Indian J Physiol Pharmacol 2018; 62(1) : 20–31

Original Article

Dynamics of Heart Rate Responses to Exercise in Normotensive Men

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Abstract

We evaluated cardiovascular responses to exercise in normotensive men according to resting systolic arterial pressure (SAP). Healthy men were split into two groups: G1- Subjects with at rest SAP between 120 and 130 mmHg (n=15) and G2- Subjects with at rest SAP < 120 mmHg (n=20). These conventions were necessary in order to check the recovery of the autonomic nervous system after exercise. Subjects performed physical exercise on a treadmill with intensity equivalent to 60% of V_{max}. HR (heart rate) variability was recorded in the following stages: at rest, the 10-minute periods before exercise, during exercise and the 60 minutes periods after exercise. During recovery from exercise G2 presented delayed recovery compared to G1 based on SDNN, RMSSD, pNN50, RRTri, HF, LF, SD1 and SD2 indices. HR recovery in the 3rd minute was higher in G1. Body mass index was greater in G1. In conclusion, normotensive men with SAP below 120 mmHg established delayed HRV recovery following an acute bout of aerobic exercise compared to normotensive men.

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(Received on August 1, 2017)

Introduction

Cardiovascular responses during recovery from exercise are necessary in sports and exercise physiology to understand the mechanisms by which the autonomic nervous system (ANS) responds to

exercise. These mechanisms provide information on the autonomic function of the patient to the medical practitioner (1).

Heart rate recovery (HRR) is a simple and non-invasive method that analyses heart rate autonomic recovery after exercise (2). An alternative meticulously documented method that evaluates heart rate control is heart rate variability (HRV) (3), which expresses fluctuations in consecutive heart beats.

Studies reveal that a high HRV reflects the robustness of cardiovascular regulation controlling heart rate through forward and feedback modulations. HRV indicates the status of cardiovascular homeostatic mechanisms acting through sympathetic and parasympathetic branches of the ANS (4).

Previous studies established that reduced HRV reflects sympathetic hyperactivity (5), malignant ventricular arrhythmias (6), cardiomyocyte damage (7), coronary constriction (8) and mortality risks (5). Intrinsic deficiency causes the individual to be less able to endure a physiological disturbance, predisposing them to risks from all causes of death (5).

Exercise is a physiological stimulus that can tolerate physiological changes through shifting the autonomic balance (9). During aerobic exercise —in the first few minutes, vagal withdrawal occurs and then later with elevated exercise intensity, sympathetic activation ensues. Directly after termination of exercise vagal re-entry followed by sympathetic withdrawal; renewal of the autonomic balance is permissible (10).

Studies reveal that cardiac autonomic restoration is necessary for cardiovascular well-being, since a delay in vagal reactivation and a persistence of sympathetic activation may increase cardiac ectopic movement in the post-exercise period, increasing the risk of cardiovascular pathologies (11).

Studies with seemingly healthy subjects within normal arterial pressure range, a change in cardiovascular responses and HRV, signifying possible autonomic imbalance, could support the identification of risk factors for cardiovascular pathology and anticipate likely causes of these pathologies. Consequently, allowing the progression of therapies for restoration of the autonomic imbalance.

Regarding the 2007 Practice Guidelines for the Management of Arterial Hypertension (12), classification of blood pressure in adults based on systolic arterial pressure (SAP) is optimum for SAP lower than 120 mmHg, normal for 120 to 129 mmHg and higher than normal for 130 to 139 mmHg. Yet, the expected threshold level for hypertension is variable and reliant on the cardiovascular risk of each separate subject (12).

Further research is imperative using a healthy cohort that excludes cardiorespiratory diseases, and could conceivably contribute to the identification of pathological predispositions even with normal cardiovascular values. So, considering that the hemodynamic response to the exercise can provide evidence that is undetected in the resting state (13), the objective was to study cardiovascular responses to exercise in normotensive men with unalike resting SAP.

Methods

Study population

This is a prospective analytical study. The subjects participating in the study were healthy males - all non-smokers, aged between 18 and 35 years old, and were divided into two groups: G1-Subjects with SAP between 120 mmHg and 130 mmHg (n=15) and G2-Subjects with SAP between 110 mmHg and 120 mmHg (n=20). All subjects were physically active, performing moderate to intense physical activity at least 1 hour for 3 days per week, but not athletic. Subjects were informed about the procedures and the objectives of the study and gave confidential written informed consent. All procedures were approved by the Ethics Committee in Research of the Faculty of Sciences of the Universidade Estadual Paulista (No. CEP-2011-385), and were in accordance with Resolution 466/2012 National Health 10/10/1996. We excluded subjects with body mass index (BMI)

>30 kg/m^2; at rest SAP>130 mmHg and at rest diastolic arterial pressure (DAP) >90 mmHg; resting heart rate (HR) beyond 100 beats per minute, cardiovascular, respiratory and neurological disorders. Volunteers under medication(s) that influence HRV were also excluded.

Initial evaluation

Baseline information was collected: age, gender, mass, height and body mass index (BMI). Mass was determined using a digital scale (W 200/5, Welmy, Brazil) with a precision of 0.1 kg. Height was determined using a stadiometer (ES 2020, Sanny, Brazil) with a precision of 0.1 cm and 220 cm of extension. BMI was calculated as mass/height^2, with mass in kilograms and height in meters.

Cardiovascular variables

Measurements of blood pressure were monitored by the same evaluator indirectly using a stethoscope (Littmann, St. Paul, USA) and aneroid sphygmomanometer (WelchAllyn, Germany) on the left arm. The subjects were seated according to the criteria established by the VI Brazilian Guidelines on Hypertension (13). HR was obtained by a heart rate monitor – Polar RS800CX (Polar Electro, Kempele, Finland) (14). HRR was computed as the difference between the heart rate at the cessation of exercise and heart rate at 1 min (HRR1) or 3 min (HRR3) after termination of the exercise (1).

HRV analysis

We enforced directives from the Task Force publication (15). Instantaneous RR intervals (RRi) were logged with a digital telemetry system, consisting of a transmitter placed on the patient's chest and a HR monitor (Polar® RS800CX; Polar Electro Oy, Kempele, Finland). This system detects ventricular depolarization, corresponding to the R wave on the electrocardiogram, at a sampling rate of 1kHz and has been previously validated (16). They were downloaded to the Polar Precision Performance program (v.3.0, Polar Electro, Finland). The software enabled the visualization of HR and the extraction of a cardiac period (RR interval) file in "txt" format. After digital filtering accompanied with manual filtering for the elimination of premature ectopic beats and artefacts, 256 stationary RR intervals were necessary for the data analysis. Only series with sinus rhythm exceeding 95% were included in the study (17). HRV was examined in the period before the exercise (M1); during exercise (M2: 15-20 min during exercise; M3: 25-30 min during exercise) and after the period of acute exercise (M4: 5-10 minutes after exercise cessation; M5: 15-20 minutes after exercise cessation; M6: 25-30 minutes after exercise cessation; M7: 35-40 minutes after exercise cessation; M8: 45-50 minutes after exercise cessation; M9: 55-60 minutes after exercise cessation). All indices were evaluated using 256 fixed successive and stationary RR intervals.

Time and frequency domain indices of HRV

For HRV analysis in the frequency domain we approved the spectral components of low frequency (LF: 0.04 to 0.15 Hz) and high frequency (HF: 0.15 to 0.40 Hz) in absolute units (ms^2) and LF/HF ratio. The spectral analysis was calculated with the Fast Fourier Transform (FFT) algorithm (18).

Time domain analysis was monitored by the SDNN (average standard deviation of normal RR intervals), pNN50 (percentage of adjacent RR intervals lasting more difference than 50 ms) and RMSSD (square root of the average square differences between normal adjacent RR intervals).

HRV analysis was also performed by geometrical methods in time domain: RRtri (Triangular index), TINN (triangular interpolation of NN interval histogram) and Poincaré plot (SD1 and SD2). The RRtri was computed from the construction of a density histogram of RR intervals, which contains the horizontal axis of all possible values of RR intervals measured on a discrete scale with 7.8125 millisecond boxes (1/128 seconds) and on the vertical axis, the frequency with which each occurred. The union of points of the histogram columns produced a triangular shape. The RRtri was obtained by dividing the total number of RR intervals used to construct the histogram by their modal frequency — the RR interval value that most frequently appeared on RR.

The TINN comprises of the measure of the base of a triangle. The method of least squares is enforced to determine the triangle. The RRtri and the TINN express the overall variability of RR intervals (15). The Poincaré plot is a map of points in Cartesian coordinates, constructed from the values of RR intervals obtained, where each point is represented on the x-axis (horizontal) by the previous normal RR interval, and on y-axis (vertical), by the following RR interval. For quantitative analysis of the plot, an ellipse was fitted to the points of the chart, with the center determined by the average RR intervals, and the SD1 indexes were calculated to measure the standard deviation of the distances of the points to the diagonal y = x, and SD2 measures the standard deviation of the distances of points to the line y = –x + RRm, where RRm is the average of RR intervals. The SD1 is an index of immediate recording of the variability of fluctuations and represents parasympathetic modulation, whilst the index SD2 represents HRV in long-term records, and reflects Conconithe overall variability (19). For analysis of linear indices in the frequency and time domain we used the Kubios HRV® analysis software (20).

Aerobic potency measurement

For exercise intensity training, we applied 60% of V_{max} found in the progressive test through Conconi threshold, which has been proposed to estimate the anaerobic threshold for identifying the HR deflection point (HRDP) using a progressive test with the use of the D_{max} method (21).

Next, the subjects endured a systematic progressive treadmill test (TPEE; Inbrasport ATL 2000) with initial speed of 8 km / hour which incremented 1 km / hour every 2 minutes until exhaustion or onset of clinical changes that prevented the continuity of test, such as dizziness, shortness of breath or “air hunger” (22, 23). The inclination of the treadmill remained fixed at 1%, since this condition reflects more precisely the energy cost of running outdoors. We recognized volunteers that reached up to 90% of maximal HR (24).

For the identification of HRDF, the matched HR and speed points were plotted. Next the values were adjusted by means of a third-degree polynomial function and a linear equation of the first degree, which are data derived from each individual. Then, the difference of the values of HR obtained through the aforesaid equations were calculated and a curve was calculated with these values. We considered the PDFC as the highest value before a change of direction in the curve (24).

Exercise protocol

Data collection originated in the same soundproofed room for all subjects. The temperature was between 21°C and 25°C and the relative humidity between 50% and 60%. Subjects were instructed not to ingest alcohol, caffeine or other ANS stimulants for 24 hours before the evaluation. Data sets were collected on an individual basis, continuously between 18:00 and 21:00 to standardize circadian influences. All procedures necessary for the data collection were explained to each subject discretely, and the subjects were instructed to remain at rest and avoid speaking during the collection.

Volunteers performed physical exercise on a treadmill with intensity of 6.0 km/hour + 1% slope in the first five minutes for physically “warming up”, followed by 25 minutes with intensity equivalent to 60% of V_{max} according to the Conconi threshold with identical slope.

Statistical analysis

Standard statistical techniques were enforced for the calculation of means and standard deviations. Normal Gaussian distribution of the data was verified by the Shapiro-Wilk goodness-of-fit test (z value >1.0) (25). To compare variables between groups, for parametric distributions we computed non-paired Student t-test and for non-parametric distributions we applied Mann-Whitney test.

To equate variables in the exercise protocol (control at rest, during exercise and post-exercise) two-way repeated measures analysis of variance was applied, then the Bonferroni post-test for parametric distributions and Friedman followed by Dunn’s test for non-parametric distributions. Differences were

considered significant when the probability of a Type I error was less than 5% ($p<0.05$).

To assess correlation between HRV indices and HRR we enforced Pearson correlation coefficient analysis for parametric distribution and Spearman's Rank correlation coefficient analysis for non-parametric distributions. Strong correlation was assumed for $r>0.5$, moderate correlation for r between 0.3 and 0.5.

To quantify the magnitude of difference between groups and moments, the effect size was calculated using Cohen's *d* for significant differences. Large effect sized was considered for values ≥ 0.9, medium for values between 0.9 and 0.5 and small between 0.5 and 0.25 (19). We used the Software Biostat® 2009 Professional 5.8.4 (Analysis Soft, Walnut, California, United States of America).

Results

Table I illustrates descriptive statistics regarding mass, height and BMI. We detected significant increased BMI and mass in G1. HRR in the first minute was unaffected between groups, however, HRR in the third minute was significantly faster in the G2 (Table I). As expected, at rest SAP ($p<0.0001$) and at rest DAP were significantly higher in the G1 ($p<0.0001$).

We detected that during exercise there was a time effect for all variables ($p<0.0001$) except LF/HF ratio ($p=0.481$). No group effect was achieved regarding RRTri ($p=0.196$), TINN ($p=0.422$), HF ($p=0.599$), LF/HF ratio ($p=0.401$), SD1 ($p=0.522$) and SD2 ($p=0.464$), and there was a group effect in LF index ($p=0.039$). Regarding the moment and group interaction we noted significant differences in the LF ($p=0.049$); for the other indices there were no interactions (RRTri, $p=0.496$; TINN, $p=0.211$; HF, $p=0.616$; LF / HF ratio, $p=0.409$; SD1, $p=0.438$ and; SD2, $p=0.660$) (Fig. 1).

During exercise (M2 and M3) we detected decreases in LF and HF compared to at rest (M1) in both groups and the LF/HF ratio significantly increased compared to at rest in both groups. Regarding the Poincaré plot indices, in both groups we noticed a significant reduction in SD1 and SD2 indices during exercise (M2 and M3) compared to at rest (M1) (Fig. 1).

Regarding the time domain indices during exercise (M2 and M3) we detected no group effect (Mean RR, $p=0.155$; SDNN, $p=0.464$; RMSSD, $p=0.524$; pNN50, $p=0.577$). There were no time or group interactions for mean RR intervals, $p=0.180$; SDNN, $p=0.634$; RMSSD, $p=0.438$ and; pNN50, $p=0.577$ (Fig. 2).

During recovery from exercise, there was a time effect for most indices ($p<0.0001$), except TINN ($p=0.478$) and LF/HF ratio ($p=0.138$). No group effect was detected for the indices (Mean RR, $p=0.466$; SDNN, $p=0.920$; RMSSD, $p=0.637$; pNN50, $p=0.678$; RRTri, $p=0.987$; TINN, $p=0.083$; LF, $p=0.386$; HF, $p=0.708$; LF/HF ratio, $p=0.075$; SD1, $p=0.714$; SD2, $p=0.826$) and there were no time or group interactions (Mean RR, $p=0.264$; SDNN, $p=0.868$; rMSSD, $p=0.274$; pNN50, $p=0.311$; RRTri, $p=0.826$; TINN $p=0.459$; LF, $p=0.2$; HF, $p=0.543$; LF/HF ratio, $p=0.581$; SD1,

TABLE I: Descriptive statistics of mass, height and body mass index (BMI), heart rate recovery at the first (HRR1) and third minute (HRR3) of the volunteers divided by group. m: meters; bpm: beats per minute; kg: kilograms; p indicates difference betweenPre VS Post (5-10 min after exercise cessation).G1: Group with SAP > 120 mmHg; G2: Group with SAP < 120 mmHg. Mean±standard deviation (minimum-maximum).

Variables	*G1*	*G2*	*p*	*Cohen's*	*Effect size*
Age (years)	21.37±2.9 (19-24)	22.05±3.2 (18-29)	0.22	0.22	Small
Height (m)	1.76±0.05 (1.7-1.85)	1.75±0.09 (1.6-1.88)	0.26	0.13	Small
Mass (kg)	79.74±8.59 (65.7-97)	72.41±10.01 (57-89.35)	0.01	0.78	Medium
BMI (kg/m^2)	25.55±7.23 (26.25-21.36)	23.56±1.99 (19.33-26.81)	0.009	0.37	Small
HRR1 (bpm)	22.85±12.8 (2-48)	17.8±12.7 (4-57)	0.13	0.39	Small
HRR3 (bpm)	42.85±15.52 (24-87)	32.9±11.15 (21-58)	0.03	0.73	Medium

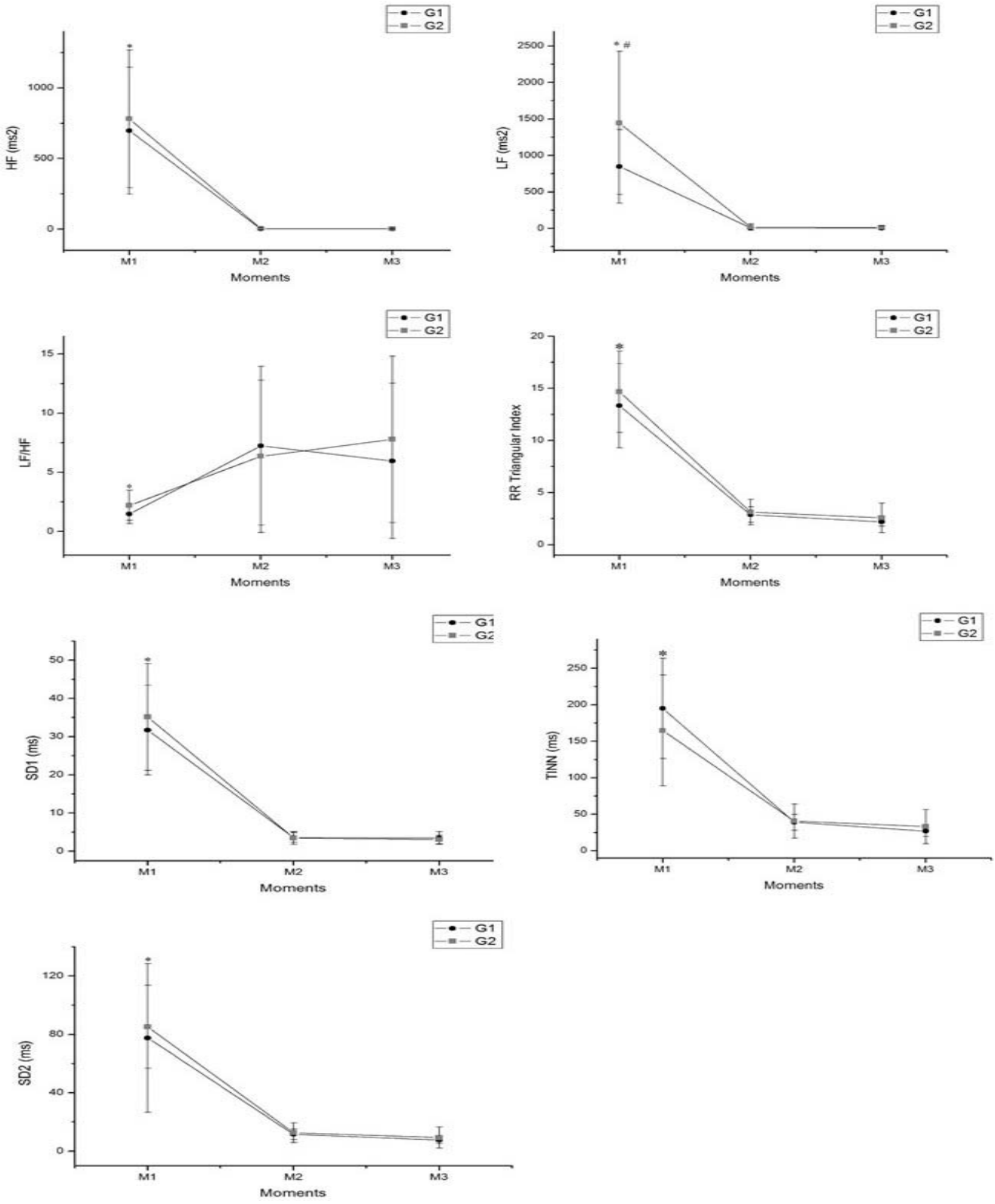

Fig. 1 : Frequency domain and geometric analysis of HRV before and during exercise. M1: Control at rest; M2: 10-15 minutes after start of exercise; M3: 25-30 minutes after start of exercise; G1: Group with SAP > 120 mmHg; G2: Group with SAP < 120 mmHg; LF: low frequency; HF: high frequency; LF/HF: low frequency/high frequency ratio; ms: milliseconds; SD1: standard deviation of the instantaneous variability of the beat-to beat heart rate; SD2: standard deviation of long-term continuous RR interval variability; TINN: Triangular interpolation of RR interval histogram; *p<0.05 Vs. M2 and M3; #p<0.05: G1 vs. G2.

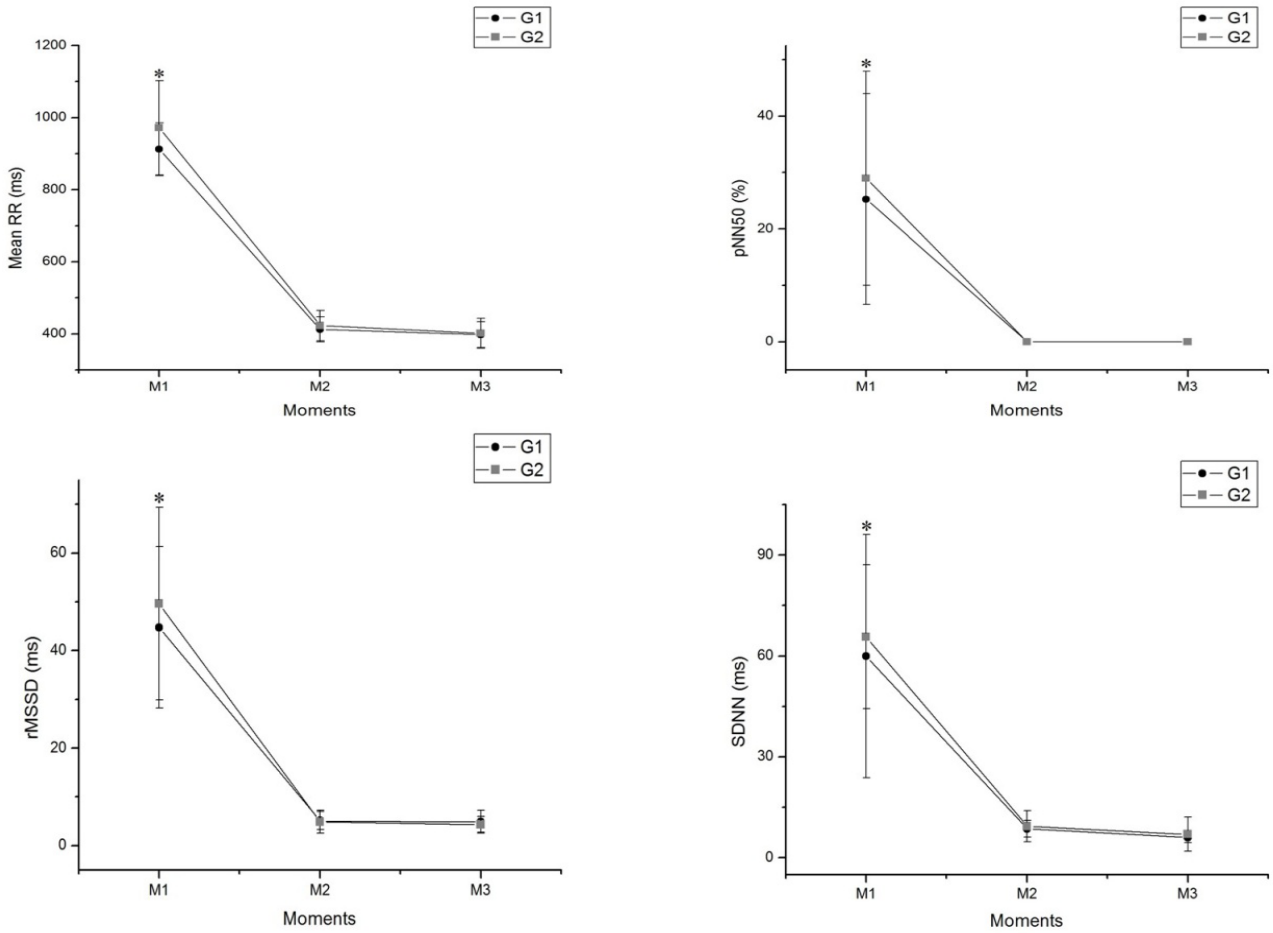

Fig. 2 : Time domain analysis of HRV before and during exercise. M1: Control at rest; M2: 10-15 minutes after start of exercise; M3: 25-30 minutes after start of exercise; G1: Group with SAP > 120 mmHg; G2: Group with SAP < 120 mmHg; pNN50: the percentage of adjacent RR intervals with a difference of duration greater than 50 ms; RMSSD: root-mean square of differences between adjacent normal RR intervals in a time interval; ms: milliseconds; SDNN: Average standard deviation of normal RR intervals; *p<0.05 Vs. M2 and M3.

p=0.452; SD2, p=0.867) (Fig. 3 and Fig. 4).

The RRTri index was lessened in M4 compared to M1 in G1 whereas in G2 it was reduced in M4 and M5 compared to M1 (Fig. 3). In both groups LF was diminished in M1 compared to M4 and M5. HF was narrowed in M1 compared to M4 and M5 in G2 and it was declined in M4 compared to M1 in G1. The LF/HF ratio was increased in M4 compared to M1 in both groups. Concerning the SD1 index, it was lessened in M4 and M5 compared to M1 in G1 while it declined in M4, M5 and M6 compared to M1 in G1. The SD2 index was diminishing in M4 and M5 compared to M1 in G2 (Fig. 3).

The mean RR interval was statistically reduced in all cases compared to M1 in G1, while in G2 it was reduced in M4, M5, M6 and M7 compared to M1. RMSSD and pNN50 indices were decreased in M4 and M5 compared to M1 in G1 and it declined in M4, M5 and M6 compared to M1 in G2. Regarding SDNN we found a reduction in M4 and M5 compared to M1 in G2 (Fig. 4).

Table II demonstrates statistical correlation of HRR1 and HRR3 with HRV indices at rest. We recognized no significant correlation in the G2. Yet, HRR3 presented significant negative correlation with RMSSD, pNN50, HF and SD1.

Table III displays correlation between BMI and cardiovascular variables before and after exercise (5 to 10 minutes after exercise cessation). Significant negative correlation of BMI with RMSSD and pNN50

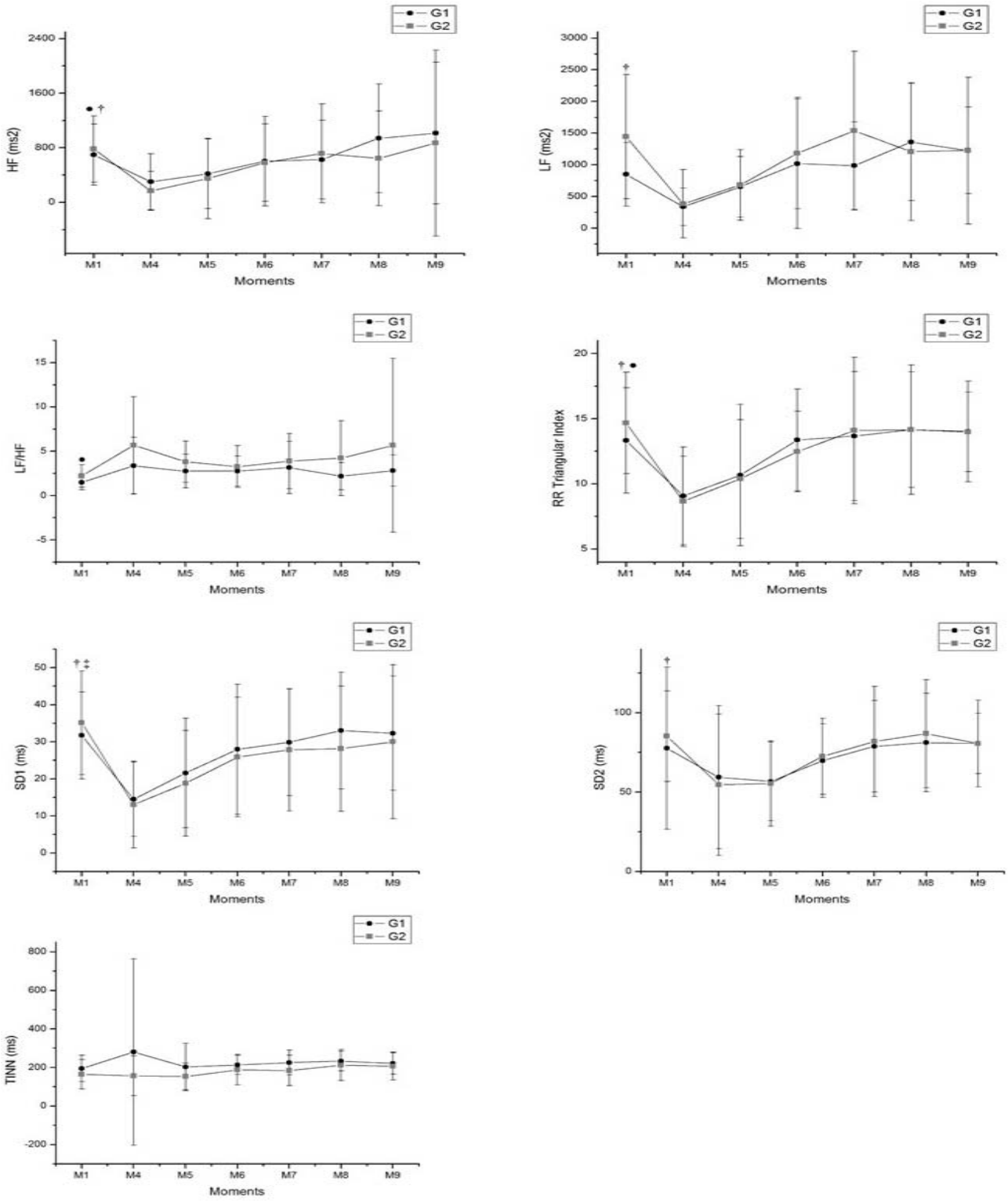

Fig. 3 : Frequency domain and geometric analysis of HRV before and after exercise. M1: Control at rest; M4: 5-10 minutes after exercise cessation; M5: 15-20 minutes after exercise cessation; M6: 25-30 minutes after exercise cessation; M7: 35-40 minutes after exercise cessation; M8: 45-50 minutes after exercise cessation; M9: 55-60 minutes after exercise cessation; G1: Group with SAP > 120 mmHg; G2: Group with SAP < 120 mmHg; LF: low frequency; HF: high frequency; LF/HF: low frequency/ high frequency ratio; ms: milliseconds; SD1: standard deviation of the instantaneous variability of the beat-to beat heart rate; SD2: standard deviation of long-term continuous RR interval variability; TINN: Triangular interpolation of RR interval histogram; $^{\dagger}p<0.05$ Vs. M4 and M5 in G2; $^{\%}p<0.05$ Vs. M4 in G1; $^{\dagger}p<0.05$ Vs. M4 and M5 in G1 and G2 for LF and in G2 for HF; $^{\%}p<0.05$ Vs. M4 in G1 for HF (ms2) and LF/HF ratio.

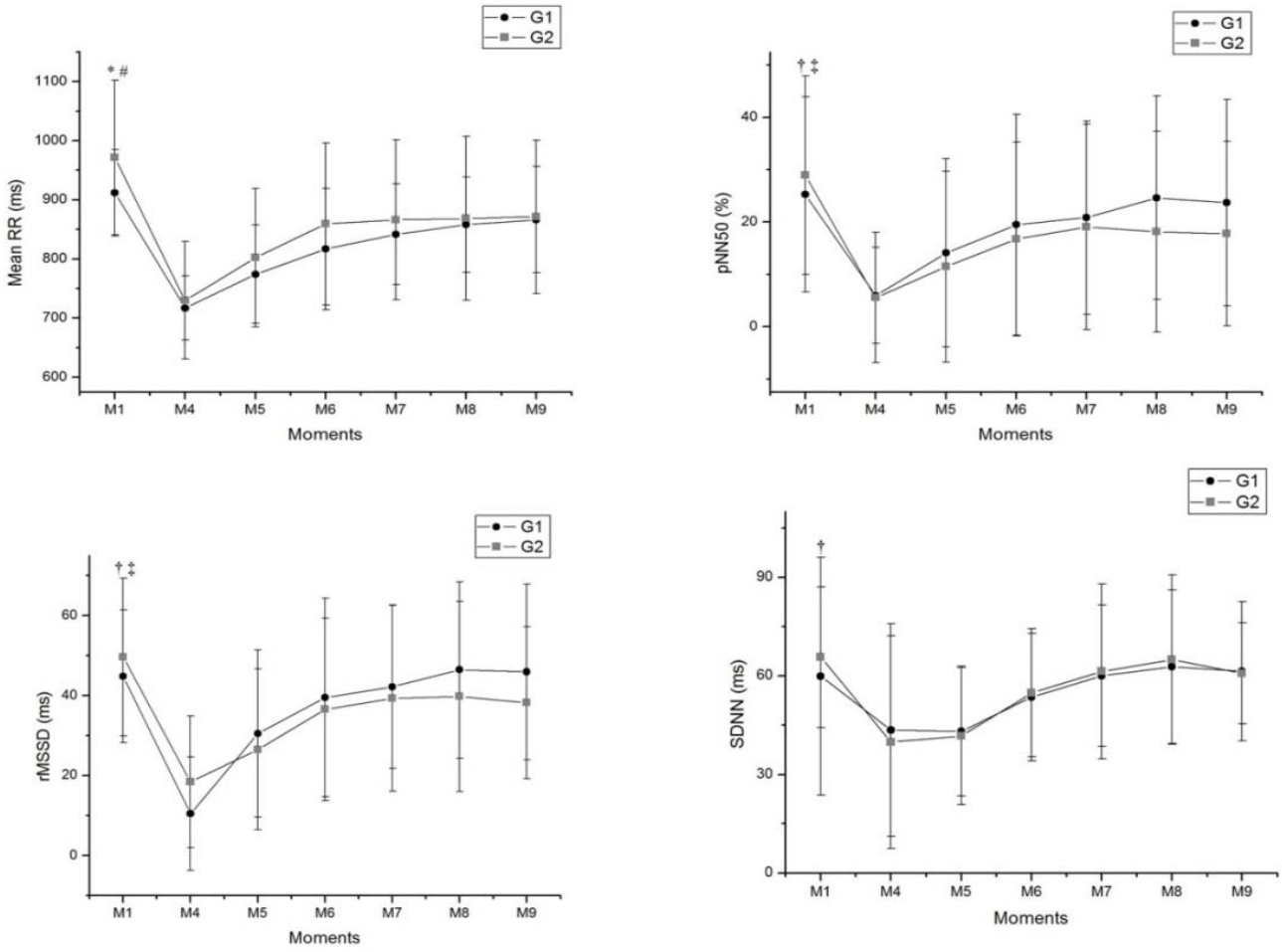

Fig. 4 : Time domain analysis of HRV before and after exercise. M1: Control at rest; M4: 5-10 minutes after exercise cessation; M5: 15-20 minutes after exercise cessation; M6: 25-30 minutes after exercise cessation; M7: 35-40 minutes after exercise cessation; M8: 45-50 minutes after exercise cessation; M9: 55-60 minutes after exercise cessation; G1: Group with SAP > 120 mmHg; G2: Group with SAP < 120 mmHg;*p<0.05 Vs. M4, M5, M6, M7, M8 and M9 in G1; #p<0.05 Vs. M4, M5, M6 and M7 in G2; †p<0.05 Vs. M4 and M5 in G2 for SDNN and in G1 for RMSSD and pNN50; ‡p<0.05 Vs. M4, M5 and M6 in G2 for RMSSD and pNN50; %p<0.05 Vs. M4 in G1.

TABLE II : Correlation of HRR1 and HRR3 with HRV indices at rest and during recovery from exercise. G1: Group with SAP > 120 mmHg; G2: Group with SAP < 120 mmHg.

G1	*HRR1*		*HRR3*	
HRV Index	*r*	*p*	*r*	*p*
RMSSD	–0.52	0.057	–0.58	0.02
pNN50	–0.47	0.09	–0.54	0.04
HF	–0.47	0.08	–0.55	0.04
SD1	–0.51	0.58	–0.58	0.02
G2	*HRR1*		*HRR3*	
HRV Index	*r*	*p*	*r*	*p*
RMSSD	–0.35	0.13	–0.34	0.051
pNN50	0.08	0.7	–0.3	0.19
HF	–0.044	0.85	–0.41	0.07
SD1	–0.004	0.98	–0.35	0.13

TABLE III : Correlation between BMI and cardiovascular parameters at rest (pre) and during recovery from exercise (post; 5–10 min after exercise cessation). G1: Group with SAP > 120 mmHg; G2: Group with SAP < 120 mmHg.

	G1		*G2*	
Variable	*r*	*p*	*r*	*p*
RMSSD pre	–0.64	0.01	0.07	0.74
pNN50 pre	0.48	0.08	–0.06	0.77
HF pre	0.38	0.18	0.12	0.6
SD1 pre	0.45	0.1	–0.035	0.88
RMSSD post	0.32	0.26	–0.55	0.01
pNN50 post	0.31	0.26	–0.53	0.01
HF post	0.48	0.08	–0.36	0.12
SD1 post	0.32	0.26	–0.4	0.051
HRR1	0.3	0.28	–0.12	0.6
HRR3	0.19	0.49	–0.22	0.34

after exercise was observed in the G2. BMI was negatively correlated with at rest RMSSD in the G1.

Discussion

We predicted estimating the HRV responses to exercise in normotensive physically active men divided into two groups (below 120 mmHg and between 120 and 130 mmHg). From our data, males with reduced SAP achieved delayed HRV recovery from exercise and decreased HRR compared to normotensive men with higher SAP. HRR was unconnected with rest parasympathetic heart rate regulation, BMI was higher in men with lower SAP and it was negatively correlated with rest vagal control in the identical group, which advocates that the slower recovery of HRV is attributable to increased BMI in the population being studied.

In this study, HRR in the first minute was unchanged between groups. Yet, HRR in the third minute was higher in the normotensive men with higher SAP. This reveals that the subjects with higher SAP experienced better autonomic read justment after exercise.

HRR is split into two stages; the first 60 seconds corresponds to the fast recovery, indicating an immediate and rapid reduction in heart rate. The slow stage includes the period after the first minute (26). Increased HRR is associated to enhanced physical fitness. Prior studies have accomplished faster HRR in physically trained individuals and athletes compared to sedentary control subjects (27, 28).

Furthermore, the slow phase was reported to be influenced by exercise intensity (27). Considering that we investigated a moderate intensity exercise, we are unable to extrapolate our data to further intense exercises.

Based on statistical analysis, normotensive men with lower SAP presented delayed recovery of mean RR interval, SDNN, pNN50, RMSSD, HF, RRTri, SD1 and SD2 indices from exercise compared to the group with higher at rest SAP. In this state, we specify that the parasympathetic modulation of HR is involved in the delayed recovery of HRV from exercise in the volunteers with lower SAP.

Impaired responses of parasympathetic HR regulation to exercise were testified in subjects with cardiovascular disorders and a predictor of mortality (29, 30). In a study by Cole et al. (29), it was reinforced that HRR could be enforced as an indicator of vagal heart rate modulation. The authors conveyed decreased values in patients with cardiovascular disorders. Myers et al. (30) assessed the association between cardiovascular parameters in response to exercise and prediction with cardiovascular risk. The authors revealed that reduced HRR was related to cardiovascular risk — the lower the HRR, the higher the cardiovascular risk.

We anticipated that the sympatho-vagal balance would be involved in the variance between both groups. Nonetheless, based on our data, there was no group effect regarding LF/HF ratio during recovery from exercise. This outcome excludes the involvement of the sympatho-vagal balance component of heart rate modulation in the delayed HRV recovery after aerobic exercise.

To confirm whether HRR was associated with at rest parasympathetic heart rate modulation we began a statistical correlation between the variables. The group with higher SAP offered negative correlation with all parasympathetic HRV indices, indicating that increased HRR in the third minute was associated with lower HRV in normotensive men with higher SAP.

Contrariwise, the slower recovery of HRV from exercise in men with lower SAP does not guarantee that this population endures risk factors to developing cardiovascular disorders. The individuals with higher SAP had elevated HRR, which issymptomatic of improved adjustment of the ANS in response to exercise (1). From both groups all volunteers were healthy, physically active and had no medical history of cardiorespiratory illnesses.

A point to highlight is concerning BMI and body mass. In this study, both variables were significantly higher in men with lower SAP, and this was accompanied by delayed HRV during recovery from

exercise. BMI was associated with HRR after exercise in healthy adults. The volunteers were divided into normal BMI, overweight and obese (31). It was noticed that BMI was inversely related with HRR after exercise. But it was recognized that subjects with impaired HRV recovery presented diminished exercise capacity than those with faster recovery.

The aforesaid study supports our data and may clarify our results, since the group with lower SAP presented higher BMI and slower HRR. It is rational to suggest that BMI was involved in the delayed recovery of HRV after exercise.

Also, we commenced a statistical correlation between BMI and cardiovascular parameters before and after exercise to further examine the role of BMI. In the group with lower SAP we detected that BMI inversely influenced parasympathetic heart rate modulation during recovery from exercise. As an alternative, BMI negatively influenced at rest HRV in the group with higher SAP. Together, it is proposed that BMI influences HRV during recovery from exercise in male normotensive subjects with lower SAP.

Therefore as a principle outcome, normotensive subjects with lower SAP (less than 120 mmHg) presented delayed HRV recovery and slower HRR to exercise compared to normotensive subjects with higher SAP (between 120 and 130 mmHg). Based on our data, we suggest that males with SAP ranging between 120 and 130 mmHg have better autonomic adjustment after moderate exercise. Our data reveals an important issue related to at rest SAP and its influence on heart rate dynamic responses to exercise.

As a limitation of the study, while every possible attention was taken regarding the selection and filtering of HRV data, we can not exclude possible misapplications of the stationarity of heart rate fluctuations during exercise and the post-exercise phases.

Conclusion

Normotensive males with lower SAP present slower recovery of HRV during recovery from exercise compared to normotensive males with higher SAP. We conclude that this is attributable to higher BMI in this specific population.

Acknowledgements

The study received financial support from Capes.

Conflict of interest statements

The authors declare that there is no conflict of interests regarding the publication of this article.

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