ORIGINAL ARTICLE

Heart rate recovery fast-to-slow phase transition: Influence of physical fitness and exercise intensity

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Background: Postexercise heart rate (HR) recovery presents an exponential decay, with two distinct phases: a fast phase, characterized by abrupt decay of HR, and determined by parasympathetic reactivation; and a slow phase, characterized by gradual decay of HR, and predominantly determined by sympathetic withdrawal. Although several methods have been proposed to assess postexercise HR recovery, none of those methods selectively assesses the time of transition from the fast to the slow phase of the HR recovery curve (HRR_{PT}), and the magnitude of decay prior to (HRR_{FP}) and after this point (HRR_{SP}). Therefore, the aim of the present study was to propose a method to identify HRR_{PT} , HRR_{FP} , and HRR_{SP} and to verify the effects of exercise intensity and physical fitness on such parameters.

Methods: Ten healthy young participants (24 ± 3 years; 23.6 ± 1.7 kg/m²) randomly underwent two exercise sessions (30 min of cycling), at moderate (MI) and high intensity (HI); followed by 5 min of inactive recovery. HR was continuously recorded during the sessions. The algorithm for HRR_{PT} analysis was written in Python and is freely available online.

Results: HRR_{PT} and HRR_{SP} were increased in HI session compared with MI (81 ± 24 vs. 60 ± 20 s; 8 ± 10 vs. 1 ± 5 bpm; $p = .04$), and there was no difference in HRR_{FP} between sessions (49 ± 15 vs. 46 ± 10 bpm; $p = .17$). In addition, HRR_{PT} for MI exercise session was significantly and negatively associated with VO_{2max} ($r = -0.85$, $p < .05$).

Conclusion: The method herein presented was sensitive to exercise intensity, and partially responsive to aerobic fitness. Next studies should perform the pharmacological and clinical validations of the method.

KEYWORDS

autonomic nervous system, heart rate variability

1 | INTRODUCTION

Postexercise heart rate (HR) recovery is a noninvasive method for the assessment of cardiac autonomic function recovery after a stressful stimulus (Peçanha, Silva-Junior, & Forjaz, 2014). HR recovery presents an exponential decay (Bartels-Ferreira, R., de Sousa, E. D., Trevizani, G. A., Silva, L. P., Nakamura, F. Y., & Forjaz, C. L. 2015) with two distinct phases: a fast phase, characterized by parasympathetic-mediated

abrupt decay of HR (Imai, K., Sato, H., Hori, M., Kusuoka, H., Ozaki, H., & Yokoyama, H. 1994), and a slow phase, characterized by more gradual HR reduction, determined by both parasympathetic reactivation and sympathetic withdrawal (Imai et al., 1994; Perini, R., Orizio, C., Comandè, A., Castellano, M., Beschi, M., & Veicsteinas, A. 1989).

Reduced HR recovery—caused by slow parasympathetic reactivation and/or sympathetic withdrawal (Peçanha, T., Silva-Junior, N. D., & Forjaz, C. L. 2014)—is a strong predictor of cardiovascular morbidity and

mortality, even in asymptomatic subjects (Cole, Foody, Blackstone, & Lauer, 2000). For this reason, in the last years several methods for HR recovery assessment have been proposed and tested (Bartels-Ferreira et al., 2015; Goldberger, Johnson, Subacius, Ng, & Greenland, 2014; Imai et al., 1994; Johnson & Goldberger, 2012; Perini et al., 1989; Pierpont, Stolpman, & Gornick, 2000). Most of these methods rely on simple arithmetic differences between peak HR and HR after 60 or 120 s in recovery (Cole, Blackstone, Pashkow, Snader, & Lauer, 1999; Cole et al., 2000; Mora et al., 2003; Vivekananthan, Blackstone, Pothier, & Lauer, 2003); other approaches include mathematical fitting of the initial or entire recovery curve (Bartels-Ferreira et al., 2015; Imai et al., 1994; Perini & Veicsteinas, 2003; Pierpont et al., 2000).

Despite the variety of methods for HR recovery assessment, as far as we know, none of those methods selectively assesses the time of transition from the fast to the slow phase of the HR recovery curve. This information is useful since this transition point ultimately quantifies the time required for HR to achieve most of its parasympathetic-mediated decay; and the HR decay prior to and after this point respectively quantify the parasympathetic- and sympathetic-mediated HR recovery (Peçanha et al., 2014). For this reason, the aim of this study was to propose a method for the identification of fast-to-slow phase transition time of HR recovery (HRR_{PT}) and to test the feasibility of this method by assessing the effects of exercise intensity and physical fitness on HRR_{PT} . Our hypothesis was that HRR_{PT} would be delayed after a high-intensity exercise (Pecanha et al., 2014) and would present an inverse relationship with physical fitness (Guerra et al., 2014).

2 | METHODS

2.1 | Participants

Ten healthy, sedentary nonsmokers and nonmedicated males participated in this study. Subjects were young (24 ± 3 years), eutrophic (body mass index = 23.6 ± 1.7 kg/m²), and moderately fit ($VO_{2peak} = 42.6 \pm 10.7$ ml.kg⁻¹.min⁻¹). After receiving written and verbal explanation of the protocol, participants signed the consent form. This study was approved by the University Human Ethics Review Board and followed the recommendations from the Declaration of Helsinki.

2.2 | Experimental protocol

The experimental protocol was conducted on 3 nonconsecutive days between 2 p.m. and 4 p.m. All subjects were instructed to avoid alcohol or caffeinated beverages, and also not to engage in any physical exercise in the 24 h preceding the tests.

In the first day, subjects answered the Physical Activity Readiness Questionnaire (PAR-Q) (Shephard, 1988), underwent assessments of body mass and height, and performed an incremental maximal exercise test, on an electromagnetically braked cycle ergometer (Ergo-Fit, Ergo Cycle 167, Pirmasens, Germany). Initial power was set to 50 W and increments of 25 W were applied every minute until participants

reached maximum voluntary exhaustion. Analysis of the expired gases was continuously performed with a metabolic analyzer (VO2000, MedGraphics, St. Paul, Minnesota, USA) properly calibrated before each test. Anaerobic threshold (AT) was identified from the loss of linearity of ventilatory equivalent for oxygen (VE/VO_2), and respiratory compensation point (RCP) was identified from loss of linearity of ventilatory equivalent for carbon dioxide (VE/VCO_2) (Wasserman, Whipp, Koyle, & Beaver, 1973). The workloads (in watts) of these physiologic transition points were registered for the prescription of subsequent sessions. Maximal workload, HR, and VO_2 attained during maximal exercise testing were defined as WL_{peak} , HR_{peak} , and VO_{2peak} , respectively.

In the second and third days, subjects randomly performed 30 min of cycle ergometer exercise, followed by 5 min of inactive recovery seated in the ergometer. In one of the sessions, subjects performed moderate-intensity exercise (MI; i.e., 30 min at the workload of the anaerobic threshold); while in the other session, exercise was performed at high intensity (HI; i.e., 15 min at anaerobic threshold workload, plus 15 min at respiratory compensation point workload). Beat-by-beat HR was continuously monitored throughout the session (Polar RS800CX, Kempele, Finland) (Nunan et al., 2009).

2.3 | Data processing and identification of heart rate recovery phase transition time

After experimental session, data were transferred to a personal computer and imported into Python for HRR_{PT} analysis (Python Software Foundation, Wilmington, Delaware, USA). The algorithm developed to detect HRR_{PT} was based on a similar approach proposed by Cheng et al. (1992) to determine both ventilatory and lactate thresholds, and by Kara et al. (1996) to identify the deflection point of the HR curve during a maximal effort test. The algorithm was written in Python and is freely available at <https://rhenanbartels.github.io/hrrpt/>. Firstly, to reduce the influence of HR fluctuations on HRR_{PT} detection, HR recovery series is filtered using the Savitzky-Golay filter (Savitzky & Golay, 1964). The algorithm then fits a main straight line using the first and last points of the series. After that, the Euclidean distance between each HR point and the main line is calculated. The distance corresponds to the length of a perpendicular segment from a HR point to the main line. This procedure is repeated until HR recovery series is completed. HRR_{PT} is considered the point presenting the largest distance (D_{MAX}) between HR values and the main line. Besides HRR_{PT} , the algorithm also returns the fast phase HR recovery (HRR_{FP}), which is calculated by the difference between the median of the first five HR points at the beginning of recovery (HR_i) and the median of five HR points around the HRR_{PT} (HR_{PT}); and the slow phase HR recovery (HRR_{SP}), which is calculated by the difference between HR_{PT} and the median of the last five HR points at the end of recovery (HR_o) (Figure 1).

2.4 | Statistical analysis

Normality was tested using the Lilliefors test. Descriptive data were compared between sessions using Student *t* test, and main outcomes were compared using the Wilcoxon signed-rank test.

FIGURE 1 Identification of heart rate (HR) recovery phase transition time (HRR_{PT}) in one representative participant. (a) Presents the postexercise HR recovery curve. The light gray line represents the actual signal, the black line represents the filtered signal. HRR_{PT} is the point in seconds with the largest perpendicular distance (D_{MAX}) between HR recovery and the main line (i.e., dashed line). HRR_{FP} quantifies the fast phase of HR recovery. HRR_{SP} quantifies the HR decay in the slow phase of recovery. (b) Presents the variations in the perpendicular distance from all HR recovery points to the main line. The first and the last points coincide with the main line and the D_{MAX} determines the HRR_{PT}

Correlations between the HRR_{PT} , HRR_{FP} , HRR_{SP} , and VO_{2peak} in MI and HI sessions, were calculated by the Spearman test. Significance level was set to 0.05. All statistical analysis was conducted using

R (A Language and Environment for Statistical Computing, Vienna, Austria). Values are presented in mean $\pm$ standard deviation, or median (interquartile range).

3 | RESULTS

HR attained during MI and HI sessions were 129 ± 22 and 165 ± 20 bpm ($p < .01$), corresponding 67 ± 10 and $85 \pm 10\%$ of

HR_{peak} ($p < .01$), respectively. Figure 2 presents the postexercise HR recovery curves of one representative participant. HRR_{PT} was delayed after HI compared with MI session ($HRR_{PT} = 46.6$ and 38.6 s, respectively).

FIGURE 2 Heart rate recovery curve of one representative participant after MI (a) and HI (b) sessions. The vertical arrow indicates the HRR_{PT} in each curve

FIGURE 3 Comparisons of HRR_{PT} , HRR_{FP} , and HRR_{SP} between moderate-intensity (MI) and high-intensity (HI) exercise sessions. * = $p < .01$ vs. MI. Data from 1 outlier were excluded of the final analysis

Figure 3 shows the median values and interquartile range of HRR_{PT} (Figure 3a), HRR_{FP} (Figure 3b) and HRR_{SP} (Figure 3c) in MI and HI exercise sessions. HRR_{PT} and HRR_{SP} were increased in HI compared with MI ($p = .04$ for both comparisons), and there was no difference in HRR_{FP} between sessions ($p = .17$). Regarding the secondary parameters returned from the algorithm, HR_i (158 ± 19 vs. 128 ± 22 bpm.; $p = .02$), HR_{pt} (108 ± 18 vs. 81 ± 19 bpm.; $p < .01$), and HR_o (100 ± 15 vs. 80 ± 18 bpm.; $p = .01$) were higher in HI when compared with MI.

HRR_{PT} in MI exercise session was significantly and negatively correlated with VO_{2max} ($r = -0.85$, $p < .05$; Figure 4). There were no significant associations between the other parameters and VO_{2peak} ($r = -0.20$ – 0.40 ; $p > .05$).

4 | DISCUSSION

The present study was the first to propose a method to identify the transition of the fast-to-slow phase of HR recovery (i.e., HRR_{PT}). Furthermore, we tested the feasibility of the method by: (1) assessing HRR_{PT} during recovery after HI and MI cycling exercise, which has been previously shown to produce different cardiac autonomic recovery (Pecanha et al., 2014), and (2) testing the correlations between HRR_{PT} , HRR_{FP} , HRR_{SP} , and physical fitness assessed by VO_{2peak} . HRR_{PT} was sensitive to exercise intensity, since HI produced greater

HRR_{PT} than MI, and partially responsive to aerobic fitness, once the expected correlations between HRR_{PT} and fitness occurred only for MI exercise.

Postexercise recovery represents crucial moment for the cardiac autonomic regulation. Immediate postexercise recovery is characterized by fast decrease in HR promoted by a prompt reactivation of parasympathetic activity, followed by slow decrease in HR evoked by gradual sympathetic withdrawal. This biphasic behavior of HR recovery after exercise is caused by complex integrative interaction of the mechanisms controlling the cardiovascular system, and modifications in such profile indicate cardiac autonomic dysfunction and may underline cardiovascular abnormalities (Imai et al., 1994). Accordingly, slow HR recovery after exercise has been observed in several cardiovascular diseases (Pecanha et al., 2014) and is independently associated with increased cardiovascular (Arena et al., 2010) and overall (Cole et al., 1999) mortality risks. For these reasons, the assessment of HR recovery provides pivotal information for detecting cardiac autonomic abnormalities and for predicting mortality risks.

Several methods have been proposed for the assessment of HR recovery (Pecanha et al., 2017). Most of these methods are based on simple subtractions of HR at a designed time in recovery from the peak HR. Despite these methods being predictive of poor cardiovascular outcomes and mortality, their physiological limitations rely on the fact that they generally do not take into account which phase of HR recovery is being covered. In the present study, we proposed a method for identification of fast to slow phase of HR recovery transition, otherwise known as HRR_{PT} ; and assessed the magnitude of HR recovery prior to and after this transition (i.e., HRR_{FP} and HRR_{SP}). In a theoretical basis, it is expected that HRR_{PT} and HRR_{FP} measure respectively, the time required for most of parasympathetic-mediated HR decay, and the magnitude of the parasympathetic reactivation; and HRR_{SP} predominantly measures the magnitude of sympathetic withdrawal (Pecanha et al., 2017).

Although the present study did not perform selective autonomic blockade to verify the specific roles of the parasympathetic and sympathetic nervous systems subdivisions on HRR_{PT} , HRR_{FP} , and HRR_{SP} , comparisons of such parameters between different intensity exercises allow making conjectures. In this sense, it has been demonstrated that high-intensity exercises delay both parasympathetic reactivation and sympathetic withdrawal (Imai et al., 1994; Perini, Orizio, Baselli, Cerutti, & Veicsteinas, 1990). In this sense, it was expected that HRR_{PT}

FIGURE 4 Correlation between HRR_{PT} and physical fitness (VO_{2max}). Data from 1 outlier were excluded of the final analysis

would be delayed (i.e., increased), and HRR_{FP} and HRR_{SP} would be blunted after HI compared with MI session. This hypothesis was partially confirmed, since HRR_{PT} was higher in HI than MI, confirming a slower cardiac autonomic recovery after a high-intensity exercise. On the other hand, there was no difference in HRR_{FP} between HI and MI, and HRR_{SP} was increased in HI compared with MI, which diverge from the study's hypothesis.

Comparisons of HR recovery between different exercise intensities are laborious. HR at the start of the recovery is greater at higher intensities (Peçanha et al., 2014), as it can be seen by the greater values of HR_i in the HI session. This greater HR_i ultimately increases the range of HR decay until the end of recovery in HI session. Supporting this hypothesis, when HRR_{FP} and HRR_{SP} are normalized by the total decay of HR recovery from the start to the end of recovery, the reported difference in HRR_{SP} between MI and HI is no longer evident, and there is even a slight trend ($p = .10$) for greater HRR_{FP} after MI than HI. The normalized analysis also reveals that HRR_{FP} accounts for almost all of the HR decay seen in MI session ($\approx 98\%$), remaining only a minor portion of the decay for HRR_{SP} ($\approx 2\%$). On the other hand, slightly different contributions are observed in HI session (91% and 9%). For these reasons, percentage values of HRR_{FP} and HRR_{SP} might provide complementary information when comparing different exercise intensities.

The present study proposed a method for identification of the transition phase of HR recovery. The aims of this preliminary study were to verify the performance of the algorithm using real exercise data, and to perform a "physiological validation" of the algorithm by verifying the influences of exercise intensity and aerobic fitness on the primary parameters, namely HRR_{PT} , HRR_{FP} , and HRR_{SP} . In this sense, the results herein presented are consistent with the expected responses to these physiological parameters, which support further investigation of the proposed method. The main limitation of this study was the absence of pharmacological validation of the autonomic mechanisms behind the calculated parameters. Next studies should test the effects of selective autonomic blockade of sympathetic and parasympathetic subdivisions of the autonomic nervous system on HRR_{PT} , HRR_{FP} , and HRR_{SP} . Data on clinical populations would also help to verify the sensitivity and specificity of the method in abnormal conditions.

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CONFLICT OF INTEREST

The authors declare that they have no conflicts of interest.

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