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ORIGINAL ARTICLE

Cardiac autonomic modulation post-maximal incremental exercise is not influenced by body mass index in young adult men

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Abstract

Background The purpose of this study was to analyze whether cardiac autonomic modulation recovery at short-term after maximal incremental exercise test in young adults is influenced by the body mass index (BMI).

Methods Forty-eight healthy untrained men (22.26 ± 3.08 years old) were evaluated; 18 were normal weight (NW: 21.89 ± 1.75 kg m^{-2}), 11 were overweight (Ov: 27.00 ± 1.38 kg m^{-2}) and 19 were obese (*O*: 34.38 ± 3.36 kg m^{-2}). After the maximal incremental exercise test, a 10 min recovery (5 min actively, followed by 5 min passively) was performed to analyze cardiac autonomic modulation recovery. The root mean square successive differences between adjacent RR intervals (RMSSD) and standard deviation of all normal RR intervals (SDNN) indexes were determined in successive non-overlapped 30 s segments transformed in natural logarithmic—Ln.

Results LnRMSSD and LnSDNN indexes were reduced at the end of the incremental exercise test and remained reduced throughout 5 min of active recovery, and throughout 5 min after this time compared to baseline resting ($p < 0.05$), with no differences ($p > 0.05$) among the groups. However, throughout 5 min (330–600 s) after active recovery, the LnSDNN values were higher than the end of exercise (NW: 0.56 ± 0.19; Ov: 0.56 ± 0.25; *O*: 0.57 ± 0.16 ms; $p < 0.05$).

Conclusions Although there was no vagal reactivation at short-term after a maximal incremental exercise test, there were alterations in the overall heart rate variability regardless of BMI in apparently healthy young adult men.

Keywords Obesity · Recovery · Autonomic control · Heart rate variability · Parasympathetic activity

Abbreviations

BMI	Body mass index
HRV	Heart rate variability
NW	Normal weight
O	Obese
Ov	Overweight
RMSSD	Root mean square successive difference between adjacent RR intervals
SDNN	Standard deviation of all normal RR intervals
VE	Ventilation
VO_2	Oxygen consumption
VCO_2	Carbon dioxide production

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Introduction

Regular exercise promotes protective action in cardiovascular and cardiac autonomic system. However, it is configured as an acute stressor stimulus that causes physiological changes, and greater exposure to cardiovascular events post-exercise [1].

The adjustments of the cardiac autonomic nervous system in post-exercise recovery are largely analyzed by heart rate measures [2–6], and its lower reduction is associated with a worse prognosis of all-cause cardiovascular mortality [4, 7]. However, heart rate variability (HRV) analysis, such as the root mean square successive differences between adjacent RR intervals (RMSSD, reflects parasympathetic activity of autonomic nervous system), and standard deviation of all normal RR intervals (SDNN, reflects overall autonomic nervous system activity) have been suggested as a validated method and a good marker of cardiac autonomic modulation changes [8]. In this way, the monitoring these variables simultaneously can provide insights not shown by heart rate or either variable alone.

Some studies [9, 10] have shown vagal reactivation in the initial 5 min post-maximal exercise recovery, different from another study [11]. Thus, the autonomic cardiac mechanisms responsible for the restoration of cardiac autonomic modulation post-exercise maximal are not well understood.

Most of the studies about the recovery of post-exercise cardiac autonomic control were performed in older individuals [7, 12] with different diseases [6, 13, 14] aiming at the stratification of mortality risks [4].

It has been well recognized that excess body mass is related to several physiological changes at rest, such as elevation of blood pressure [2], heart rate and sympathetic nervous system activity and parasympathetic nervous activity reduction [15]. In addition, excess body mass causes impairment in several post-exercise parameters, such as a delay in heart rate recovery in people with hypertension [6, 14], metabolic syndrome [16], and in healthy young adults [2]. However, few studies have evaluated the influence of excess body mass on heart rate reestablishment [3] and, especially, post-exercise HRV indexes [17] in healthy young adult men. In addition, the effect of different physical exercise intensities [18–21] and modalities [9] and also, type of physical training [10] in HRV has been assessed in physically active individuals.

Therefore, this study aimed to analyze whether the cardiac autonomic modulation in post-maximal exercise recovery in young adults is influenced by body mass index (BMI). We assumed that there is no vagal reactivation in the initial minutes of post-maximal exercise and that young adult men with excess body mass show impaired autonomic cardiac modulation recovery.

Methods

Participants

Forty-eight young men between 18 and 30 years old, apparently healthy and considered untrained for having not practiced any kind of physical exercise for three months prior to the beginning of the study were evaluated. They were divided into three groups according to BMI: 18 normal weight (NW: 20–24.9 kg m^{-2}), 11 overweight (Ov: 25–29.9 kg m^{-2}) and 19 obese (*O*: 30–39.9 kg m^{-2}). Exclusion criteria were BMI < 20 or ≥ 40 kg m^{-2}, cardiovascular, metabolic or orthopedic diseases, smoking, and currently taking any prescribed medications (e.g., anti-hypertensive or anti-depressive agents).

The study procedures were approved by the Institutional Ethics Committee in Human Research (no 19109213.2.0000.5541). All participants were informed about the study procedures and signed a free informed consent form.

Experimental protocol

One visit to the laboratory was performed for rest, maximal incremental exercise test and post-maximal exercise recovery evaluations. The study procedures were carried out between 8:00 a. m. and 12:00 p.m., with an average temperature of 24 °C and relative air humidity of 42%. The participants were instructed not to engage in vigorous physical activity and not to drink stimulant and alcoholic beverages 24 h prior to the evaluations. In addition, a standardized snack (360.7 kcal), with 73.19% carbohydrates (66 g), 20.71% lipids (8.3 g) and 6.10% proteins (5.5 g) was consumed 2 h before the evaluations.

Initially, abdominal circumference (CARDIOMED®, Brazil) was measured, so as, the percentage of body fat was determined using tetrapolar bioimpedance (MALTRON, BF 907, Australia). After 15 min of sitting at rest, a maximal incremental exercise test was performed in a cycle ergometer (INBRASPORT, CG-04, Brazil), with an initial workload of 15 W and 15 W increments per min, keeping 60 rpm until volitional exhaustion [22]. The rate of perceived exertion was monitored in the final 10 s of each stage, using 15-points Borg's scale [23].

Following the period of maximal incremental test, participants performed 5 min recovery period with 15 W at 60 rpm, and subsequently another 5 min with participants seated on the cycle ergometer.

Procedures

The participants adopted a sitting position at rest for 15 min to measure blood pressure (automatic sphygmomanometer, MICROLIFE, BP 3BT0-A, Brazil). Two measurements, with a 2 min interval between each measurement were performed. When a difference of 5 mmHg was found between the measurements, a third one was performed, and the mean between the two measurements with the closest values was calculated.

During the incremental exercise test, oxygen consumption was measured by direct uptake of exhaled gases (i.e., ventilation, oxygen consumption, and carbon dioxide production) by a portable gas analyzer (VO2000 MEDICAL GRAPHICS, USA). This device had been calibrated using ambient air according to the manufacturer's recommendations. The data were recorded every 10 s and the highest value obtained in the final of the incremental test was considered as $VO_{2\ peak}$.

The incremental exercise test was considered maximal when the participants reached the following criteria [24]: inability to maintain 60 rpm for more than 10 s, at least

90% of maximal heart rate predicted (220—age; NW: 94.63 ± 3.29; Ov: 96.09 ± 5.00 and *O*: 97.19 ± 3.69%—$p = 0.145$), values on the Borg's scale > 17 points (NW: 19.22 ± 1.63; Ov: 19.18 ± 1.17 and *O*: 19.79 ± 0.42—$p = 0.252$) and respiratory exchange ratio > 1.1 (NW: 1.25 ± 0.14; Ov: 1.17 ± 0.09 and *O*: 1.18 ± 0.06 – $p = 0.270$).

Data analysis

The RR intervals were records using a portable heart rate monitor (POLAR, RS800CX, Finland) during the entire final 10 min at rest. The RR intervals were treated with the Kubios HRV® software, with the artifacts filtered at a moderate level. The percentage of error observed was less than 2% in all records. The data were acquired with a noninvasive heart-rate monitor that has shown a good agreement with data obtained by electrocardiography [25]. HRV indexes at rest were performed using a fixed number of 256 consecutive RR intervals.

HRV indexes were determined from time-domain methods, with parasympathetic nerve activity being assessed by root mean square of successive differences of RR intervals (RMSSD) and the overall autonomic nervous system activity by standard deviations of all normal intervals (SDNN) [26]. These HRV indexes showed moderate-to-high reproducibility post-maximal exercise [27].

Heart rate was measured in the final 5 s of each 60 s in the entire recovery time, and the HRV indexes were determined in successive non-overlapped 30 s segments [8] at the end of exercise ($RMSSD_{end}$ and $SDNN_{end}$) and during entire the 10 min post-exercise period ($RMSSD_{30s}$; $RMSSD_{60s}$ … $RMSSD_{600s}$ and $SDNN_{30s}$, $SDNN_{60s}$ … $SDNN_{600s}$).

In addition, the areas under the curve of the RMSSD and SDNN indexes from the end of maximal incremental exercise test and post-exercise recovery were obtained by the equation: Σ [(Area 1 + Area 2 + Area 3…)], each area being: [(Base major + Base minor) × height]/2. For the height was considered the value of 30 s. Twenty areas were obtained which together determined the value of the total area.

Statistical analysis

A natural logarithm transformation (LnRMSSD and LnSDNN) was performed on the HRV indexes to allow parametric analysis. The Shapiro–Wilk and Levene tests were used to verify the normality and homogeneity of the data, respectively. One-Way ANOVA with Tukey post-hoc for parametric data and Kruskal–Wallis test with Dunn's post-hoc for non-parametric data were used to compare the variables at rest and during maximal incremental exercise test among groups. The behavior of heart rate recovery in segments of 60 s and HRV indexes in segments of 30 s during the 10 min of post-exercise recovery were evaluated using two-way (group vs. time) repeated measures ANOVA, with verification of the data regarding breach of sphericity by the Mauchly test. The Greenhouse-Geisser correction was used when sphericity was violated. When a major effect or interaction was significant, Bonferroni post-hoc test was conducted. The correlation between variables was tested by Pearson's linear correlation. The level of significance was $p < 0.05$.

Results

The values of body mass, BMI and body fat (%) were higher for the overweight and obese groups than for the normal weight group, with significant differences among all groups ($p < 0.05$) (see Table 1). The systolic and diastolic blood pressure values were higher in the obese group compared to the normal weight group, with no differences compared to the overweight group. However, no participant had elevated blood pressure (> 140/90 mmHg).

The absolute VO_{2peak} values were higher in the overweight and obese groups compared to the normal weight group, while the VO_{2peak} values adjusted to total body mass were higher in the normal weight and overweight groups compared to the obese group. On the other hand, the heart rate, LnRMSSD and LnSDNN indexes at rest were not different ($p > 0.05$) among the groups (see Table 1).

There were no differences for the heart rate behavior in the recovery among the groups (group: $F = 0.381$, $p = 0.685$, group vs. time: $F = 1.068$, $p = 0.388$). Regardless of group, heart rate decreased (time: $F = 1014.472$, $p < 0.001$) during the entire 10 min recovery compared to the end of the exercise, but did not reach resting levels (see Fig. 1).

Figure 2 shows the LnRMSSD (a) and LnSDNN (b) indexes at rest, end of maximal incremental test and post-exercise recovery analyzed in 30 s segments, with no differences among groups at any time point in both HRV indexes LnRMSSD (group: $F = 1.304$, $p = 0.281$; time: $F = 94.730$, $p < 0.001$, group vs. time: $F = 0.923$, $p = 0.503$) and LnSDNN (group: $F = 0.054$, $p = 0.947$; time: $F = 85.234$, $p < 0.001$, group vs. time: $F = 1.000$, $p = 0.463$).

The LnRMSSD and LnSDNN indexes at the end of the maximal incremental test (NW: 0.52 ± 0.09; Ov: 0.53 ± 0.16; *O*: 0.57 ± 0.12/NW: 0.56 ± 0.19; Ov: 0.56 ± 0.25; *O*: 0.57 ± 0.16 ms) were suppressed below resting baseline (NW: 1.51 ± 0.22; Ov: 1.47 ± 0.29; *O*: 1.50 ± 0.21/NW: 1.79 ± 0.19; Ov: 1.69 ± 0.21; *O*: 1.74 ± 0.18 ms) and remained reduced throughout 5 min of active recovery (NW: 0.56 ± 0.15; Ov: 0.49 ± 0.13; *O*: 0.49 ± 0.20 / NW: 0.69 ± 0.22; Ov: 0.86 ± 0.20; *O*: 0.79 ± 0.29 ms) and throughout 5 min after this time (NW: 0.64 ± 0.26, Ov: 0.51 ± 0.18, *O*: 0.59 ± 0.28 / NW: 0.98 ± 0.23, Ov: 0.94 ± 0.17; *O*: 0.91 ± 0.24 ms), with no differences ($p > 0.05$) among the

Table 1 Anthropometric, body composition, hemodynamic, autonomic variables on the rest and maximal incremental exercise test

	Normal weight ($n=18$)	Overweight ($n=11$)	Obese ($n=19$)	(p value)
Age (years)	22.78 ± 3.11	21.46 ± 2.87	22.24 ± 3.21	0.542
Body mass (kg)	67.48 ± 6.04[a]	83.41 ± 11.24[b]	105.78 ± 13.71[c]	< 0.001
Body mass index (kg m^{-2})	21.89 ± 1.75[a]	27.00 ± 1.38[b]	34.38 ± 3.36[c]	< 0.001
Abdominal circumference (cm)	78.44 ± 6.66[a]	93.09 ± 7.54[b]	109.21 ± 10.36[c]	< 0.001
Body fat (%)	17.04 ± 4.47[a]	27.22 ± 3.01[b]	35.94 ± 4.52[c]	< 0.001
Systolic blood pressure (mmHg)	111.03 ± 8.75[a]	114.95 ± 3.95[ab]	119.66 ± 10.00[b]	0.013
Diastolic blood pressure (mmHg)	68.81 ± 5.86[a]	73.41 ± 6.15[ab]	74.18 ± 7.65[b]	0.046
Heart $rate_{rest}$ (bpm)	76.57 ± 8.89	78.09 ± 10.24	76.68 ± 8.83	0.897
$LnRMSSD_{rest}$ (ms)	3.48 ± 0.52	3.38 ± 0.67	3.46 ± 0.47	0.891
$LnSDNN_{rest}$ (ms)	4.11 ± 0.44	3.90 ± 0.48	4.01 ± 0.41	0.437
Peak workload (W)	191.88 ± 36.44	224.68 ± 76.13	200.76 ± 27.85	0.180
VO_{2peak} (mL min^{-1})	2434.31 ± 284.32[a]	2946.75 ± 592.75[b]	2959.00 ± 485.94[b]	0.011
VO_{2peak} (mL kg^{-1} min^{-1})	36.24 ± 4.69[a]	35.05 ± 4.69[a]	28.19 ± 3.77[b]	< 0.001
VO_{2peak}/LBM (mL kg^{-1} min^{-1})	43.57 ± 5.16	48.02 ± 7.95	44.15 ± 4.89	0.256

LBM lean body mass

Different letters indicate significant ($p < 0.05$) difference among groups (a ≠ b ≠ c) by One-way ANOVA and Tukey post-hoc

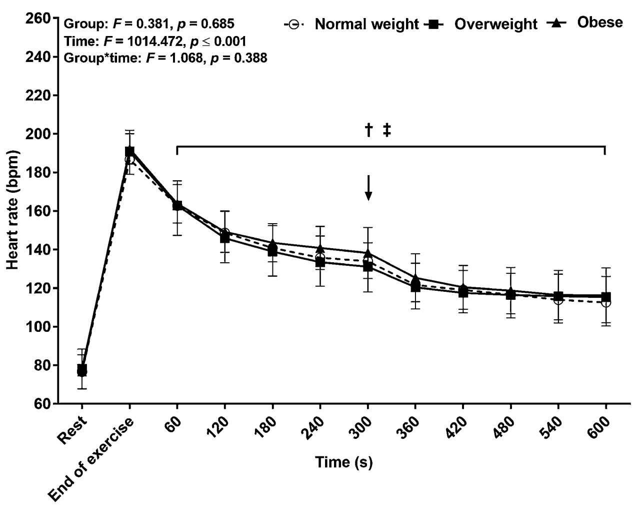

Fig. 1 Heart rate at rest, end of maximal incremental exercise test and 60 s segments post-maximal exercise test. The arrow indicates the end of active recovery. †$p < 0.05$ compared to the rest for all groups, ‡$p < 0.05$ compared to the end of maximal incremental exercise test for all groups. Two-way (group vs. time) repeated measures ANOVA with Bonferroni post-hoc test

groups. However, the LnSDNN index values increased during the throughout 5 min after active recovery (330–600 s) from the end of maximal incremental exercise test ($p < 0.05$) in all groups, with no differences among them (see Fig. 2).

In addition, there were no difference among groups in the areas under the curve of LnRMSSD (NW: 348.01 ± 101.93; Ov: 297.61 ± 70.56; *O*: 309.96 ± 86.20 ms s^{-1}—$p = 0.272$) and LnSDNN indexes (NW: 506.66 ± 93.46; Ov: 503.46 ± 70.63; *O*: 499.33 ± 77.46 ms s^{-1}— $p = 0.964$). The area under the curve of the LnRMSSD (r: −0.353; $p = 0.014$) and the LnSDNN indexes (r: −0.354, $p = 0.014$) correlated negatively with resting heart rate.

Discussion

The main results of this study showed that although there was no vagal reactivation at short-term after a maximal incremental exercise test, there were alterations in the overall HRV regardless of BMI in healthy young adult men.

The impairment absence in cardiac autonomic modulation at recovery post-exercise in obese people suggest that there may be a threshold of total body fat amount (BMI > 35 kg m^{-2}) or visceral fat necessary to trigger changes in cardiac autonomic modulation at rest and/or recovery after physical exercise. In the current study, 68.4% of the individuals with obesity were degree 1 and obesity grade 3 was an exclusion criterion.

It has been reported that the absolute VO_{2peak} is higher and the VO_{2peak} adjusted to body mass is lower in the obese group, compared to the normal weight group, but similar when adjusted for lean body mass [28, 29]. This is in agreement with our results.

To the best of our knowledge, this is one of the first studies to evaluate the obesity influence on cardiac autonomic modulation reestablishment in short-term post-maximal exercise test in the healthy young adults. Heart rate recovery is often evaluated in older individuals [7, 12], with cardiovascular risk factors [6, 15] or metabolic syndrome [16], chronic diseases [13, 30, 31], and for analysis of mortality risk [4], with HRV indexes analysis in a few of these studies

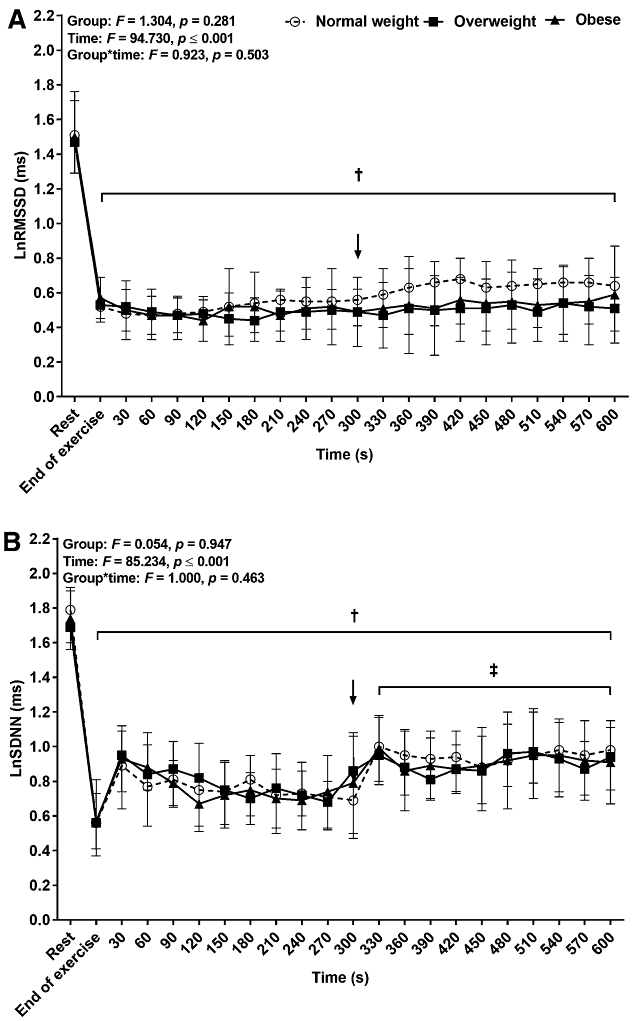

Fig. 2 LnRMSSD (**a**) and LnSDNN (**b**) indexes at rest and $LnRMSSD_{30}$ (**a**) and $LnSDNN_{30}$ (**b**) at the end maximal incremental exercise test and post-exercise recovery in 30 s segments. The arrow indicates the end of active recovery. $^{\dagger}p<0.05$ compared to the rest for all groups, $^{\ddagger}p<0.05$ compared to the end of maximal incremental exercise test for all groups. Two-way (group vs. time) repeated measures ANOVA with Bonferroni post-hoc test

[6, 12]. On the other hand, when young people are evaluated, they are physically active [9, 11, 21] or trained men [10], with few studies in untrained youth men [19]. This factor is very important, one time that most people are untrained physically.

The non-reactivation of the parasympathetic nervous system due the continuous suppression of the LnRMSSD index in the recovery period compared to baseline resting and at the end of maximal incremental exercise test is similar to the previous findings after 5 min of passive recovery of a maximal exercise in healthy sedentary or strength-trained young [10] and physically active men [11]. In addition, these measures did not return to baseline by 10 min following low-, moderate- or high-intensity exercise [21].

On the other hand, the vagal reactivation seems to be dependent on aerobic fitness, since individuals with high aerobic fitness showed complete vagal reactivation after submaximal exercise in a shorter period of time than those with low aerobic fitness [32]. Additionally, the RMSSD index increased significantly following a period of maximal exercise at ~ 3 min of passive recovery in aerobically trained [10] and physically active individuals [9].

Vagal reactivation also depends of the HRV indexes values at rest, since normal weight women presented higher LnRMSSD and high-frequency index values post-exercise, compared to overweight/obese women and with lower LnRMSSD and high-frequency indexes at rest [17]. This study did not utilize the signal selection proposed by Goldberger et al. [8] in the HRV indexes analysis.

We did not examine frequency domain indexes because they could be influenced by the non-stationarity of recovery HRV data, by respiratory parameters and baroreflex sensitivity, independent of changes in cardiac autonomic nervous activity [21, 33, 34]. In addition, we evaluated the RMSSD and SDNN indexes in successive non-overlapped 30 s because they have been pharmacologically validated [8] and its clinical application has been proven [35].

A previous study has shown that the sympathetic nervous activity and sympatho–vagal balance remained superior to rest 15 min post-submaximal exercise in the elderly people [12]. The present study corroborates with a study that found the elevation of the SDNN index after 90 s of passive recovery in young people with higher VO_{2peak} than in our study [11]. In addition, a previous study reported that the sympatho–vagal reorganization preceded complete vagal restoration in the maximal post-exercise recovery [36].

Active recovery, due to increased blood flow to the active muscles, accelerates lactate removal and reduces H^+ and PO_2 levels [37], which may have contributed to the recovery of overall autonomic indexes. Thus, it may be suggested that the after maximal test exercise, the vagal reactivation is preceded by global variability indexes reestablishment. Also, this mechanism is not impaired in apparently healthy young adult men with obesity and preserved cardiac autonomic modulation at rest.

Several factors may explain the divergence between studies concerning the mechanisms involved in maximal post-exercise recovery, such as aerobic fitness [7, 10, 32], resting vagal activity [17], physical exercise modality performed [9] and the form of signal selection analysis of HRV indexes [36]. Another important factor is physical exercise intensity [20] since the RMSSD index differed throughout 10 min of recovery after light, moderate and intense exercise [20]. Similarly, high-intensity exercise caused a greater disturbance in cardiac autonomic modulation, since its reestablishment is delayed compared to moderate exercise [18].

This study has some limitations that must be considered. (1) The central obesity has a noticeable role in cardiovascular autonomic dysfunction, that we did no analyse in this study. Although we verified a high correlation between BMI and abdominal circumference (r: 0.952; $p < 0.05$). (2) The breathing pattern during recovery was not controlled similar the several previous studies [9–11, 17], because this recommendation can cause discomfort to the participant. (3) The lack of evaluation of lactate during post-exercise recovery. (4) Furthermore, this study was limited to apparently healthy young men. Thus, these findings cannot be extrapolated to clinical populations or older individuals. Perhaps a similar study that involves a broader age and BMI range or includes clinical populations may demonstrate different findings.

Conclusion

In summary, although there was no vagal reactivation at short-term after a maximal incremental exercise test, there were alterations in the overall HRV regardless of BMI in apparently healthy young adult men.

Our results suggest future studies with different populations, physical fitness level, a longer time for HRV post-exercise analysis, as well as, the sensibility to the training programs to assess the improvement in cardiac autonomic modulation.

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Compliance with ethical standards

Conflicts of interests The authors declare that they have no competing interests.

Ethical approval All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the Declaration of Helsinki. All procedures were approved by the University Research Ethics Committee.

Informed consent Informed consent was obtained from all individual participants in this study.

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