CLINICAL SCIENCE

Cardiovascular and autonomic responses after exercise sessions with different intensities and durations

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BACKGROUND: Several studies have reported the phenomenon of post-exercise hypotension. However, the factors that cause this drop in blood pressure after a single exercise session are still unknown.

OBJECTIVE: To investigate the effects of aerobic exercise on the acute blood pressure response and to investigate the indicators of autonomic activity after exercise.

METHODS: Ten male subjects (aged 25 $\pm$ 1 years) underwent four experimental exercise sessions and a control session on a cycle ergometer. The blood pressure and heart rate variability of each subject were measured at rest and at 60 min after the end of the sessions.

RESULTS: Post-exercise hypotension was not observed in any experimental sessions ($P > 0.05$). The index of parasympathetic neural activity, the RMSSD, only remained lower than that during the pre-exercise session after the high-intensity session (Δ =-19 $\pm$ 3.7 for 15-20 min post-exercise). In addition, this value varied significantly ($P < 0.05$) between the high- and low-intensity sessions (Δ = -30.7 $\pm$ 4.0 for the high intensity session, and Δ = -9.9 $\pm$ 2.5 for the low intensity session).

CONCLUSION: The present study did not find a reduction in blood pressure after exercise in normotensive, physically active young adults. However, the measurements of the indicators of autonomic neural activity revealed that in exercise of greater intensity the parasympathetic recovery tends to be slower and that sympathetic withdrawal can apparently compensate for this delay in recovery.

KEYWORDS: Blood Pressure; Heart Rate Variability; Exercise; Post-Exercise Hypotension; Cardiovascular Physiology.

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INTRODUCTION

Post-exercise hypotension (PEH) is characterized by a blood pressure (BP) value that is lower than the pre-exercise value that persists for minutes or hours after an exercise session.[1] Although the first report on PEH[2] was published over 100 years ago, systematic investigations of this phenomenon did not begin until the 1980s. The interest in the PEH has been mainly motivated by its clinical implications; it has potential as a non-pharmacological agent that can treat several cardiovascular disorders, such as hypertension.[3,4]

The exact mechanisms that are responsible for PEH remain unknown. However, several indicators suggest that sympathetic neural activity may be inhibited during PEH, which could cause a reduction in the peripheral vascular resistance.[5] In this context, heart rate variability (HRV) has emerged as a simple and noninvasive measure of autonomic impulses; this measure represents one of the most promising quantitative markers of the functioning of the autonomic nervous system.[6]

Despite the physiological mechanisms involved, PEH may depend on the specifics of the exercise session. The relationship between the intensity and duration of the exercise session and PEH is still controversial; several studies have reported a positive correlation between the intensity[7-9] and duration[7,10,11] of the session and the magnitude and/or duration of the PEH, whereas other experiments have shown no such relationship,[12-14] particularly in normotensive subjects. The majority of these experiments, however, were designed to investigate the

effect of only one variable (the intensity or duration of the session) on PEH. Because the intensity and duration of exercise can be adjusted simultaneously, the relationship between the interaction of these two variables and PEH is of interest. Therefore, the present study aimed to investigate the effects of four endurance exercise sessions with different intensities and durations on the BP values and autonomic indicators in a non-hypertensive sample.

METHODS

Subjects

Assuming a standard deviation of 10 mmHg[7] for the systolic BP (SBP), an alpha of 5% and a desired statistical power of 80%, detecting a minimum difference of 8 mmHg[7] in the SBP required 10 subjects. Therefore, ten healthy young men that were recreationally active and were without arterial hypertension[15] were recruited for the present study. After being informed of the study procedures, all of the subjects signed an informed consent form, according to the norms of the Resolution 196/96 of the National Health Council. The present study was approved by the Ethics and Research Committee (decision 022/2008).

The participants did not use tobacco and/or drugs that could interfere with the cardiovascular variables that were being measured in this study. Additionally, the subjects did not ingest alcohol and/or caffeinated beverages for at least 12 h before the exercise sessions and the assessment and did not participate in physical exercise and/or vigorous activity for at least 24 h before the experimental session. Initially, each subject's body mass was measured using a digital anthropometric scale (Uranus, OS 180A, Canoas, Brazil) and his height was measured using a wooden stadiometer. The general characteristics of the sample are presented in Table 1.

The subjects were randomly divided into five groups (four aerobic exercise groups and one control group) using a random number table with concealment allocation. The exercise groups corresponded to the following exercise sessions: an intense short session, a moderate long session, a moderate short session and a light long session. All of the subjects participated in all of the exercise sessions.

Blood pressure measurements

The SBP and the diastolic blood pressure (DBP) values were measured using automatic oscillometric equipment (Omron HEM 742-E, Bannockburn, USA). The subjects were assessed in a seated position following 10 min of rest. Three measurements were performed using the subject's left arm, with an interval of 5 min between each measurement; this procedure was consistent with the recommendations of the Fifth Brazilian Guidelines on Hypertension.[15] The resting BP was defined as the mean of the three measurements.

Table 1 - General characteristics of the sample.

Variables	Values (n = 10)
Age (yrs)	24.70 ± 1.27
Body mass (kg)	75.70 ± 3.44
Height (m)	1.75 ± 0.03
BMI ($kg.m^{-2}$)	24.58 ± 0.41
VO_{2peak} ($ml.kg^{-1}.min^{-1}$)	37.92 ± 1.26
Resting SBP (mmHg)	115.60 ± 2.26
Resting DBP (mmHg)	68.60 ± 1.41

The values presented are the mean ± the SE.
BMI = body mass index, SBP = systolic blood pressure and DBP = diastolic blood pressure.

Measures of heart rate variability

The heart rate variability (HRV) were assessed using a heart rate (HR) monitor (Polar S810i, Kempele, Finland). The RR intervals were recorded using this equipment, and all of the signals were transferred to a computer using *Polar Precision Performance software* (release 3.00, Kempele, Finland). A Fourier transform was used to quantify the low ($LF_{R\text{-}R}^{nu}$) and high frequency ($HF_{R\text{-}R}^{nu}$) bands in normalized units, which was in agreement with the recommendations of the Task Force of the European Society of Cardiology and the North-American Society of Electrophysiology.[16] The analysis in the time domain was performed using the root mean square of the squared successive differences between the adjacent R-R intervals (RMSSD). The analysis of the variables that were related to the time and frequency domains was performed in windows of 5 min, using the *HRV Analysis software*, version 1.1 (Kuopio, Finland).

Experimental Design

Before the beginning of the experiment, the subjects underwent anthropometric and hemodynamic assessments. Next, the VO_{2peak} was determined for each participant using a progressive test protocol, a cycle ergometer[17] and a K4b2 ergospirometer (Cosmed, Rome, Italy), with data filtering every 10 seconds. The participants performed 5 min of submaximal exercise as a standard warm-up. The work rate started at 50 W and was increased by 25 W every two minutes until voluntary exhaustion was reached. Voluntary exhaustion was defined as the point at which the subject failed to maintain the working frequency (60 rpm). The oxygen consumption of each participant was plotted as a function of their exercise work rate at 40%, 60% and 80% of their VO_{2peak} using a linear regression.

After a minimum interval of 48 h, the subjects were taken to a peaceful, noise-free environment, and their resting BPs were assessed after they had been sitting for 10 min. The subjects then used a cycle ergometer to perform the exercise session to which they had been allocated. The minimum interval between these sessions was 24 h.

After performing the exercise sessions, the subjects sat in a noise-free environment, with a temperature between 15℃-22℃ and a relative humidity between 59%-80%, for 60 min of BP, HR and HRV monitoring. The BP was measured at 5, 10, 15, 20, 30, 40, 50 and 60 min post-exercise. The HR and HRV were monitored continuously.

Table 2 - Description of the exercise sessions.

	Intensity	Duration
Intense short session (ISS)	80% VO_{2peak}	30 min
Moderate long session (MLS)	60% VO_{2peak}	$T = W_{T(ISS)} \div I$
Moderate short session (MSS)	60% VO_{2peak}	30 min
Light long session (LLS)	40% VO_{2peak}	$T = W_{T(MSS)} \div I$
Control session (CS)*	-------	30 min

*The subjects remained seated in a calm environment. T = Session duration (min), $W_{T(ISS)}$ = Total work (Watts) of ISS → $W_{T(ISS)} = W_{(ISS)} \times T_{(ISS)}$, I = Intensity (Watts) and $W_{T(MSS)}$ = Total work (Watts) of MSS → $W_{T(MSS)} = W_{(MSS)} \times T_{(MSS)}$.

Statistical analysis

The data were analyzed using a repeated measures ANOVA model. Before this analysis, we used Mauchly's test to verify the sphericity of the data. The Bonferroni *post-hoc* correction was used to test for pairwise group differences. After analyzing the distribution of the data using the Shapiro-Wilk's test, we calculated the Spearman correlation coefficients for the relationships between the SBPs at rest and the changes in these values after exercise. The delta values represent the difference between the post-exercise values and the rest values. The significance level was set at 5%. All of the calculations were performed using SPSS 13.0 software.

RESULTS

Two subjects started on the intense short exercise session, two subjects started on the moderate long session, one subject started on the moderate short session, one subject started on the light long session and six subjects started on the control session. The means and standard errors of the work intensities for the intensive session (the intense short session), the moderate sessions (the moderate long session and the moderate short session) and the light session (the light long session) were 134.0 $\pm$ 5.4 W, 87.6 $\pm$ 5.2 W and 45.8 $\pm$ 3.5 W, respectively.

The values of the hemodynamic and autonomic variables that were measured prior to the exercise sessions are shown in Table 3. No significant differences between the pre-intervention values for the exercise sessions were observed.

Figure 1 shows the variation in the post-exercise SBPs and DBPs over time. The SBP behavior was similar for all of the exercise sessions, with no significant differences in the resting values. There was a significant difference in the DBP at the first post-exercise measurement time point when the intense short session's value and the resting value were compared (7.1 $\pm$ 1.2 mmHg).

We also identified a strong relationship (r = 0.81, P = 0.004) between the SBP at rest and the post-exercise variation.

The changes in the autonomic indicators of neural activity that were observed after all of the exercise sessions are shown in Table 4. Compared with the pre-exercise values, there were no significant changes in the $LF_{R\text{-}R}{}^{nu}$ or $HF_{R\text{-}R}{}^{nu}$ after the intense short session, the moderate short session, the light long session and the control session or in the LF/HF after any of the sessions . There was a significant increase in the $LF_{R\text{-}R}{}^{nu}$ and $HF_{R\text{-}R}{}^{nu}$ between zero and five minutes post-exercise for the moderate long session.

By contrast, the RMSSD variable decreased significantly from the resting values in the first 5 min after the intense short session, the moderate long session and the moderate short session and in the interval from 15 min to 20 min after the intense short session and after the control session. Furthermore, this variable decreased significantly from the pre-exercise value in the interval from 55 min to 60 min after the light long session and in the intervals from 35 min to 40 min and 55 min to 60 min after the control session.

When comparing the sessions, the increase in the RMSSD variable at 0-5 min after exercise for the control session was significantly greater than the increases during the same time interval after all of the other sessions. The reductions in this variable during the intervals from 15 min to 20 min and 35 min to 40 min post-exercise for the intense short session were significantly greater than those for the control session, the moderate short session and the moderate long session.

DISCUSSION

The present study sought to identify the relationship between the duration (a moderate long session *vs.* a moderate short session), intensity (an intense short session *vs*. a moderate short session and a moderate long session *vs.* a light long session) and total amount of work (an intense short session *vs.* a moderate short session and a moderate long session *vs.* a light long session) of an aerobic exercise session and the magnitude and duration of the hypotensive response after exercise using four experimental conditions. Similar to several previous studies, the present study did not find significant evidence for the PEH phenomenon in normotensive individuals.[18-21] However, several other investigations have observed PEH in normotensive individuals.[7,8,22,23]

The present study found that there may be a relationship between the resting BP values and the magnitude of PEH in normotensive individuals; for one of the sessions, the subjects who had higher initial BP values showed a more pronounced reduction in BP (r = 0.81, P = 0.004). In the present study, this relationship was independent of the significant reduction in BP. Similarly, in the 1950s, Wilder[24] found (in what was called ''the law of initial values'') that the physiological responses to stress may be related to the initial values of the variables . This fact has been established not only for the relationship between exercise and BP[13,25-28] but also for the other variables that are related to health and

Table 3 - Hemodynamic parameters and pre-exercise autonomic indicators.

	ISS	MLS	MSS	LLS	CS
SBP (mmHg)	114.3 $\pm$ 2.9	113.3 $\pm$ 2.4	114.8 $\pm$ 3.0	115.0 $\pm$ 2.2	113.8 $\pm$ 2.6
DBP (mmHg)	66.8 $\pm$ 2.3	67.0 $\pm$ 1.9	66.8 $\pm$ 3.2	66.1 $\pm$ 2.2	66.4 $\pm$ 1.8
HR (bpm/min)	69.4 $\pm$ 4.1	66.0 $\pm$ 3.5	76.6 $\pm$ 4.3	75.9 $\pm$ 4.3	76.6 $\pm$ 3.9
RMSSD	44.9 $\pm$ 7.8	47.5 $\pm$ 5.2	39.1 $\pm$ 6.0	43.4 $\pm$ 5.5	36.4 $\pm$ 5.5
$LF_{R\text{-}R}{}^{nu}$	72.6 $\pm$ 4.4	71.3 $\pm$ 3.0	76.1 $\pm$ 3.4	68.2 $\pm$ 5.5	75.5 $\pm$ 3.4
$HF_{R\text{-}R}{}^{nu}$	27.4 $\pm$ 4.4	28.6 $\pm$ 3.0	21.4 $\pm$ 4.0	31.7 $\pm$ 5.5	24.4 $\pm$ 3.4
LF/HF	3.5 $\pm$ 0.7	2.7 $\pm$ 0.4	4.0 $\pm$ 0.7	2.9 $\pm$ 0.5	4.1 $\pm$ 1.0

The values presented are the mean $\pm$ the SE.
ISS = intense short session, MLS = moderate long session, MSS = moderate short session, LLS = light long session, CS = control session, SBP = systolic blood pressure, DBP = diastolic blood pressure, HR = heart rate; RMSSD = the square root of the mean of the sum of the squares of differences between adjacent NN intervals, $LF_{R\text{-}R}{}^{nu}$ = low frequency power in normalized units, $HF_{R\text{-}R}{}^{nu}$ = high frequency power in normalized units and LF/HF = ratio LF [ms^2]/HF [ms^2].

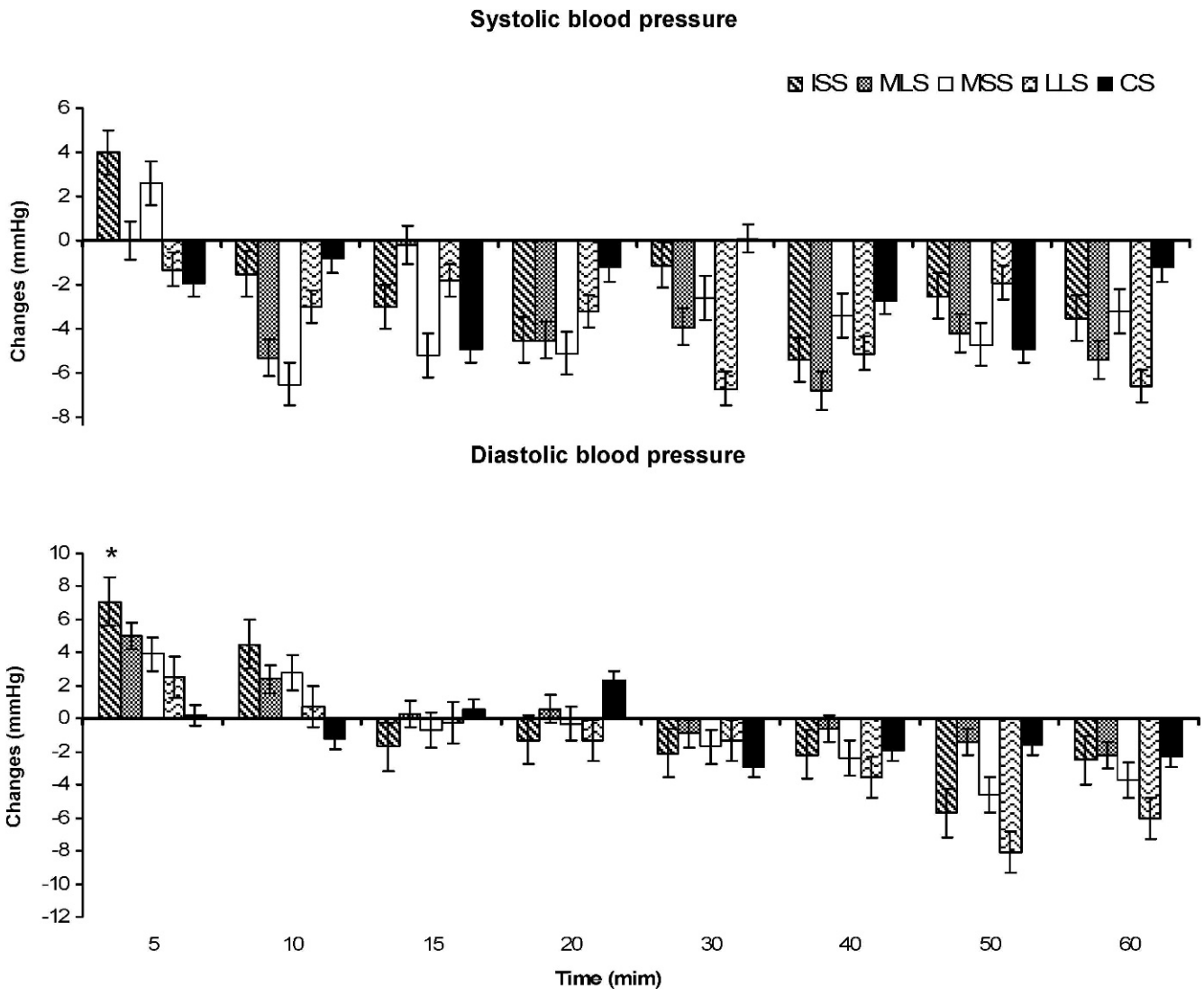

Figure 1 - Post-exercise changes in the systolic and diastolic blood pressures.
ISS = intense short session, MLS = moderate long session, MSS = moderate short session, LLS = light long session and CS = control session.
* = significantly different from the pre-exercise value.

performance, such as the maximum oxygen consumption, muscular strength and endurance, lipid profile and body fat.[29] Thus, the behavior of a given variable after an intervention seems to depend on its value before the intervention. In the present study, the subjects had a reasonable degree of physical fitness (VO_{2peak} = 38 ± 1.26 $ml.kg^{-1}.min^{-1}$), which might have been related to the lower BP values that were observed at rest[30] and the robust cardiovascular adjustments in the rest-exercise and exercise-rest transitions.[31] Thus, several studies that have used a normotensive and physically trained sample have observed PEH inconsistently[7] or for relatively short periods of time.[32]

The majority of the experimental sessions in the present study were conducted in the morning (40 sessions in the morning and 10 sessions in the afternoon), which is a limitation of this study. Within this context, it was recently demonstrated that the magnitude and duration of PEH in normotensive young adults may be linked to the time of day that the exercise session is performed because BP tends to be lower in the morning[33]. These researchers demonstrated that PEH occurred only after the exercise sessions that were performed in the afternoon.

This finding may be related to the fact that the resting BP may be affected by a circadian influence in many individuals; this influence is characterized by a reduction in blood pressure during the night and a gradual rise in blood pressure after awakening.[34] The mechanisms that are responsible for this natural variation in BP may be related to the activation of the sympathetic nervous system.[35]

The indicators of autonomic activity that were used in the present study, the LF_{R-R}^{nu} (which is modulated by the sympathetic and parasympathetic systems), the HF_{R-R}^{nu} (which is typically modulated by the parasympathetic system) and the LF/HF (which provides information about the balance between the sympathetic and parasympathetic systems), did not identify the important and representative differences in post-exercise autonomic modulation. In contrast, the indicator of autonomic activity in the time

Table 4 - Variation in the indicators of autonomic activity.

		Post-Exercise			
	Pre	0-5 min	15-20 min	35-40 min	55-60 min
RMSSD					
ISS	0	-30.7 ± 4.0*	-19.3 ± 3.7*	-9.7 ± 3.0	-3.1 ± 5.0
MLS	0	-23.6 ± 3.2*	-5.1 ± 4.1	2.0 ± 3.2	4.7 ± 2.8
MSS	0	-16.4 ± 3.5*	-2.8 ± 3.8†	6.0 ± 5.1†	13.8 ± 7.0
LLS	0	-9.9 ± 2.5†‡	-2.1 ± 3.3†	4.0 ± 1.8†	9.0 ± 1.9*
CS	0	7.3 ± 3.2†‡§//	-12.4 ± 2.7*†‡	12.56 ± 2.8*†	14.4 ± 3.0*
LF_{R-R}^{nu}					
ISS	0	10.06 ± 4.41	1.65 ± 4.66	5.71 ± 5.33	4.01 ± 3.47
MLS	0	9.54 ± 2.40*	5.32 ± 2.34	1.04 ± 3.02	5.00 ± 1.84
MSS	0	-3.16 ± 5.51	-0.81 ± 5.46	-6.04 ± 5.50	-2.47 ± 8.12
LLS	0	7.50 ± 5.62	3.67 ± 5.68	9.86 ± 5.64	7.01 ± 5.26
CS	0	1.84 ± 3.72	-9.14 ± 4.07	-2.57 ± 2.60	-3.82 ± 3.03
HF_{R-R}^{nu}					
ISS	0	-10.06 ± 4.41	-1.65 ± 4.66	-5.71 ± 5.33	-4.01 ± 3.47
MLS	0	-9.54 ± 2.40*	-5.32 ± 2.34	-1.04 ± 3.02	-5.00 ± 1.84
MSS	0	5.93 ± 5.18	3.59 ± 5.70	8.82 ± 5.48	5.24 ± 8.09
LLS	0	-7.50 ± 5.62	-3.67 ± 5.68	-9.86 ± 5.64	-7.01 ± 5.26
CS	0	-1.84 ± 3.72	9.14 ± 4.07	2.57 ± 2.60	3.82 ± 3.03
LF/HF					
ISS	0	3.13 ± 1.25	0.34 ± 0.61	1.30 ± 0.73	0.19 ± 0.70
MLS	0	2.89 ± 0.98	1.08 ± 0.44	0.33 ± 0.45	0.90 ± 0.41
MSS	0	0.63 ± 1.16	-0.26 ± 0.88	-1.09 ± 0.84	0.00 ± 1.20
LLS	0	0.99 ± 0.83	0.11 ± 0.73	1.07 ± 0.57	0.27 ± 0.63
CS	0	-0.52 ± 0.95	-1.24 ± 1.03	-1.20 ± 0.93	-1.09 ± 0.91

Values described as the mean ± the SE.
ISS = intense short session, MLS = moderate long session, MSS = moderate short session, LLS = light long session, CS = control session, SBP = systolic blood pressure, DBP = diastolic blood pressure, HR = heart rate; RMSSD = the square root of the mean of the sum of the squares of differences between adjacent NN intervals, LF_{R-R}^{nu} = low frequency power in normalized units, HF_{R-R}^{nu} = high frequency power in normalized units and LF/HF = the ratio of LF [ms^2] to HF [ms^2].
* = significantly different from the pre-exercise values; † = significantly different from the ISS; ‡ = significantly different from the MLS; § = significantly different from the MSS; // = significantly different from the LLS.

domain (RMSSD) revealed that the parasympathetic recovery was slower after performing intense and moderate exercise than after performing low-intensity exercise. Moreover, the LF/HF ratio did not change significantly, suggesting that when the parasympathetic recovery is slower for intense exercises, the sympathetic withdrawal tends to be faster.

Future studies should advance the findings of this research by precisely controlling the time of day for the exercise sessions, thereby avoiding to the possible circadian influences. Additionally, a more efficient control of the participants' food intake may contribute to better results.

CONCLUSIONS

The present study did not find any evidence of the PEH phenomenon after four different types of sessions of aerobic exercise. Therefore, we conclude that the reduction in BP to below the resting level that was observed after a single session of exercise in normotensive young adults appears to be linked to other conditions, such as the resting BP. Moreover, the indicators of autonomic activity revealed that for higher intensity exercise, the parasympathetic recovery appears to occur later and that sympathetic withdrawal compensates for this potential delay in recovery.

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