Acute and Chronic Effects of Exercise

Post-exercise cardiac autonomic recovery is associated with resting chronotropic response in different body positions in men

Giliard Lago Garcia[1,2,3], Carlos Janssen Gomes da Cruz[1,2], Edgard Melo Keene von Koenig Soares[1,2], Luiz Guilherme Grossi Porto[1,2], Guilherme Eckhardt Molina[1,2]

[1]*Universidade de Brasília, Faculdade de Educação Física, Laboratório de Fisiologia do Exercício, Brasília, DF, Brazil;* [2]*Universidade de Brasília, Grupo de Estudos em Fisiologia e Epidemiologia do Exercício e da Atividade Física, Brasília, DF, Brazil;* [3]*Centro Universitário Instituto de Educação Superior de Brasília, Brasília, DF, Brazil.*

Associate Editor: Katia de Angelis, Universidade Federal de São Paulo, São Paulo, SP, Brazil. E-mail: prof.kangelis@yahoo.com.br.

Abstract - Aim: This study evaluated the association between heart rate recovery (HRR) and cardiac parasympathetic reactivation following a submaximal exercise test with resting heart rate (RHR) in the supine and orthostatic positions. **Methods:** The heart rate (HR), HRR, relative HRR (Δ% HRR), and cardiac parasympathetic reactivation (r-MSSD) values during passive orthostatic recovery at the 1st, 3^{rd,} and 5th min following the submaximal exercise test were correlated with RHR in both the supine (RHR_{sup}) and orthostatic (RHR_{ort}) positions in 24 physically active men. The statistical analysis employed Pearson and Spearman correlation tests with a two-tailed p-value set at ≤ 0.05. **Results:** At the 1st min, HR was significantly correlated with RHR_{ort} ($r = 0.40$; $p = 0.05$). HR, HRR, and Δ% HRR at the 3rd and 5th min were significantly correlated with RHR (r = from -0.67 to 0.73; $p \leq 0.02$; R^2 = 32-53%), independent of the body position at rest. At the 3rd and 5th min, the r-MSSD was significantly correlated with RHR_{sup} ($r = -0.60, -0.46$; $p \leq 0.02$). At the 3rd min, the r-MSSD was significantly correlated with RHR_{ort} ($r = -0.46$; $p = 0.02$). **Conclusion:** Faster HRR and parasympathetic reactivation on the 3rd and 5th minute after the submaximal exercise test were associated with low RHR in the supine and orthostatic positions in active young men.

Keywords: cardiac autonomic function, heart rate recovery, resting heart rate, submaximal exercise test, heart rate variability.

Introduction

Heart rate (HR) recording is often used in clinical and experimental studies, which indirectly infer the influences of sympathetic and parasympathetic balance on the sinus node[1]. The monitoring of the resting heart rate (RHR) is a low-cost, noninvasive measure, and there is recording feasibility across a wide range of settings, with the technique offering a way to individually screen for risk factors with unfavorable prognoses, such as increased cardiovascular morbimortality and sudden death[2,3].

Based on this, RHR is frequently assessed in distinct postures (supine, seated, or standing) because body position influences cardiac autonomic drive in humans[4,5]. This analysis demonstrates the shift in cardiac autonomic balance, which may provide valuable information about cardiac autonomic adjustments during rest[4,6].

After an incremental exercise test, heart rate recovery (HRR) has been proposed in several studies as an important prognostic and mortality index in individuals with distinct clinical and functional conditions[7-10]. Following exercise testing during the first minute (the fast phase), the short-term HR adjustment responds to rapid parasympathetic reactivation; from the third minute onwards (the slow phase), it is more dependent on parasympathetic reactivation with simultaneous sympathetic deactivation[11,12]. In addition, most studies on HRR have focused on recovery in the first minute after exercise, and few have examined both early and late HRR. Additionally, different implications of exercise physiology can be appreciated with a comprehensive analysis of HRR beyond the first minute (5 min), which can indicate a crucial determinant of heart rate decline.

In this scenario, when considering that slow HRR following incremental exercise test is a marker of impaired cardiac parasympathetic reactivation[7-9], as well as the fact that an increased RHR is a marker of reduced parasympathetic activity[2,3,13], it is reasonable to expect that these chronotropic adjustments over the post-exercise recovery phase and at rest may be interrelated.

Some studies have shown that faster HRR immediately after maximal treadmill exercise testing was positively associated with parasympathetic reactivity (reduction) after a postural change from supine to standing positions[14,15]. Some studies have focused only on the association between resting parasympathetic activity in supine or seated positions and HRR after submaximal exercise tests in the same positions[16,17]. Other studies have examined this relationship by considering parasympathetic activity, as evaluated by heart rate variability (HRV), and analyzing it only before exercise in the supine position, as well as the use of HRR in the standing position after the maximal treadmill test[18].

Conversely, other studies have not shown an association between parasympathetic activity at rest and HRR, or between parasympathetic activity after maximal exercise tests[19-22]. Therefore, excluding analysis of resting parasympathetic activity in the standing position may lead to conflicting results because of discrepancies in evaluating the relationship between HRR and resting parasympathetic activity across positions. Additionally, autonomic activity is different in the supine and standing positions, and the exercise test is performed in the standing position, with HRR being measured in this condition.

Therefore, it is essential to evaluate the relationship between both functional phenomena when considering the different resting autonomic statuses in supine and standing positions related to the submaximal exercise protocol. To the best of our knowledge, no studies have shown information on the relationship between HRR and parasympathetic activity after submaximal exercise testing and RHR in different postures. Thus, this analysis may provide helpful information as a preliminary decision-making tool for health care professionals, disseminating essential and complementary information on individuals' cardiac autonomic capacities without the expense of clinical exercise tests or the maximal/near-maximal effort required for recovery analysis.

Thus, this study explored the relationship between heart rate recovery and parasympathetic reactivation after a submaximal exercise test, with resting heart rate measured in supine and standing postures, in young active men.

Methods

Participants

We conducted an analytical cross-sectional study enrolling 24 young and physically active males aged 26.5 (23.7-30.0) years with a body mass index of 23.9 (23.3-28.5) kg/m^2. Participants were eligible for inclusion if they were men, physically active (≥ 150 min of moderate-vigorous physical activity per week, according to the International Physical Activity Questionnaire [IPAQ])[23], nonathletes, healthy (no medical restrictions or known disease), and aged between 20 and 40 years old. Volunteers underwent exercise testing at 2h after breakfast (between 8:00 and 10:00 a.m.) and were previously instructed to abstain from stimulants and alcoholic beverages and physical activity for at least 24h before the evaluation. This study involving human participants was reviewed and approved by the Ethical Committee on Human Research of the University Center Euro-Americano (UNIEURO, approval number: 006/2011) in agreement with the Declaration of Helsinki. The participants provided written informed consent to participate in this study, and each participant signed a written informed consent form.

Study design

Initially, we collected basic clinical physiological data (RHR, blood pressure, and respiratory rate), anthropometrical measurements, and information on lifestyle habits. Consequently, in a quiet exercise physiology laboratory room at an ambient temperature between 22-24 °C and a relative humidity of 50-60%, continuous HR was recorded according to a previously described standardized protocol to obtain the R-R interval series[14,15,24]. In summary, a valid five-minute RR series of HR was first obtained following 10 min of rest in the supine position. Afterward, the participants were asked to actively adopt the orthostatic posture at the bedside. Two minutes after this postural change, the blood pressure was measured to verify the absence of significant postural hypotension, and an additional five minutes of RR series of HR was recorded. Blood pressure was measured by using the auscultatory method[25] via a sphygmomanometer and stethoscope (Premium®, Brazil).

The submaximal treadmill exercise test was immediately applied after the two 5-minute RR series of HR (in the supine and orthostatic postures), and data were recorded. Soon after the interruption of the exercise test, the participants proceeded to the post-exercise passive orthostatic position for a cool-down stage[24].

Heart rate and heart rate variability analysis

The HR and R-R interval series were recorded by using a valid and reliable heart rate monitor known as Polar® (model RS800CX, Polar ™, Kempele, Finland) with a sample rate of 1,000 Hz[26,27]. Subsequently, each R-R interval series file was transferred to a computer for offline data processing and analysis of HR and the variability of the R-R interval by utilizing the Polar Pro Trainer 5 software and the Kubios HRV software (version 2.2, Kuopio, Finland), respectively[28].

Regarding the R-R interval series analysis, all the R-R segments were visually analyzed, and occasional artifacts were automatically removed (< 1% of recordings)[29]. The automated artifact identification and removal were performed by using the threshold method, which consists of selecting R-R intervals that were larger or smaller than 0.45 s (very low), 0.35 s (low), 0.25 s (medium), 0.15 s (strong), or 0.05 s (very strong) compared to average R-R intervals[28]. We used the medium threshold that only removed the visually observed ectopic points, if the tracing did not lose the physiological pattern, and the removal did not exceed 1% of the recording.

The HR measurements in the supine (RHR_{sup}) and orthostatic (RHR_{ort}) positions were recorded as previously described under resting conditions. During the submaximal treadmill exercise test, HR recording was immediately initiated before the start of the exercise test ($HR_{initial}$) and was stopped when participants reached 85% of their maximum HR (HR_{peak}), as predicted by Tanaka et al.[30]. During passive recovery, HR was recorded at the 1st, 3rd and 5th minute over the recovery phase, and the absolute and relative values of HRR were calculated by subtracting HR at the 1st, 3rd and 5th minute over the recovery phase from HR_{peak}[7].

The time-domain indices that we measured were (a) the mean of the R-R interval (RRi) series (which is an index of the global cardiac autonomic activity) and (b) the square root of the mean of the square of successive adjacent R-R interval differences (r-MSSD), which reflects the parasympathetic activity associated with respiratory sinus arrhythmia[31].

Post-exercise autonomic recovery was assessed by using two different methods. We recorded the short-term HRV measurements (i.e., five min) and the ultrashort-term HRV measurements that were analyzed in each segment of one minute throughout the passive recovery phase (i.e., the 1st, 3^{rd,} and 5th minutes). These quantitative analysis methods are based on the notion of different temporal effects of changes in parasympathetic modulation on the heart (without a requirement for the stationarity of the data)[31].

Treadmill submaximal exercise testing

All the participants performed submaximal exercise testing on a conventional treadmill (Centurion - Micromed, Brazil). The use of submaximal exercise testing was based on the risk level to the volunteers and the availability of appropriate equipment and personnel and was used to determine the HR and HRV responses to one determined submaximal work rate[32].

The exercise protocol started at a speed of 3.0 km/h and a grade of 2.5%; the grade remained constant throughout the test. After two minutes at this initial speed, the speed was increased by 1.0 km/h every minute until participants reached HR_{peak}. After achieving this submaximal intensity (85% of their maximum HR), the exercise test was interrupted, and a 5-minute passive recovery period was immediately initiated with volunteers in the standing position, as previously described[24,33]. We adopted this protocol to avoid the potential influence of movement on parasympathetic reactivation[24].

Statistical analysis

The statistical analysis employed Prism® 8 for Windows software (GraphPad Software, Inc., USA, 2019) and IBM SPSS Statistics 23 (SPSS Software, Inc., USA, 2015). The observed power (OP) was calculated by using post hoc power analyses for each correlated variable via G*Power 3.1.9.7 for Windows software[34].

The normality of the distribution of the variables was verified by using the Shapiro-Wilk test and by using a visual Q-Q plot analysis, and scores greater than 1.5 times the interquartile range outside of the boxplot were considered to be outliers[35,36]. We verified linearity via a scatter plot and homoscedasticity via plots of standardized residuals against predicted values. We used the median and quartiles (25% and 75%) as descriptive statistics.

The paired t-test was used to compare HR at rest, during exercise, and during recovery. The correlation analysis was performed by using the Pearson correlation test when compliance with normality, linearity, homoscedasticity, and the absence of outlier assumptions was verified. The Spearman correlation test was conducted when normality assumptions were not met. We adopted the following criteria for the interpretation of the correlation coefficient: < 0.1: none; $\geq 0.1 < 0.3$: poor; $\geq 0.3 < 0.6$: fair; $\geq 0.6 < 0.8$: moderate; $\geq 0.8 \leq 0.9$: very strong; and = 1.0: perfect [37]. The two-tailed level of statistical significance was set at $p \leq 0.05$. A Pearson's coefficient 95% confidence interval was calculated, whereas the Spearman's coefficient was estimated by using an approximation due to a sample size > 10[38].

Results

Table 1 shows the chronotropic response at rest, exercise, and the 1st, 3^{rd,} and 5th minute of the recovery period after submaximal exercise testing, and the arterial blood pressure at rest. The baseline physiological variables and chronotropic responses during and after exercise were within the normal range in all the subjects.

Table 2 and Figure 1 describe the correlation coefficients between HRR and HRV with RHR_{sup} following submaximal exercise testing. At the 1st minute, all the HRR and HRV values throughout the post-exercise period exhibited no correlations with RHR_{sup}. However, HR values at the 3rd and 5th min were positively correlated (fair and moderate) with RHR_{sup} ($r = 0.60$ and $r = 0.57$, $p \leq 0.002$), which explained 32% to 36% of the variance in HR at these time points during recovery. We observed a negative/moderate correlation between HRR, Δ% HRR, and RHR_{sup} at the 3rd and 5th min of recovery (rs = -0.62 -

Table 1 - Sample values of arterial blood pressure at HR at rest, during exercise test (initial and peak), and first, third, and fifth minutes of the recovery period after submaximal exercise test (n = 24).

	Rest period				Exercise period		Passive recovery period		
	Supine HR	**Orthostatic HR**	**Supine BP**	**Orthostatic BP**	**Initial HR**	**Peak HR**	**1st min HR**	**3rd min HR**	**5th min HR**
Median	62	80^{a*}	118/72	116/78	84	165^{b*}	126	102^{c*}	$99^{d*; e*}$
Quartiles	57-66	76-88	113/69-120/78	110/75-118/80	78-89	163-166	122-132	93-109	91-104
Extremes	43-73	57-94	102/58-132/82	110/62-129/90	63-100	152-169	103-143	80-117	77-111

Heart rate (HR) in beats/min; Blood pressure (BP) in millimeters of mercury; $^*p \leq 0.01$; a: RHR_{sup} vs. RHR_{ort}; b: $HR_{initial}$ vs. HR_{peak}; c: $HR_{1^{st}min}$ vs. $HR_{3^{rd}min}$; d: $HR_{1^{st}min}$ vs. $HR_{5^{th}min}$; e: $HR_{3^{rd}min}$ vs. $HR_{5^{th}min}$.

Table 2 - Correlation between resting heart rate in supine position and cardiac autonomic function after a submaximal exercise test (n = 24).

Correlated variables	Post-exercise passive recovery period											
	1st min				3rd min				5th min			
	CC	**R^2**	**p**	**OP**	**CC**	**R^2**	**p**	**OP**	**CC**	**R^2**	**p**	**OP**
HR (bpm)	r = 0.34	0.11	0.09	39%	r = 0.60	0.36	0.002^*	93%	r = 0.57	0.32	0.003^*	90%
HRR (bpm)	r = - 0.33	0.10	0.10	37%	r = - 0.67	0.44	0.0001^*	98%	r = - 0.62	0.38	0.001^*	95%
Δ%HRR (%)	r = - 0.34	0.11	0.10	39%	r = - 0.65	0.42	0.0001^*	97%	r = - 0.62	0.38	0.001^*	95%
mean RRi (ms)	r_s = - 0.20	#	0.34	15%	r_s = - 0.59	#	0.002^*	92%	r_s = - 0.49	#	0.01^*	74%
r-MSSD (ms)	r_s = - 0.19	#	0.35	14%	r_s = - 0.60	#	0.002^*	93%	r_s = - 0.46	#	0.02^*	67%

RHR: resting heart rate; bpm: beats per minute; sup: supine position; HR: heart rate; HRR: heart rate recovery in absolute decrement; Δ%HRR: heart rate recovery in relative decrement; min: minute; RRi: R-R interval; ms: milliseconds; CC: correlation coefficient; OP: observed power; r: Pearson's r; r_s: Spearman's rho; $^*p \leq 0.05$.

-0.67, $p \leq 0.001$), in which RHR_{sup} explained 38% to 44% of the HRR and Δ% HRR variance at these time points during the postexercise period. A negative correlation (fair to moderate) between HRV markers (mean RRi and r-MSSD) and RHR_{sup} was observed at the 3rd and 5th min of recovery (r_s = -0.46 - -0.60, $p \leq 0.02$).

Table 3 and Figure 1 describe the correlation coefficients between HRR and HRV following submaximal exercise testing with RHR_{ort}. We observed a fair/moderate positive correlation between HR and RHR_{ort} from the 1st to 5th min of postexercise recovery (r = 0.40, r = 0.58, and r = 0.73, $p \leq 0.05$), with RHR_{ort} explaining 16% to 53% of the variance in HR at this time point during recovery. The results showed a fair/moderate negative correlation between HRR and Δ% HRR with RHR_{ort} at the 3rd and 5th min postexercise (r = -0.57 to - -0.73, $p \leq 0.004$). Moreover, RHR_{ort} explained 32% to 53% of the HRR and Δ% HRR variance at these time points during recovery. Regarding the correlation between r-MSSD and $RHR_{ort,}$ we observed a fair negative correlation at the 3rd minute postexercise (rs = -0.46, p = 0.02). Additionally, we observed a fair/moderate negative correlation between the mean RRi and RHR_{ort} from the 1st to 5th min of post-exercise recovery (r_s = -0.49 to - -0.63, $p \leq 0.01$). Furthermore, no correlation was found between HRR, Δ% HRR, and r-MSSD with RHR_{ort} at the 1st minute and r-MSSD at the 5th minute postexercise recovery.

Discussion

Our study observed new and relevant findings in the relationship between the dynamics of HR and parasympathetic activity after submaximal exercise testing with RHR in different postural positions (supine and orthostatic) in active young men.

We observed that active young men with lower RHR (independent of body position) showed faster HRR and higher parasympathetic reactivation in the 3rd and 5th min of recovery. Therefore, we observed that active young men with lower RHR (independent of body position) showed faster HRR and higher parasympathetic reactivation in the 3rd and 5th min of recovery.

Although the evaluation of HR and parasympathetic activity at rest and during the recovery phase after exercise is practical, straightforward, and feasible, a comprehensive understanding of the relationship between these measures could provide important clinical and functional implications for research and exercise evaluation[11,39]. In other words, clinical studies have shown that high RHR is associated with reduced parasympathetic activity[2,3], and delayed heart rate recovery (HRR) is associated with impaired parasympathetic reactivation[10]. Additionally, this scenario is associated with unfavorable prognoses, such as overtraining syndrome[39].

When considering the present outcomes, the RHR analysis in different postural positions (supine and

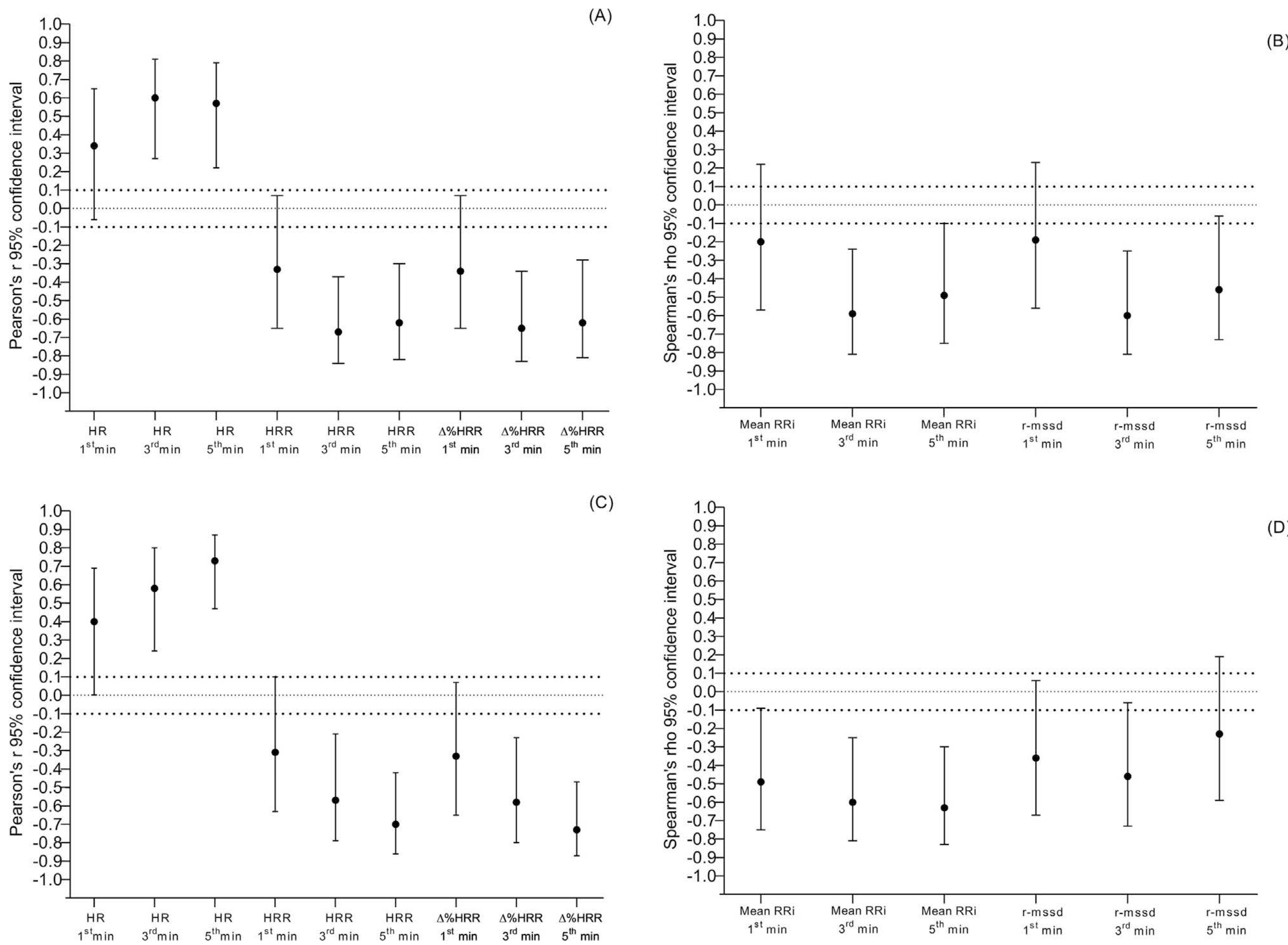

Figure 1 - Correlation Coefficient (r and rs) and 95% confidence interval between resting heart rate in supine position (panel A and B) and orthostatic position (panel C and D) with cardiac autonomic function after a submaximal exercise test (n = 24).

Table 3 - Correlation between resting heart rate in orthostatic position and cardiac autonomic function after a submaximal exercise test (n = 24).

Correlated variables	**Post-exercise passive recovery period**											
	1st min				**3rd min**				**5th min**			
	CC	**R^2**	**p**	**OP**	**CC**	**R^2**	**p**	**OP**	**CC**	**R^2**	**p**	**OP**
HR (bpm)	r = 0.40	0.16	0.05	53%	r = 0.58	0.33	0.002*	91%	r = 0.73	0.53	0.0001*	99%
HRR (bpm)	r = - 0.31	0.09	0.13	33%	r = - 0.57	0.32	0.0004*	90%	r = - 0.70	0.49	0.0001*	99%
Δ%HRR (%)	r = - 0.33	0.10	0.10	37%	r = - 0.58	0.33	0.003*	91%	r = - 0.73	0.53	0.0001*	99%
mean RRi (ms)	r_s = - 0.49	#	0.01	74%	r_s = - 0.60	#	0.002*	93%	r_s = - 0.63	#	0.01*	96%
r-MSSD (ms)	r_s = - 0.36	#	0.08	43%	r_s = - 0.46	#	0.02*	67%	r_s = - 0.23	#	0.26	19%

RHR: resting heart rate; bpm: beats per minute; ort: orthostatic position; HR: heart rate; HRR: heart rate recovery in absolute decrement; Δ%HRR: heart rate recovery in relative decrement; min: minute; RRi: R-R interval; ms: milliseconds; CC: correlation coefficient; OP: observed power; r: Pearson's r; r_s: Spearman's rho; *$p \leq 0.05$.

standing positions) may be used as a preliminary tool for decision-making, considering its potentially helpful information related to the "flexibility" of everyone's cardiac autonomic capacity without the expense of clinical exercise tests that require effort to infer the parasympathetic activity sympathovagal balance response in the heart. In this manner, our data preclude a causal relationship, but our analysis demonstrated the direction and magnitude of the relationship between these variables. Specifically, young men who presented with faster HRR after a submaximal exercise test showed lower RHR independent of body position, and RHR explained between 16% and 53% of the variance in the HRR after exercise.

Our findings can aggregate knowledge on the challenging (and controversial) understanding of the interactions between cardiac autonomic control at rest and during post-exercise recovery following the exercise test. In this context, some studies have addressed the relationship between HRR and parasympathetic activity (as evaluated by HRV) at rest and have observed no significant correlations between these measures[19-22].

Of note, studies with similar exercise protocols have addressed the correlation between markers of parasympathetic activity at rest with the HRR and HRV during the recovery period after submaximal exercise testing[16,17]. For example, Evrengul et al.[17] observed a correlation between HRV measures at rest and HRR in the 3rd minute during passive recovery (in the supine position). Moreover, Danieli et al.[16] observed a correlation between markers of cardiovagal activity at rest and HRR at 30s and at the 1st and 2nd min during passive recovery (in the seated position) in the same direction. In addition, Javorka et al.[21], who are pioneers in the field, observed no correlations between measures of HRV at rest with measures of HRV and the Δ% HRR in the 1st minute of recovery after submaximal exercise. However, they observed a significant correlation from the 5th minute of recovery onwards. The present study results partially agree with the literature cited above, in which it was shown that there is a correlation between the RHR (independent of the body position) and cardiac parasympathetic reactivation at the 3rd and 5th min of orthostatic passive recovery.

These discordant results may be due to (a) inappropriate statistical analyses in some studies via uses of a parametric correlation test with no normalization of the variables, instead of the use of a nonparametric test (because HRV indices are notoriously nonnormally distributed)[15]; (b) a lack of sample size calculation[40]; (c) distinctive age, gender, physical fitness and functional or clinical conditions of the evaluated participants[40]; (d) a nonuniformity of the exercise protocols that were employed[14]; (e) the fact that measurements of HRV and HR during the recovery period were made in different postural positions; (f) the use of different recovery phase protocols and (g) the usage of different min during the recovery period.

Some limitations of this study included the sample size, which may have restricted the analyses. However, the significant correlation at the 3rd and 5th min, with a power greater than 80% mitigated the risk of a type I error. Additionally, the characteristics of the results cannot be extrapolated to women, athletes, and older adults. Although our results cannot be precisely extrapolated to men in general, we chose to prioritize the study's internal validity to overcome some of the common heterogeneity that may explain the controversy in the field. Consequently, we selected a sample that was solely of young, physically active, and healthy men in a narrow range of ages. Therefore, even though this homogeneity between individuals may represent a limitation, it is also one of the strengths of our study because it reinforces our findings due to its strong internal validity.

As highlighted, our results are based on submaximal treadmill exercise testing, which has been less commonly used in research protocols but has important practical implications due to its improved safety. Nevertheless, our findings cannot be extrapolated to other workload exercises or protocols, such as cycle or arm ergometry, in which individuals are seated during rest. Additionally, the participants performed the passive recovery phase in the orthostatic position. Thus, the results cannot be extrapolated to other recovery protocols, such as passive recovery in the supine position or active cool-down stages.

Conclusion

In conclusion, our data support the hypothesis that lower RHR (independent of body position [supine and standing position]) demonstrated faster HRR (cardiac parasympathetic reactivation) in the 3rd and 5th min after submaximal exercise, and RHR explained between 16% and 53% of the variance in the HRR after the submaximal exercise test in young men.

Acknowledgments

EMKVKS is currently receiving a doctoral study scholarship from Coordenação de Aperfeiçoamento de Pessoal de Nível Superior - Brasil (CAPES) - Finance Code 001. This work was also supported by Decanato de Pesquisa e Pós-Graduação - UnB (DPG-UnB) under Grant 07/2021.

References

1. Lahiri MK, Kannankeril PJ, Goldberger JJ. Assessment of autonomic function in cardiovascular disease: physiological basis and prognostic implications. J Am Coll Cardiol. 2008;51(18):1725-33.
2. Park D-H, Jeon JY. The prognostic value of resting heart rate for health status. Exer Scie. 2020;29(1):24-33.
3. Zhang DF, Wang WJ, Li F. Association between resting heart rate and coronary artery disease, stroke, sudden death, and noncardiovascular diseases: a meta-analysis. Canadian Medical Association Journal. 2016;188(15):E384-E92.
4. Carnethon MR, Liao D, Evans GW, Cascio WE, Chambless LE, Heiss G. Correlates of the shift in heart rate variability with an active postural change in a healthy population sample: the atherosclerosis risk in communities study. Am Heart J. 2002;143(5):808-13.
5. Carnethon MR, Liao D, Evans GW, Cascio WE, Chambless LE, Rosamond WD, et al. Does the cardiac autonomic response to postural change predict incident coronary heart disease and mortality? The atherosclerosis risk in communities study. Am J Epidemio. 2002;155(1):48-56.

6. Hottenrott L, Ketelhut S, Hottenrott K. Commentary: vagal tank theory: the three Rs of cardiac vagal control functioning-resting, reactivity, and recovery. Front Neurosci. 2019;13:1300.
7. Cole CR, Blackstone EH, Pashkow FJ, Snader CE, Lauer MS. Heart-rate recovery immediately after exercise as a predictor of mortality. N Engl J Med. 1999;341(18):1351-7.
8. Cole CR, Foody JM, Blackstone EH, Lauer MS. Heart rate recovery after submaximal exercise testing as a predictor of mortality in a cardiovascularly healthy cohort. Ann Intern Med. 2000;132(7):552-5.
9. Jouven X, Empana J-P, Schwartz PJ, Desnos M, Courbon D, Ducimetière P. Heart-rate profile during exercise as a predictor of sudden death. N Engl J Med. 2005;352(19):1951-8.
10. Qiu S, Cai X, Sun Z, Li L, Zuegel M, Steinacker JM, et al. Heart rate recovery and risk of cardiovascular events and all-cause mortality: a meta-analysis of prospective Cohort studies. J Am Heart Assoc. 2017;6(5):e005505.
11. Michael S, Graham KS, Davis GM. Cardiac autonomic responses during exercise and post-exercise recovery using heart rate variability and systolic time intervals - a review. Front Physiol. 2017;8:301.
12. Peçanha T, Bartels R, Brito LC, Paula-Ribeiro M, Oliveira RS, Goldberger JJ. Methods of assessment of the post-exercise cardiac autonomic recovery: a methodological review. International Journal of Cardiology. 2017;227:795-802.
13. Romero-Ortuno R, O'Connell MD, Finucane C, Fan CW, Kenny RA. Higher orthostatic heart rate predicts mortality: The Irish Longitudinal Study on Aging (TILDA). Aging Clin Exp Res. 2015;27(2):239-42.
14. Molina GE, da Cruz CJG, Fontana KE, Soares E, Porto LGG, Junqueira Jr. LF. Post-exercise heart rate recovery and its speed are associated with cardiac autonomic responsiveness following orthostatic stress test in men. Scand Cardiovasc J. 2021;55(4):220-6.
15. Molina GE, Fontana KE, Porto LG, Junqueira Jr. LF. Post-exercise heart-rate recovery correlates to resting heart-rate variability in healthy men. Clin Auton Res. 2016;26 (6):415-21.
16. Danieli A, Lusa L, Potocnik N, Meglic B, Grad A, Bajrovic FF. Resting heart rate variability and heart rate recovery after submaximal exercise. Clin Auton Res. 2014;24(2):53-61.
17. Evrengul H, Tanriverdi H, Kose S, Amasyali B, Kilic A, Celik T, et al. The relationship between heart rate recovery and heart rate variability in coronary artery disease. Ann Noninvas Electro. 2006;11(2):154-62.
18. Esco MR, Olson MS, Williford HN, Blessing DL, Shannon D, Grandjean P. The relationship between resting heart rate variability and heart rate recovery. Clin Auton Res. 2010;20(1):33-8.
19. Bosquet L, Gamelin F-X, Berthoin S. Is aerobic endurance a determinant of cardiac autonomic regulation? Eur J Appl Physiol. 2007;100(3):363-9.
20. Dewland TA, Androne AS, Lee FA, Lampert RJ, Katz SD. Effect of acetylcholinesterase inhibition with pyridostigmine on cardiac parasympathetic function in sedentary adults and trained athletes. Am J Physiol Heart Circ Physiol. 2007;293(1):H86-92.
21. Javorka M, Zila I, Balharek T, Javorka K. Heart rate recovery after exercise: relations to heart rate variability and complexity. Braz J Med Biol Res. 2002;35(8):991-1000.
22. Lee CM, Mendoza A. Dissociation of heart rate variability and heart rate recovery in well-trained athletes. Eur J Appl Physiol. 2012;112(7):2757-66.
23. Matsudo S, Araújo T, Marsudo V, Andrade D, Andrade E, Braggion G. Questinário internacional de atividade fisica (IPAQ): estudo de validade e reprodutibilidade no Brasil. Rev Bras Ativ Fís Saúde. 2001(6):05-18.
24. Garcia GL, Porto LGG, Fontana KE, Gomes CJ, Junqueira LF, Molina GE. Efeito de diferentes protocolos de recuperação sobre a função autonômica cardíaca. Rev Bras Med Esporte. 2017;23:16-20.
25. Barroso WKS, Rodrigues CIS, Bortolotto LA, Mota-Gomes MA, Brandão AA, Feitosa ADM, et al. Diretrizes Brasileiras de Hipertensão Arterial-2020. Arquivos Brasileiros de Cardiologia. 2021;116:516-658.
26. Hernando D, Garatachea N, Almeida R, Casajus JA, Bailon R. Validation of heart rate monitor polar RS800 for heart rate variability analysis during exercise. J Strength Cond Res. 2018;32(3):716-25.
27. Wallen MB, Hasson D, Theorell T, Canlon B, Osika W. Possibilities and limitations of the Polar RS800 in measuring heart rate variability at rest. Eur J Appl Physiol. 2012;112 (3):1153-65.
28. Tarvainen MP, Niskanen J-P, Lipponen JA, Ranta-Aho PO, Karjalainen PA. Kubios HRV-heart rate variability analysis software. Comput Methods Programs Biomed. 2014;113 (1):210-20.
29. Tulppo MP, Makikallio TH, Takala TE, Seppanen T, Huikuri HV. Quantitative beat-to-beat analysis of heart rate dynamics during exercise. Am J Physiol. 1996;271(1 Pt 2): H244-52.
30. Tanaka H, Monahan KD, Seals DR. Age-predicted maximal heart rate revisited. J Am Coll Cardiol. 2001;37(1):153-6.
31. Camm AJ, Malik M, Bigger JT, Breithardt G, Cerutti S, Cohen R, et al. Task Force of the European Society of Cardiology the North American Society of Pacing. Heart rate variability: standards of measurement, physiological interpretation, and clinical use. Circulation, 1996;93(5):1043-65.
32. Riebe, D. senior editor. ACSM's guidelines for exercise testing and prescription. Philadelphia; Lippincott Williams & Wilkins, 2018.
33. Cruz CJGd, Rolim PS, Pires DS, Mendes CMO, Paula GM, Porto LGG, et al. Reliability of heart rate variability threshold and parasympathetic reactivation after a submaximal exercise test. Mot Rev de Educ Fis. 2017;23:65-70.
34. Faul F, Erdfelder E, Lang AG, Buchner A. G*Power 3: a flexible statistical power analysis program for the social, behavioral, and biomedical sciences. Behav Res Methods. 2007;39(2):175-91.
35. Ghasemi A, Zahediasl S. Normality tests for statistical analysis: a guide for non-statisticians. Int J Endocrinol Metab. 2012;10(2):486-9.

36. Razali NM, Wah YB. Power comparisons of shapiro-wilk, kolmogorov-smirnov, lilliefors, and anderson-darling tests. JOSMA. 2011;2(1):21-33.
37. Akoglu H. User's guide to correlation coefficients. Turk J Emerg Med. 2018;18(3):91-3.
38. Zar, JH. Biostatistical analysis. London; Pearson Education Limited, 2014.
39. Bosquet L, Merkari S, Arvisais D, Aubert AE. Is heart rate a convenient tool to monitor over-reaching? A systematic review of the literature. British Journal of Sports Medicine. 2008;42(9):709-14.
40. Laborde S, Mosley E, Thayer JF. Heart rate variability and cardiac vagal tone in psychophysiological research-recommendations for experiment planning, data analysis, and data reporting. Frontiers in Psychology. 2017;8:213.

Corresponding author

Giliard Lago Garcia. Centro Universitário Instituto de Educação Superior de Brasília, Brasília, DF, Brazil.
E-mail: giliardlgarcia@gmail.com

Manuscript received on June 17, 2022
Manuscript accepted on March 21, 2023