed a health screening procedure and were fully informed of the nature of the study. Participants then provided written consent to participate. All procedures were approved by the University Research Ethics Committee. Participants had no history of diabetes, hypertension or cardiovascular disease, no symptoms of underlying disease, and received no medication during the course of the study for any diagnosed condition.

Study Design

A moderate (MOD) and severe (SEV) intensity condition were administered in a counterbalanced order within a fully repeated measures design. Both conditions consisted of six 800 m bouts of running with 3 min recovery intervals. The MOD was completed at 90% of the velocity at gas exchange threshold (v-GET). The SEV was completed at a velocity corresponding to 1 km·h^{-1} below the velocity at maximal oxygen uptake (v-VO$_{2max}$). A minimum of 72 h separated each condition and participants undertook no exercise for 72 h prior to the first test within each condition.

One hour prior to (-1 h) and at various intervals following (+1 h, +24 h, +48 h, +72 h) each interval exercise session, participants sat quietly for 25 min whilst HRV and blood pressure were recorded. Participants were instructed not to consume alcohol or caffeine for 24 h prior to each assessment, and not to consume food or fluid other than water "*ad libitum*" in the 4 h prior to each assessment. In addition to performing no exercise for 72 h prior to the first assessment, participants were instructed to perform no exercise between subsequent assessments. All tests were conducted at the same time of day for each participant in a comfortable temperature controlled (temperature 19 ± 2.0 ºC; humidity 34 ± 6 %; barometric pressure 766.2 ± 7.7 mmHg) exercise physiology laboratory. Prior to exposure to the first condition, participants attended the laboratory for a familiarization visit. In addition to familiarizing participants with the procedures for the determination of HRV and blood pressure outcomes, participants' descriptive anthropometric and physiological characteristics were assessed, including the determination v-GET and v-VO_{2max}.

HRV Outcome Measures

According to the Task Force of the European Society of Cardiology [1] there are a variety of indices that are used to assess HRV, which can be divided into two major categories: time domain indices and frequency domain indices. In the present investigation the included time domain indices are the mean NN interval (MNN) (*i.e.*, normal-to-normal intervals between adjacent QRS complexes), the standard deviation of the NN intervals (SDNN), square root of the mean sum of squares of the differences between adjacent NN intervals (rMSSD), and proportion of pairs of adjacent NN intervals differing by more than 50 ms (pNN50). The included frequency domain indices are: high frequency (HF), HF in normalized units (HF_{nu}), low frequency (LF), LF in normalized units (LF_{nu}), very low frequency (VLF), total power (TP) and LF:HF ratio.

Procedures

During the HRV assessment points at -1 h, +1 h, +24 h, +48 h and +72 h, participants sat quietly for 20 min and controlled their breathing frequency. Frequency was set at 0.20 Hz (12 breath.min^{-1}), [18] with each breath comprising 2 s of inspiration and 3 s of expiration. [5] Breathing frequency was paced using a computer-based metronome providing both audio and visual cues. Participants were instructed to maintain a normal depth of breathing throughout. Participants wore a chest strap consisting of two electrodes and a transmitter (T61, Polar Electro Oy, Kempele, Finland). The NN interval data were transmitted to a watch receiver (Polar S810i series) by coded short-range telemetry and then to a computer via an infrared interface (Polar Electro Oy, Kempele, Finland) for storage. NN interval data were collected over the final 5 min of the 20 min period and stored for subsequent analysis. The data were presented graphically and visually inspected to identify any spurious beats. No spurious beats were identified, and this was confirmed with the error detection algorithm in the Precision Performance analysis programme (Polar Electro Oy, Kempele, Finland) which filters the data using median and moving average based methods in order to identify artefacts in the signal. During a further 5 min period, participants continued to sit quietly whilst three repeat measurements of arterial blood pressure were made using a clinical grade automated sphygmomanometry (DynaPulse DP-200M, Pulse Metric Inc., San Diego, U.S.A) according to the approach of Forjaz et al. [19] with two minutes between each measurement. Collected data were transmitted via the communication cable to a computer for storage and analysis (DynaPulse software version 3.8).

The progressive exercise test and the two interval exercise sessions were conducted on a motorised treadmill (Ergo ELG 70, Woodway, Weil am Rhein, Germany) and for participant comfort laboratory air was circulated using a floor mounted fan. Prior to the progressive exercise test participants undertook a five minute warm-up at a self selected speed below v-GET and completed their personal stretching routine. In accordance with the recommendations of Buchfuhrer et al. [20] starting speed was selected so that exhaustion was observed at 10 ± 2 min. Participants initiated running when the treadmill had reached the pre-determined start speed of 8 $km \cdot h^{-1}$. Following two minutes at the first speed, treadmill speed increased 1.2 $km \cdot h^{-1} \cdot min^{-1}$ (0.16 $km \cdot h^{-1}$ every 8 s) at a 0% gradient. The progressive exercise test was used to determine VO_{2max}, v-VO_{2max}, GET, v-GET and HR_{max}. Throughout the test HR was measured using a two-electrode chest strap and data was transmitted by coded short-range telemetry to a watch receiver (S810i series, Polar Electro Oy, Kempele, Finland) for storage. During the progressive test, expirate was collected continuously and analysed using a conventional Douglas bag technique. [21] The procedure for determination of VO_{2max}, GET and the associated velocities has been described previously. [21]

Data Analysis

Prior to analysis, the NN interval time-series were interpolated at a rate of 4 Hz and detrended using the smoothness priors approach. NN interval data were analysed using HRV Analysis Software (version 1.1 for Windows, Biomedical Signal Analysis Group, University of Kuopio, Finland).[22] A continuous five minute data segment was selected for analysis consistent with previous studies [5,11,23] since spectral approaches are conventionally performed on stationary records of 200-500 consecutive heart beats.[24] NN intervals were at a steady level which is a requirement for the application of spectral analysis.[25] Power spectrum analysis was undertaken using the autoregressive method. Autoregressive coefficients were estimated using the forward-backward-linear-least-squares-algorithm with a fixed model order of 18. Frequency bands were selected conventionally [1]: VLF 0.00-0.04 Hz; LF 0.04-0.16 Hz and HF 0.16-0.40 Hz. TP and LF:HF ratio was also computed. The LF and HF power components were also expressed using normalized units (nu) providing a relative value for each power component.[1] The normalization procedure requires dividing either HF or LF component (ms^2) by total spectral power (TP) minus the VLF component (ms^2) [1] which results in a dimensionless ratio.[5] Normalization is suggested to result in a reduction of the effect total power changes would have on LF and HF components.[1,26] HRV outcomes in the frequency domain are not normally distributed [27] so, consistent with the approach of Bernardi et al. [2] and James et al., [5] these data were transformed using the natural logarithmic function prior to statistical analysis. The logarithmic transformation provides data that meets the assumptions required for parametric statistical analysis. [11] Consistent with James et al., [5] all data are presented as mean (68% confidence interval) as it is not possible to 'back-transform' a log transformed standard deviation into the original measurement unit. The presentation of data as mean (68% confidence interval) is consistent with the normal convention of presenting data as mean (one standard deviation).

Interactions between condition (exercise intensity; SEV or MOD) and time (-1 h, +1 h, +24 h, +48 h, +72 h) were explored using 2 x 5 fully repeated measures ANOVA. Whether or not a significant interaction was present, we were also interested in main effects for time, since we were interested in the response following each condition. One-way repeated measures ANOVA were conducted for each condition to examine effects over time. When significant main effects were observed, post-hoc t-tests were conducted to locate the differences. Differences were considered significant when $p < 0.05$.

Results

Participant characteristics are presented in Table 1. The heart rate responses to the moderate and severe interval exercise bouts are shown in Figure 1. The mean (68% confidence interval) heart rate during the moderate exercise demonstrated a slight increase from 131 (123-139) to 139 (128-150) b·min^{-1} across the six 800 m bouts (i.e., 68-72 $\%\text{HR}_{\text{max}}$). Heart rate during the severe exercise increased significantly ($p < 0.01$) from 165 (156-174) to 178 (167-189) b.min^{-1} across the six bouts (i.e., 86-93% HR_{max}).

Data for the time domain and frequency domain HRV parameters are presented in Table 2 and 3, respectively.

Time domain outcomes. Interactions between condition and time were revealed for SDNN, rMSSD and pNN50, but not for MNN. Post-hoc analysis revealed lower values for SEV than MOD at +1 h ($p < 0.01$) for all time domain outcomes (MNN, 11%; SDNN 31%; rMSSD 12%; pNN50 55%). Main effects for time were revealed for SEV for all time domain outcomes ($p < 0.001$) but not for MOD. Compared to the values at -1 h, values at +1 h were significantly ($p < 0.001$) decreased for SEV condition (MNN, 20%; SDNN, 39%; rMSSD, 54%; and pNN50, 68%) but values returned to baseline levels by +24 h (Table 2).

Frequency domain outcomes*.* A significant interaction ($p = 0.001$) between condition and time was observed for TP, with the value at +1 h significantly lower (50%, $p = 0.003$) for SEV (404ms^2) than MOD (810ms^2).

A main effect for time for SEV was revealed for TP ($p < 0.001$), HF_{ms}^2 ($p < 0.001$), LF_{nu} ($p = 0.003$) and HF_{nu} ($p = 0.021$), with post-hoc analysis revealing a significant decrease for TP (64%, $p < 0.001$), HF_{ms}^2 (78%, $p < 0.001$) and HF_{nu} (47%, $p = 0.008$) respectively, and a significant increase for LF_{nu} (42%, $p = 0.007$) between -1 h and +1 h, but all of these values had returned to baseline by +24 h. No condition by time interaction was revealed (Table 3).

No interaction between condition and time, and no main effects for time were revealed for the LF: HF ratio (Table 3).

Blood pressure outcomes*.* Although no interaction was revealed between condition and time for either systolic or diastolic blood pressures, a main effect for time was revealed for systolic blood pressure for SEV exercise, with post hoc analysis indicating a significant decrease (7%, p = 0.001) at +1h compared with -1h but values had returned to baseline by +24 h (Table 4).

Discussion

Although an increasing number of studies have investigated the HRV response following a single bout of exercise, the majority of these studies have included either one exercise intensity, [5,8,9,11] or those that have included different intensities [7,10,12,17] have not assessed HRV after long term recovery (i.e., >24h). This growing body of evidence is further compounded, for comparative purposes, by the diverse range of exercise bouts and the methodological differences between the studies. Therefore, to our knowledge, the present study is the first to explore the influence of exercise intensity alone on the post-exercise HRV response in a controlled design.

The findings of the present study suggest that severe intensity exercise reduces indicators of parasympathetic influence on the heart (MNN, SDNN, rMSSD, pNN50, HF, HF_{nu}) and increases an indicator of sympathetic influence on the heart (LF_{nu}) at +1 h following the exercise bout. Interestingly, these changes were observed in the presence of a reduction in systolic blood pressure. All changes were reversed by +24 h, and values remained constant at +24h, +48 h and + 72 h at the baseline (i.e., -1 h) level.

An increase in sympathetic influence on the heart between 30 min and one-hour post exercise cessation has been noted in a number of studies. [2,3,7,9] A decrease in parasympathetic influence on the heart between 30 min and one-hour post exercise cessation has also been noted in a number of studies with a decrease in the HF component of the total power spectrum consistently observed. [2,3,5,7,9] Reductions in other indicators of parasympathetic influence have also been observed, including HF_{nu} [3,9] and SDNN. [2,5]

It is possible that the elevation in sympathetic influence and accompanying reduction in parasympathetic influence on the heart at one-hour post severe intensity exercise cessation in the present study was a compensatory response to a reduced systolic blood pressure. A reduced systolic blood pressure at one hour following exercise cessation has been reported previously in normotensive participants. [28] It is interesting that, even in the presence of an increased sympathetic influence and an associated decrease in MNN in the present study, blood pressure was not restored to baseline values. We cannot be sure that the increased heart rate (i.e., reduced MNN) provided an increased cardiac output, so the shortfall in systolic blood pressure at one hour is either a result of reduced stroke volume (and therefore cardiac output) or reduced vascular resistance. A reduced stroke volume at rest has been reported following a single bout of exercise, [29] although typically such reductions have been demonstrated following very prolonged exercise. [29] Reduced arterial resistance, as indicated by increased arterial diameter, has also been reported at one-hour post exercise. [30]

In many of the previous studies in this area, it is difficult to determine the exact exercise intensity from the information provided, not least due to the lack of data regarding the physiological response to the exercise bout. Perhaps this is not surprising, given that these studies were not primarily designed to compare the responses following exercise of differing intensity. Mourot et al [7] compared continuous and interval exercise where the work done was fixed at 9.4 $kJ.kg^{-1}$. In the present study, interval exercise was conducted at 1 $km.h^{-1}$ below the velocity at VO_{2max} (i.e., severe intensity) and 90% of the velocity at GET (i.e., moderate intensity), respectively. It was important to ensure that the exercise that took place was genuinely of a moderate and severe intensity respectively, thereby ensuring significantly different exercise intensities in the two conditions. The physiological responses to exercise at these two intensities confirmed the differing nature of the physiological challenge, where 86-93 $\%HR_{max}$ was attained in the severe intensity exercise condition as opposed to 68-72 $\%HR_{max}$ in the moderate condition. It was considered particularly important to ensure that the moderate intensity condition was completed at a speed below that attained at gas exchange threshold.

The findings of the present study have implications for practice. In particular, it appears that severe intensity exercise may acutely alter the autonomic influence on the heart post-exercise towards a greater sympathetic influence. This suggests that for the duration of this changed autonomic influence, it is likely that the exercise participant is at an increased risk of cardiac events such as arrhythmias. Although it is well recognised that the acute risks of conducting high intensity exercise are far outweighed by the chronic benefits of regular moderate exercise in the healthy population, [31] a cautious approach might be necessary in higher risk populations particularly when exercise intensity exceeds a moderate intensity. It is also important to note that in the present study, a moderate intensity exercise bout had no influence on post-exercise HRV at +1 h compared with baseline values. It would therefore appear that moderate intensity exercise (86-93 $\%HR_{max}$) may present less of an acute risk.

In summary, the findings of the present study demonstrate a clear intensity effect on the post-exercise heart rate variability response, where severe intensity exercise results in a reduced parasympathetic influence on heart rhythm at one hour after exercise cessation. In contrast, moderate intensity exercise results in no change in either sympathetic or parasympathetic influence on the heart. Future studies should explore the time course of the heart rate variability response following severe intensity exercise, particularly between one hour and 24 h following exercise cessation.

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