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Original research

Influence of exercise modality on cardiac parasympathetic and sympathetic indices during post-exercise recovery

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ABSTRACT

Objectives: This study investigated indirect measures of post-exercise parasympathetic reactivation (using heart-rate-variability, HRV) and sympathetic withdrawal (using systolic-time-intervals, STI) following upper- and lower-body exercise.
Design: Randomized, counter-balanced, crossover.
Methods: 13 males (age 26.4 ± 4.7 years) performed maximal arm-cranking (MAX-ARM) and leg-cycling (MAX-LEG). Subsequently, participants undertook separate 8-min bouts of submaximal HR-matched exercise of each mode (ARM and LEG). HRV (including natural-logarithm of root-mean-square-of-successive-differences, Ln-RMSSD) and STI (including pre-ejection-period, PEP) were assessed throughout 10-min seated recovery.
Results: Peak-HR was higher ($p = 0.001$) during MAX-LEG (182 ± 7 beats min^{-1}) compared with MAX-ARM (171 ± 12 beats min^{-1}), while HR ($p < 0.001$) and Ln-RMSSD ($p = 0.010$) recovered more rapidly following MAX-ARM. PEP recovery was similar between maximal bouts ($p = 0.106$). HR during submaximal exercise was 146 ± 7 (LEG) and 144 ± 8 beats min^{-1} (LEG) ($p = 0.139$). Recovery of HR and Ln-RMSSD was also similar between submaximal modalities, remaining below baseline throughout recovery ($p < 0.001$). PEP was similar during submaximal exercise (LEG 70 ± 6 ms; ARM 72 ± 9 ms; $p = 0.471$) although recovery was slower following ARM ($p = 0.021$), with differences apparent from 1- to 10-min recovery ($p \leq 0.036$). By 10-min post-exercise, PEP recovered to baseline (132 ± 21 ms) following LEG (130 ± 21 ms; $p = 0.143$), but not ARM (121 ± 17 ms; $p = 0.001$).
Conclusions: Compared with submaximal lower-body exercise, HR-matched upper-body exercise elicited a similar recovery of HR and HRV indices of parasympathetic reactivation, but delayed recovery of PEP (reflecting sympathetic withdrawal). Exercise modality appears to influence post-exercise parasympathetic reactivation and sympathetic withdrawal in an intensity-dependent manner. These results highlight the need for test standardization and may be relevant to multi-discipline athletes and in clinical applications with varying modes of exercise testing.

1. Introduction

Autonomic dysregulation is associated with a range of poor health and functional outcomes.[1,2] Quantification of beat-to-beat fluctuations in heart rate (HR), termed heart rate variability (HRV), has emerged as a popular tool with which to assess autonomic activity, in particular cardiac parasympathetic neural activity (cPNA).[3–5] HRV has classically been investigated under resting conditions. However, consistent with the 'reactivity hypothesis',[6] monitoring autonomic responses to a stressor (challenge test) may be of prognostic/diagnostic value. As an indicator of 'parasympathetic reactivation' following dynamic exercise stress, the immediate HR recovery is a strong predictor of all-cause mortality[7] and has also been used to monitor athletic training loads.[1] HRV has also been utilized to monitor the time-course of parasympathetic reactivation following an exercise challenge test.[3,5]

When interpreting post-exercise autonomic responses, it is important to consider the influence of the exercise dose. HRV is strongly affected by exercise intensity, with higher intensities eliciting slower HRV recovery.[5,8] However, very few studies have investigated the effect of dynamic exercise modality. A more rapid HR[9,10] and HRV[11,12] recovery has been reported following exercise of a smaller muscle mass and/or lower energy expenditure.

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As these reports have all investigated indices of parasympathetic reactivation following maximal incremental exercise, it is not clear how exercise modality may affect HRV recovery after submaximal efforts.

Furthermore, although certain HRV measures are sometimes interpreted either as indices of sympathetic activity or 'sympathovagal balance',[13] these are controversial and are not widely accepted. Systolic time intervals (STI), in particular the pre-ejection period (PEP), are inversely associated with sympathetic influences on myocardial contractility, and have previously been employed as non-invasive indices of cardiac sympathetic neural activity (cSNA).[4] The measurement of post-exercise STI may provide quantitative assessment of dynamic cardiac sympathetic withdrawal following exercise, to complement HRV measures of parasympathetic reactivation. While a higher preceding exercise intensity has been shown to elicit a slower STI post-exercise recovery,[14,15] it has not been established how STI recovery is affected following different modalities of acute exercise.

Therefore, it is currently not clear as to what extent differences in exercise modality may confound the interpretation of results when investigating post-exercise autonomic recovery, particularly following submaximal exercise. The purpose of this study was to investigate the post-exercise responses of indices of cPNA and cSNA to two different dynamic exercise modalities – upper-body arm-cranking versus lower-body cycling – when performed at HR-matched submaximal intensities. Since these modes of exercise were likely to result in differening descending and ascending neural inputs regulating cardiac autonomic activity, it was hypothesized that non-invasive autonomic indices might demonstrate distinctly different recovery patterns following upper-body versus lower-body exercise.

2. Methods

Thirteen healthy males (age 26.4 ± 4.7 years; height 1.75 ± 0.06 m; mass 80.7 ± 9.4 kg; peak oxygen uptake 47.1 ± 9.9 ml kg^{-1} min^{-1}) volunteered for this study, which had been previously approved by the University of Sydney Human Research Ethics Committee (HREC reference: 2014/894). Written informed consent was obtained from all individuals. The participants completed two preliminary assessments, followed by two constant-load exercise tests of different modalities on separate days in a randomized order. All four visits were performed at the same time of day, over a period of 2–4 weeks and were separated by at least 72 h.

During the first preliminary visit, participants performed a maximal graded cycling exercise test (MAX-LEG) on an Ergoline 800 electrically-braked cycle ergometer (Ergoline, Bitz, Germany) to determine their peak oxygen uptake (VO_2peak). Initial power was 50 W, followed by 30 W increments every three minutes until volitional fatigue. Respiratory gases were continuously measured (Ultima Cardio$_2$, Medical Graphics Corp, St. Paul, MN, USA). Immediately following exercise (within 5 s), participants were re-seated in a chair and recovered passively for 10 min with instructions to remain motionless without talking. Within 1 week, participants returned to the laboratory for the second preliminary visit to perform a maximal graded exercise test (MAX-ARM) on an arm-crank ergometer (Ergomed 840L, Siemens, Erlangen, Germany). The protocol was identical to that of the cycle test, except that initial power was set at 30 W, with 15 W increments.

For the submaximal constant-load trials, participants performed two 8 min exercise bouts of different modalities – cycling (LEG) and arm-cranking (ARM) – in a randomized order during different visits. For each visit, participants underwent a 10 min seated rest period, then exercised for 8 min to achieve a pre-determined steady-state target HR for at least 3 min of the 8 min bout. Since HR per se can influence HRV,[16] the target HR for both modes of exercise was the same absolute HR. However, because peak-HR is typically 10–20 beats min^{-1} lower during incremental arm-cranking compared with cycling,[9] the absolute target HR was that which corresponded to the average of 70% HR reserve for each mode of exercise ('mixed-mode HR reserve'). Using this approach, the exercise bouts were HR-matched but also performed at close to the desired relative intensity, i.e. 70% mode-specific HR reserve. Post-exercise recovery was the same as the maximal tests (10 min seated, motionless without talking).

Beat-to-beat heart rate (RS800, Polar Electro, Kempele, Finland) was continuously recorded with 1 ms resolution throughout rest, exercise, and recovery. The R–R interval (RRI) tachogram was visually inspected and manually edited for ectopic beats and artifacts. Immediate HR-recovery was calculated as the absolute difference between HR at end-exercise and at 1 min recovery (HRR_{60}).[7] HRV was analyzed using Kubios HRV (Version 2.1, Biosignal Analysis and Medical Imaging Group, Kuopio, Finland). The primary HRV outcome was the root mean square of successive differences (RMSSD), calculated during successive 30 s epochs with a 3-value rolling-median filter applied. This form of HRV analysis provides a quantitative dynamic assessment of post-exercise cPNA, i.e. parasympathetic reactivation.[3] Frequency-domain analysis was performed using the autoregressive method with a 16th order model. Low frequency power (LF, 0.04–0.15 Hz), high frequency power (HF, 0.15–1.50 Hz) and total power (TP, 0.04–1.50 Hz) were calculated. 1.50 Hz was employed as the upper-bound for HF rather than the conventional limit of 0.40 Hz to account for the elevated respiration rate during exercise and early recovery. LF and HF were also normalized to total power (LF-nu and HF-nu). Frequency-domain measures were calculated over 2 min epochs using a smooth-priors detrending method.[17]

Impedance cardiography (Ambulatory Monitoring System, Version 3.9, Vrije University, Amsterdam, The Netherlands) was also continuously measured to determine STI. The primary STI outcome was the pre-ejection period (PEP), calculated using 30 s ensemble averages. Representing the delay between myocardial contraction and opening of the aortic valve, PEP strongly reflects cardiac contractility and has been demonstrated to be a probable inverse indicator of cSNA.[4] Additionally, left ventricular ejection time (LVET) and the PEP-LVET ratio (PEP:LVET) were calculated. Arterial systolic and diastolic blood pressures (SBP and DBP, respectively), were measured using an automatic sphygmomanometer (HEM-7203, Omron Healthcare, Kyoto, Japan) at rest and at 3, 5 and 10 min recovery. Mean arterial pressure (MAP) was calculated as 'DBP + $\frac{1}{3}$(SBP − DBP)'. The rate pressure product (RPP) was also calculated as 'HR × SBP × 10^{-3}'. Subjective effort (rate of perceived exertion, RPE) was also collected at end-exercise during each trial using the 10-point Borg category-ratio scale.[18]

All data were reported as means ± standard deviation (SD). If data were not normally distributed (Shapiro–Wilk test), a natural-logarithm (Ln) transformation was performed. All resting measures, as well as RPE and HRR_{60} were compared between trials using paired-samples t-tests. For HR, time-domain HRV and impedance-derived measures, a 2-way repeated measures analysis of variance (ANOVA) with the repeated factors of exercise modality (2 levels: LEG and ARM) and time (6 levels: end-exercise, 30 s, 1, 3, 5 and 10 min post-exercise) was conducted. Frequency-domain HRV was analyzed with 5 time levels (i.e. without 30 s recovery), while blood pressure data were analyzed with 3 time levels (3, 5 and 10 min). STI could not be reliably determined in 2 participants, therefore these were analyzed for the remaining 11 participants. If an interaction was found, paired-samples t-tests were performed to elucidate pair-wise differences between intensities at specific time-points. Additionally, as an interesting point of reference, a

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