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Activation of mechanoreflex delays heart rate recovery after exercise in healthy men

Article

Activation of mechanoreflex, but not central command, delays heart rate recovery after exercise in healthy men

Running title: Heart rate recovery mechanisms

ABSTRACT

This study tested the hypotheses that activation of central command and muscle mechanoreflex during post-exercise recovery delay fast-phase heart rate recovery with little influence on slow-phase. Twenty-five healthy men underwent three submaximal cycling bouts, each followed by a different 5-min recovery protocol: active (cycling generated by the own subject), passive (cycling generated by external force) and inactive (no-cycling). Heart rate recovery was assessed by the heart rate decay from peak exercise to 30s and 60s of recovery (HRR_{30s}, HRR_{60s} -fast-phase) and from 60s-to-300s of recovery ($HRR_{60\text{-}300s}$ -slow-phase). The effect of central command was examined by comparing active and passive recoveries (with and without central command activation) and the effect of mechanoreflex was assessed by comparing passive and inactive recoveries (with and without mechanoreflex activation). Heart rate recovery was similar between active and passive recoveries, regardless of the phase. Heart rate recovery was slower in the passive than inactive recovery in the fast- (HRR_{60s}=20±8vs.27±10bpm, $p<0.01$), but not in the slow-phase ($HRR_{60\text{-}300s}$=13±8vs.10±8bpm, $p=0.11$). In conclusion, activation of mechanoreflex, but not central command, during recovery delays fast phase heart rate recovery. These results elucidate important neural mechanisms behind heart rate recovery regulation.

Key words: exercise pressor reflex, baroreflex sensitivity, cardiovascular control, parasympathetic nervous system, heart rate variability

INTRODUCTION

Heart rate (HR) responses to exercise are regulated by central and peripheral neural mechanisms, including, but not limited to central command (i.e., descending signals from higher brain areas related to volition and effort sensation) and muscle mechanoreflex (i.e., a reflex arising predominantly from thinly-myelinated group III afferents in muscle fibers triggered by mechanical deformation of muscle fibers and/or joint movement) [1]. During voluntary exercise, inputs provided by such mechanisms are integrated in the medullary cardiovascular control centers, producing baroreflex resetting, sympathovagal activation, and increases in HR, thus providing appropriate cardiovascular responses to the metabolic demand of exercise [1,2].

Although the role of central command and mechanoreflex on HR responses during exercise have been widely explored [3-6], their roles in post-exercise HR recovery (HRR) are less well known. A reduced HRR after exercise is a marker of cardiac autonomic dysfunction and has been reported in different cardiovascular diseases [7], which highlights the importance of expanding the knowledge of the mechanisms underlying HRR. HRR presents a biphasic behavior, with an initial fast decay mainly determined by parasympathetic reactivation followed by a subsequent slow decay promoted by the combination of parasympathetic reactivation and sympathetic withdrawal [7,8]. Deactivations of central command and mechanoreflex at exercise cessation have been suggested to produce the stimuli for the parasympathetic reactivation immediately after exercise (i.e., 0 – 60 s), while the role of these mechanisms in the slow phase of HRR (i.e., 60 – 300 s) seems to be less important [7,9,10]. Accordingly, previous studies have shown that when central command and the mechanoreflex continue to be activated during recovery, such as active recovery, the fast-phase of HRR is slower than in conditions in which none of these mechanisms are active, such as inactive recovery [11,12]. However, the independent roles of central command and mechanoreflex on fast- and slow-phase HRR and its underlying autonomic regulation are yet to be comprehensively tested. Due to the important decrease in blood pressure (BP) that typically occurs immediately after exercise [13], the effects of central command and mechanoreflex on HRR may act via changes in baroreflex regulation, which has yet to be investigated.

In humans, it is possible to non-invasively verify the effects of central command on cardiovascular regulation by comparing voluntary and involuntary movement [5,11], whereas, the role of the mechanoreflex can be verified by comparing involuntary, e.g., passive, movement with no movement [11,12]. Thus, this study used these experimental protocols during the recovery from exercise to assess the role of central command and mechanoreflex activation during post-exercise recovery on HRR, baroreflex sensitivity and BP. To avoid any possible influence of pathological conditions or fluctuations due to menstrual cycle on HRR, healthy middle-aged men were investigated. The hypotheses were that both central command and mechanoreflex activation would independently delay the fast-phase of HRR but not affect the slow-phase of HRR.

MATERIAL & METHODS

Study design

This is a randomized crossover trial testing the effects of central command and mechanoreflex on HRR, in healthy middle-aged men. Data reported herein are derived from a larger trial that verified the effects of different neural regulatory mechanisms on HRR in healthy normotensive and hypertensive men [14,15].

Before taking part in the experimental sessions, participants performed an initial visit to the laboratory to check eligibility criteria and to perform a maximal cardiopulmonary exercise test. Following that, they attended the laboratory on three occasions for the experimental sessions.

Participants

Twenty-five healthy middle-aged men participated in this study. To participate, they needed to be between 30 and 60 years-old and to have normal BP levels (i.e., systolic/diastolic BP < 120/80 mmHg [16]). BP was defined from the average of six measurements performed in two separate visits as recommended in guidelines [16]. The exclusion criteria included smoking, presence of established cardiovascular or metabolic disease, body mass index equal to or greater than 35 kg/m^2, use of anti-hypertensive medication or other drugs that directly affects cardiovascular function, and abnormal resting or exercise ECG. Prior to participation, participants received detailed explanation about the experimental procedures and provided informed written consent. This study was conducted in accordance with the Declaration of Helsinki and was approved by the Ethics Joint Committee on Human Research of the School of Physical Education and Sport at the University of São Paulo (281.905/2013). The study also meets the ethical standards of the International Journal of Sports Medicine [17].

Exercise Test

On a preliminary visit to the laboratory, all participants underwent a maximal cardiopulmonary exercise test conducted on a cycle ergometer (Computrainer Pro 3D, RacerMate, Seattle, USA), in order to individualize the exercise intensity for the experimental sessions. The protocol started with an initial 3-min warm up at 50 watts followed by increments of 20 watts every 3 min until they were unable to keep pedaling at 60 rpm. During the test, ventilatory variables were continuously measured using a metabolic cart (CPX Ultima, Medical Graphics Corporation, Minnesota, USA), and peak oxygen consumption

(VO_{2peak}) and heart rate (HR_{peak}) were determined by the maximal values attained at the end of exercise (data analyzed in averages of 30 s).

Experimental Sessions

Participants underwent three experimental sessions in a temperature-controlled laboratory. Sessions were conducted in the morning (07:00 – 11:00), on three separate days and with intervals of at least 48 h between them. Participants were instructed to arrive in fasted state and to avoid caffeinated and alcoholic beverages for 24 h, as well as intense exercise for 48 h prior to each session. As food intake may influence autonomic function [18], food ingestion and time prior to the start of the session were standardized for all subjects and sessions. Thus, in each session, upon arrival to the laboratory, the participants received a standardized meal (two 25 g cereal bars and 50 ml of juice), and the experiments began 30 min afterwards.

In all sessions, the experiment started with a 10-min rest in the seated position (pre-exercise). Then, the participants performed 30 min of exercise on a cycle-ergometer (Tandem cycle + Computrainer Pro 3D, RacerMate, Seattle, USA) at 70% of VO_{2peak} (102 ± 12 Watts) and with a pedaling frequency of 60 rpm. Immediately after the exercise, they performed 5 min of recovery seated on the cycle ergometer. In each session, the recovery followed a different protocol (Figure 1): (a) inactive recovery, characterized by absence of movement (i.e., both central command and mechanoreflex were inactive); (b) active recovery, characterized by active loadless pedaling at 60 rpm (i.e., both central command and mechanoreflex were active); and (c) passive recovery, characterized by passive loadless pedaling at 60 rpm but with the driving force coming from another person seated on the second seat of the cycle (i.e., central command was inactive while mechanoreflex was active).

>>>>>>>>>>>>>>> FIGURE 1 <<<<<<<<<<<<<<<

Measurements

HR was measured using a 3-lead ECG (EMG System, São Paulo, Brazil) and beat-by-beat BP using finger photoplethysmography (Finometer, Finapres Medical System, Arnhem, Netherlands). These signals were continuously recorded online (Windaq, Dataq Instruments, Akron, Ohio, USA) with a sampling rate of 500 Hz per channel. To assess exercise intensity, VO_2 was continuously measured during the exercise by a metabolic cart (CPX Ultima, Medical Graphics Corporation, Minnesota, USA). To confirm similar thermal and metabolic stimuli between the sessions, core temperature (T_c) and blood

lactate concentration (BLC) were assessed. T_c was measured from intestinal temperature via a temperature pill system (CorTemp Wireless Ingestible Temperature Sensor, HQInc., Palmetto, USA) ingested, at least, 2 hours before the experiments [19]. BLC was measured from blood samples (25 µl) collected from the participants' earlobes at rest, in the last minute of exercise and immediately after the recovery period. Blood samples were centrifuged (5000 rpm for 5 min at 4°C) and plasma BLC was determined in duplicate using spectrophotometry (wavelength 546 nm, EON, Biotek instruments, USA).

Data Analysis

HR and beat-by-beat BP signals were exported to Heart Scope software (v. 1.3.0.1, A.M.P.S. LLC, New York, USA) for the generation of RR intervals (RRi) and beat-by-beat systolic BP (SBP) time series. These series were visually inspected, and occasional misdetections were manually corrected. Likewise, ectopic beats were identified and replaced with interpolated RRi values (less than 2% of the total signal). Pre-exercise and exercise HR and SBP were respectively calculated from averages of the last 5 min of the pre-exercise resting period and from 15 to 25 min of the exercise bout. Post-exercise HR and SBP were determined by the average of each successive 30 s during the entire 5 min of recovery. Additionally, SBP was expressed as the area under the curve for this entire period (post-exercise SBP_{AUC}) calculated by the trapezoid method [20].

Post-exercise RRi time series were transferred to Matlab software (Matlab 6.0, MathWorks, Massachusetts, USA) and HRR were assessed with a previously developed algorithm [14,21]. Fast-phase HRR indices were calculated from the absolute differences between peak exercise HR (mean of the last 60 s of exercise) and the HR obtained at 30 and 60s of recovery (HRR30s and HRR60s) [22]. The slow-phase HRR index was calculated from the absolute difference between the HRs obtained at 60s and 300s of recovery ($HRR_{60\text{-}300s}$) [23].

Spontaneous cardiac baroreflex sensitivity (cBRS) was assessed in the last 5 min of the pre-exercise resting period and during the entire 5 min of recovery using the sequence technique [14,24]. Briefly, the Heart Scope software (v. 1.3.0.1, A.M.P.S. LLC, New York, USA) identified sequences of three or more consecutive beats in which SBP and RRi changed in the same direction (at least 1 mmHg for SBP and 4 ms for RRi). In each sequence, the slope of the linear regression line between SBP and RRi was determined and the mean of all of the slopes from each timepoint was accepted as the mean cBRS (only sequences with $r^2 \geq 0.8$ were used) for that timepoint.

Statistics

Box plot was employed to verify outliers. The Shapiro-Wilk test was employed to verify data distribution. Homogeneity of variance was verified by the Levene test, and sphericity by the Mauchly test. One-way ANOVA was used to compare pre-exercise and exercise data between the three sessions.

As the aim of the study was to focus on the isolated role of each regulatory mechanism on HRR, the role of central command was assessed via comparisons of post-exercise data from active and passive recoveries (i.e., with and without central command activation, respectively), while the role of the mechanoreflex was assessed via comparisons of post-exercise data from passive and inactive recoveries (i.e., with and without mechanoreflex activation, respectively). These analyses were conducted using paired t-tests (for HRR indices) and two-way (session vs. time) repeated measures ANOVAs (for 30 s data). When a main effect or an interaction was significant, post-hoc comparisons were made using the Newman-Keuls test. For all analyses, values of $p \leq 0.05$ were considered significant. Data are present as mean ± SD.

RESULTS

Characteristics of participants are presented in Table 1. Participants were middle-aged, overweight, normotensive and with below-average fitness levels [25].

>>>>>>>>>>>>>>> TABLE 1 <<<<<<<<<<<<<<<

Experimental Session Results

Pre-exercise HR, SBP and cBRS were similar in the three sessions. There were also no differences between sessions for HR, SBP, VO_2, BLC and T_c during exercise and for BLC and T_c during the 3 different recovery modes (Table 2).

>>>>>>>>>>>>>>> TABLE 2 <<<<<<<<<<<<<<<

Effects of central command

The comparisons between the active and passive recoveries (i.e., role of central command) are shown in Figure 2. There was no difference in the HRR curve between the sessions (p=0.99 for time vs. session

interaction). All HRR indices. as well as post-exercise SBP_{AUC} and cBRS were not different between the active and passive sessions ($p = 0.14 – 0.77$).

>>>>>>>>>>>>>>>> FIGURE 2 <<<<<<<<<<<<<<<<

Effects of mechanoreflex

The comparisons between passive and inactive recoveries (i.e., role of mechanoreflex) are shown in Figure 3. HR showed a slower decrease throughout the recovery (i.e., from 30s to 300s) in the passive compared with the inactive session ($p<0.01$ for time vs. session interaction). Additionally, HRR_{30s} ($p<0.01$), HRR_{60s} ($p<0.01$) and cBRS ($p=0.03$) were lower, while post-exercise SBP_{AUC} ($p<0.01$) was higher in the passive than the inactive session. There was no difference in $HRR_{60\text{-}300s}$ between passive and inactive sessions ($p=0.11$).

>>>>>>>>>>>>>>>> FIGURE 3 <<<<<<<<<<<<<<<<

DISCUSSION

The main findings of the present study were that mechanoreflex activation delayed the fast-phase of HRR with no further effect on the slow-phase, while central command activation had no additional influence on HRR neither in its fast- or slow-phase.

The present study compared HRR between active, passive, and inactive recoveries. Previous studies have already employed these protocols to test central command and mechanoreflex influences either during [5,6] or after [11,12] exercise, and they are based on the assumption that central command is primarily activated by voluntary movement (e.g., active recovery), while mechanoreflex is activated by limb movement (e.g., both active and passive recoveries). These approaches have the advantages of being non-invasive and examining central command and mechanoreflex in physiological conditions. The suitability of the present study protocol has been shown by previous studies demonstrating absence of voluntary activation of the quadriceps during passive recovery [5,6]. In the present study, 3 subjects returned for an additional session in which their *vastus lateralis* electromyographic activity was assessed in the three experimental sessions, and it was also demonstrated absence of EMG activity in the passive and inactive recoveries (results not shown). It is also important to point out that metabolic and thermal impacts of the exercise/recovery were similar among the three sessions, as confirmed by similar BLC

and T_c. These aspects support that differences in HRR between the sessions and groups should not be attributed to other regulatory mechanisms such as metaboreflex [14] and/or thermoregulation [15,26].

Activation of the central command during recovery (i.e., active vs. passive recovery) did not promote additional influence on fast- or slow-phase HRR nor on SBP and cBRS. Therefore, these results do not support the role of central command in the autonomic regulation of HR after exercise. This finding diverges from previous mechanistic research investigating the effect of central command on HR. In fact, studies in animals or humans using electrical stimulation of locomotor areas in the midbrain [27,28] and studies with humans using partial neuromuscular block by tubocurarine [29,30] have all demonstrated a role of central command on HR. The difference between these experimental models and the one used in the present study may explain divergence between findings, as the use of brain electrical stimulation or neuromuscular blockage could overstimulate central command-related pathways [31]. The results of the present study also diverge from studies comparing active and passive movements in the HR response at the onset of exercise [6,32], which suggests that the role of central command may be restricted to the first instants of exercise, losing importance thereafter. Finally data herein reported is also different from Carter et al. [11], that observed a reduced HRR after active compared with passive recovery. Differences in the exercise protocols between studies might help to explain the different results, since Carter et al. [11] employed a 3-min moderate-intensity (i.e. 60%HR_{peak}) exercise bout, which might have elicited lower physiological stress than the present study. Indeed, there are evidence that higher exercise intensity and duration can greatly impact autonomic responses during exercise and HRR [10,33]. Therefore, the results of the present study originally demonstrate that central command activation does not significantly impact HRR after longer and more intense exercise.

In line with the study hypothesis, mechanoreflex activation delayed fast phase HRR with no remaining effect on slow phase HRR. These results suggest that mechanoreflex activation during recovery delays parasympathetic reactivation occurring immediately after exercise, but does not have a role in subsequent sympathetic withdrawal. Previous studies have already reported the relationship between mechanoreflex and parasympathetic regulation of HR using other stimuli such as passive limb manipulations in humans [3,4]. As for the post-exercise period, Shibasaki et al. [12] also observed increased HR during 10 min of passive recovery compared with inactive recovery. However, this study did not quantify the fast- and slow-phase HRR indices and, therefore, did not provide information on the effects of mechanoreflex on specific parasympathetic indices. There is less evidence on the effect of mechanoreflex on sympathetic regulation of HR in humans, with some studies relying on the spectral analysis of heart rate variability, which has been questioned as a marker of sympathetic modulation [34,35]. In the present study, the slow-phase HRR was employed as an index of cardiac sympathetic modulation. Although this is also an indirect measure, data from previous studies using pharmacological blockade give support to the sympathetic role of this measure [36]. Therefore, the results of the present

study suggest that, at least during immediate post-exercise recovery, mechanoreflex activation does not affect sympathetic regulation of HRR.

Due to the changes of BP after exercise, it was hypothesized that the effects of the mechanoreflex on HRR would be modulated by cBRS responses. Accordingly, SBP was higher in the passive than the inactive recovery, which should have resulted in a greater baroreflex-mediated decay of HR in the passive recovery (i.e., greater HRR) [37]. However, cBRS was reduced in passive recovery, which possibly prevented the baroreflex buffering of SBP. The effect of mechanoreflex activation decreasing cBRS is in agreement with previous studies [38] and suggests that, at least in part, mechanoreflex effects on HRR might involve its effects on cBRS.

From a physiological standpoint, the results of the present study bring new information on the roles of central command and mechanoreflex in autonomic regulation of post-exercise HRR, an indirect marker of autonomic dysfunction. The results of the present study also rise possibilities regarding the pathophysiology of reduced HRR observed in different diseases. For instance, patients with cardiovascular diseases (e.g., heart failure, hypertension) present both reduced HRR and increased mechanoreflex sensitivity [7,39]. As most of the HRR studies involving chronic disease populations employ active recovery protocols, it is likely that part of the slower HRR observed in these studies may be caused by increased mechanoreflex-mediated responses. Future studies should investigate the link between mechanoreflex sensitivity and HRR in these diseases and verify the effects of pharmacological and non-pharmacological therapies (e.g., exercise training) in the mechanoreflex-mediated HRR regulation.

Some limitations should be mentioned. First, this study used a convenience sampling of healthy, overweight and unfit middle-aged men and therefore the results cannot be extrapolated to other populations, such as women or elderly. Second, the present study results are restricted to moderate-intensity aerobic exercise and it is possible that different results could be obtained in high-intensity exercise conditions, characterized by a higher sympathetic activity [33]. Additionally, the assessments of central command and mechanoreflex influences were performed using non-invasive physiological maneuvers. It is possible, though, that different results could be obtained using supra-physiological stimulation (e.g., electrical stimulation) or pharmacological interventions (e.g., fentanyl, or partial curarization). However, the study opted to assess the role of such mechanisms using physiologically relevant stimuli, and for this reason, the results may represent the functioning of central command and mechanoreflex in typical physiological conditions.

In conclusion, mechanoreflex but not central command activation, influence fast-phase HRR in healthy middle-aged men. These results reinforce the role of mechanoreflex on parasympathetic control of HRR.

Conflict of Interest

The authors declare no conflict of interest.

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FIGURE LEGENDS

Figure 1 - Recovery protocols. a) inactive recovery, characterized by absence of movement; b) active recovery, characterized by active loadless pedaling; c) passive recovery, characterized by passive loadless pedaling with the driving force coming from another person seated at the second seat of the Tandem cycle.

Figure 2 - Heart rate recovery (HRR) curve (panel a), HRR indices (panels b-d), area under the curve of post-exercise systolic blood pressure (post-exercise SBP_{AUC}; panel e), and cardiac baroreflex sensitivity (cBRS; panel f) assessed during active and passive recovery sessions. HRR_{30s} = HRR after 30s; HRR_{60s} = HRR after 60s; $HRR_{60\text{-}300s}$ = HRR between 60s and 300s of recovery.

Figure 3 - Heart rate recovery (HRR) curve (panel a), HRR indices (panels b-d), area under the curve of post-exercise systolic blood pressure (post-exercise SBP_{AUC}; panel e), and cardiac baroreflex sensitivity (cBRS; panel f) assessed during passive and inactive recovery sessions. HRR_{30s} = HRR after 30s; HRR_{60s} = HRR after 60s; $HRR_{60\text{-}300s}$ = HRR between 60s and 300s of recovery. ‡ $p \leq 0.05$ vs. inactive.

393 **TABLE LEGENDS**

394 Table 1 – Sample characteristics (n=25). Values are presented as mean ± SD. BMI, body mass index.
395 SBP, systolic blood pressure. DBP, diastolic blood pressure. HR, heart rate, VO_{2peak}, peak oxygen
396 consumption during the exercise test. HR_{peak}, peak heart rate during the exercise test. PPO, peak power
397 output during the exercise test.

398

399 Table 2 – Physiological responses to the experimental sessions. Values are presented as mean ± SD.
400 HR, heart rate; SBP, systolic blood pressure; cBRS, cardiac baroreflex sensitivity; BLC, blood lactate
401 concentration; Tc, core temperature; VO2, oxygen uptake. † $p \leq 0.05$ vs. NT.

402