# Tittle: Consistency of hemodynamic and autonomic mechanisms underlying post-exercise hypotension

**Running Tittle: PEH mechanisms consistency**

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# DECLARATIONS


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## Conflict of interest

The authors declare that they have no conflict of interest.

## Ethics Approval

The current data was conducted in accordance with the principles of the Declaration of Helsinki. This study is a part of a bigger study that was approved by the local Ethics Committee (nº 2015/06) and included at Brazilian Clinical Trials register (www.ensaiosclinicos.gov.bR-RBR-3nxn34).

## Consent to participate

All participants provided informed consent before beginning their participation in the study.

**Abstract**

Post-exercise hypotension (PEH) is a clinically relevant phenomenon, but its mechanisms vary between different studies and between the participants within each study. Additionally, it is possible that PEH mechanisms are not consistent in each individual (i.e. within-individual variation), which has not been investigated yet. Thus, the aim of the current study was to assess the within-individual consistency of PEH hemodynamic and autonomic mechanisms. For that, 30 subjects performed 4 sessions divided in 2 blocks (test and retest). In each block, an exercise (cycling, 45min, 50%$VO_2$peak) and a control (seated rest, 45min) session was randomly conducted. Blood pressure (BP) and its mechanisms were evaluated pre and post-interventions. In each block, individual responses were calculated as post-exercise minus post-control, and a response was considered present when its magnitude reached the typical error of the measurement. Consistencies were evaluated by comparing test and retest responses through kappa coefficient (k). PEH consistency was calculated using role sample, while mechanisms consistency was evaluated in those with consistent PEH. Twenty-one (70%) participants showed consistent PEH, 5 (17%) presented PEH in only test or retest and 4 (13%) had absent PEH response, characterizing a good consistency (k= 0.510). Regarding mechanisms' responses, good consistency was found for heart rate (k=0.456), sympathovagal balance (k=0.438) and baroreflex sensitivity (k=0.458); while systemic vascular resistance (k=0.152), cardiac output (k=-0.400), stroke volume (k=-0.055) and sympathetic vasomotor modulation (k=-0.096) presented marginal consistencies. Thus, PEH is a highly consistent physiological phenomenon, although its mechanisms present variable consistencies.

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**Keywords:** "blood pressure"; "aerobic exercise"; "reproducibility"; "within-individual variation".

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**What is know about topic**

- Post-exercise hypotension (PEH) is a well-documented phenomenon with clinical relevance.
- Previous studies have demonstrated discrepant results regarding the hemodynamic and autonomic mechanisms of PEH, which has been related to differences in the characteristics of populations studied and exercise protocols employed.

**What this study adds**

- The divergent results found in PEH literature are also related to the within-individual variation of these responses.
- PEH presents good within-individual consistency, while the consistencies of its mechanisms are good for heart rate increase, sympathovagal balance increase and baroreflex sensitivity decrease, but only marginal for systemic vascular resistance decrease, cardiac output decrease, stroke volume decrease and sympathetic vasomotor modulation decrease.

## Introduction

Post-exercise hypotension (PEH) is characterized by a decrease in blood pressure (BP) observed after a single session of exercise when compared with control values obtained pre-exercise or in a non-exercise day (1). PEH observed after aerobic exercise is accepted as clinically relevant due to its significant magnitude and duration (2). Additionally, it has been suggested as a tool to predict individual responsiveness to BP decrease after an aerobic training period (3,4).

Several studies have focused on PEH mechanisms, but their results are very controversial. Part of the studies have attributed PEH to a systemic vascular resistance (SVR) reduction (5–9) mainly resulting from a decrease in peripheral sympathetic nervous activity (10) and/or responsiveness (i.e. functional sympatholysis) (10) associated with a release of vasodilatory substances (e.g. histamine) (10), both leading to a sustained post-exercise skeletal-muscle vasodilation (11,12). In contrast, other studies reported PEH as determined by a cardiac output (CO) decrease produced by a stroke volume (SV) reduction not offset despite the post-exercise heart rate (HR) increase (13–15) mediated by an augmented cardiac sympathovagal balance (15).

The conflicting results regarding PEH mechanisms have been attributed to differences in the populations and experimental protocols employed in the studies (16). PEH via CO reduction has been mainly reported in overweight, hypertensive and elderly individuals as well as when exercise was conducted in the morning or BP was assessed in the seated position; while the decrease in SVR appears be the main mechanism of PEH in the absence of these specific conditions (16). However, these factors do not fully explain the variation in PEH mechanisms, since within a study employing a specific population and the same experimental protocol for all subjects, 50% of them presented PEH due to a reduction in CO while the other 50% had a decrease in SVR (17).

Another possible factor to explain the divergent results observed in literature and even inside a specific study is an inconsistency of the mechanism of PEH. It is possible that these mechanisms vary from day to day within the same subject even under similar conditions, showing a large within-individual variation. However, to the best of our knowledge, the consistency of PEH mechanisms has not been investigated yet. Along this line, a previous study (18) showed that PEH is reproducible, but the consistency of its mechanisms was not determined. Thus, the current study was designed to assess the within-individual consistency of PEH hemodynamic and autonomic mechanisms, and the hypothesis is that this consistency is low, explaining the large variability observed in literature.

## Methods

### Participants

Participants were included if they fulfilled the following criteria: 1) age between 20-60 years old; 2) absence of cardiovascular (except for hypertension), neurological, respiratory, immunological, renal, endocrine or metabolic (except for diabetes, obesity and dyslipidemia) diseases; 3) absence of resting and exercise electrocardiographic abnormalities suggesting cardiovascular disease; 4) resting systolic (SBP) and diastolic (DBP) BPs below 160 and 105 mmHg, respectively; 5) not taking beta-blockers nor non-dihydropyridine calcium channel blocker; and 6) not having limitations (e.g. orthopedic problems) that restrain exercise execution.

All participants provided written consent to participate. This study is part of a bigger study that was approved by the local Ethics Committee (n$^{o}$ 2015/06), included at Brazilian

Clinical Trials register (www.ensaiosclinicos.gov.bR-RBR-3nxn34). Other findings from the greater study have been previously published (18,19).

**Preliminary evaluation**

To check adherence to the study criteria, participants had three visits to the laboratory. In the first visit, they were interviewed, and anthropometric and clinic BP measurements were obtained. In the second visit, clinic BP was measured again, and in the third visit, the participants underwent a maximal cardiopulmonary exercise test.

Clinical interview obtained information regarding personal data and known health status (i.e presence of the diseases mentioned in the study criteria and current medication treatment). Anthropometric data (body weight and height) were measured (Filizola S.A, Personal, Campo Grande, Brazil), and body mass index was calculated. In each visit for BP assessment, BP was measured in triplicate after 5 min of seated resting using the auscultatory method and a mercury column sphygmomanometer (Unitec, São Paulo, Brazil). Measurements were taken in both arms and SBP and DBP were determined, respectively, by the I and V phases of Korotkoff sounds. The mean of the six measurements (2 visits x 3 measures) of each arm was calculated and the higher mean was registered as the BP level of each participant. The cardiopulmonary maximal test was performed on a cycle ergometer (Lode Medical Technology, Corival, Groningen, Netherlands) employing a protocol with an initial load of 50W followed by increments of 30W every 3 min until exhaustion that was determined as the impossibility to maintain pedalling at 60 rpm. A physician evaluated rest and exercise electrocardiogram (ECG) that was conducted to identify abnormalities suggestive of cardiovascular disease. BP was measured at rest and at the last min of each exercise stage. Oxygen consumption ($VO_2$) was continuously measured (CPX

Ultima, Medical Graphics Corporation) and analysed in means of 30s. Its highest value during exercise was considered as $VO_2$ peak.

**Experimental Protocol**

The participants underwent four experimental sessions, being two exercise and two control sessions performed in a randomized order. Firstly, experimental sessions were divided in two blocks: test and retest. Each block was composed of one exercise and one control session. These blocks were executed successively with sessions being randomized within each block.

For each experimental session, the participants were instructed to: 1) avoid intense physical efforts for the previous 48h; 2) maintain habitual routine for the previous 24h; 3) avoid alcohol consumption for the previous 24h; and 4) avoid smoking and consumption of caffeinated foods or drinks on the session days. The participants who took regular medications were instructed to take them according to the medical prescription, assuring the use at similar times on the session days.

All experimental sessions were conducted by the same experienced evaluator in a temperature-controlled laboratory (20–22$^{o}$C). Each participant performed all the sessions at the same time of day. The sessions were composed of three different periods: 1) pre-intervention; 2) intervention (exercise or control); and 3) post-intervention.

In the pre-intervention period, the participants remained seated for 60 min. ECG, respiratory movements and photoplethysmographic BP were continuously recorded from 10 to 20 min for cardiovascular autonomic evaluation. Then, from 20 to 35 min, auscultatory BP, HR and CO were assessed in this sequence and in triplicates, and the mean value was calculated for hemodynamic evaluation. During the intervention period, the participants followed the specific protocol for each session. In exercise sessions, they exercised for 45 min on a cycle ergometer at

50% of $VO_2$ peak and $VO_2$ was measured from 15 to 35 min of exercise to check the intensity. In the control sessions, they stayed seated on the cycle ergometer for 45 min without pedalling. In the post-intervention period, the participants returned to the seated rest, and autonomic and hemodynamic evaluations were performed, respectively, from 30 to 40 min and 40 to 55 min.

**Measurements**

Auscultatory BP was measured on the dominant arm using the auscultatory method and a mercury column sphygmomanometer (Unitec, São Paulo, Brazil). Mean BP (MBP) was obtained by: MBP = (SBP + 2 DBP) / 3. CO was estimated by the indirect Fick method (20), using the $CO_2$ rebreathing technique and a metabolic cart (CPX Ultima, Medical Graphics Corporation). Briefly, the participants spontaneously breathed ambient air until a steady $CO_2$ production was achieved. At this moment, $VCO_2$ was determined and the arterial content of $CO_2$ ($CaCO_2$) was estimated. Then, the participants performed a $CO_2$ rebreathing manoeuvre with a mixed gas containing a high $CO_2$ concentration (8-10%) and 35% of $O_2$ until an equilibrium was achieved. At this moment, venous content of $CO_2$ ($CvCO_2$) was determined. Thus, CO was estimated by Fick formula: $CO = VCO_2 / (CaCO_2 – CvCO_2)$. SV and SVR were calculated as: SV = CO / HR and SVR = MBP / CO.

For autonomic evaluation, HR was assessed by ECG (Cardioperfect, ST 2001 model, Netherlands), respiratory movements by a thoracic piezoelectric belt (Pneumotrace 2, UFI, Morr Bay, USA) and beat-to-beat BP by photoplethysmography (FMS – Finapress Measurement System, Arnhem, Netherland). These signals were recorded for 10 min using a data acquisition system (Windaq – DI-720, Akron, USA; 500 Hz/channel). Cardiovascular autonomic modulation was evaluated by spectral analysis according to the recommendations of the “Task Force” (21) and using Heart Scope II software (A.M.P.S. LLC, Version 1.3.0.3, New York, USA). The temporal series of R-R intervals, respiration, SBP and DBP were obtained in stationary segments of 250±50 heart beats and were decomposed by the autoregressive method. For interpretation of the results, cardiac sympathovagal balance was considered the ratio between the low- (LF = 0.04-0.15 Hz) and high-frequency (HF = 0.15-0.4 Hz) components of R-R interval variability ($LF/HF_{R-R}$). Sympathetic vasomotor modulation was considered the low-frequency component of SBP variability ($LF_{SBP}$). Baroreflex sensitivity (BRS) was analysed by the maximum magnitude of the transfer function between the R-R interval and the SBP variabilities at the low-frequency band.

Data and statistical analysis

Box-plot graphs were employed to identify extreme values, and Shapiro-Wilk test (SPSS, Illinois, USA) to check the normal data distribution. Non-normal variables were log-transformed (i.e. natural logarithm - ln) to attend analysis of variance (ANOVA)'s statistical assumptions.

Similarity of pre-intervention values among the four experimental sessions were checked by one-way ANOVA for repeated measures. To check whether responses to exercise were in accordance with literature and were similar between the testing blocks, two-way ANOVAs for repeated measures were performed comparing post-intervention values between the sessions (i.e. post-exercise vs. post-control) and the blocks (test and retest). The Newman-Keuls post-hoc test was planned to be applied if necessary.

For consistency analyses, initially, the typical error of measurement (TE) was calculated for each variable (22) using pre-intervention values of the test and retest control sessions (i.e. pre-control test and pre-control retest). These sessions were chosen to avoid any possible influence of an anticipatory response to exercise (i.e. central command activation) on the cardiovascular parameters (23). Afterwards, in each block (test and retest), the individual response to exercise was calculated by the difference in the post-intervention values obtained in the exercise and control sessions (i.e post-exercise – post-control). A change was considered present when the difference was equal to or higher than previously calculated TE (24). Finally, consistency of the response between test and retest was evaluated by Kappa coefficient (k - an agreement index for categorical data: present vs. not-present) and considered as excellent for $k \geq 0.75$, good for k between 0.40 and 0.75, and marginal for $k < 0.40$ (25). In addition, consistency results were also shown by the relative frequencies of consistent response (i.e. response present at both test and retest), inconsistent response (i.e. response present only in test or retest), and consistent absent response (i.e. response not present in either test nor retest). For all analyses, $p \leq 0.05$ was considered as significant.

Based on the main objective of the present study (consistency), the minimum number of subjects required for PEH kappa analysis was calculated using the PASS software (version 19.0.3, NCSS, LCC, Kaysville, USA). Thus, considering a k of 0.60, an alpha error of 5%, a statistical power of 80% and a PEH occurrence rate of 64% (26), the minimum sample size required was 16 subjects. As the consistency of PEH mechanisms could only be evaluated with subjects who show consistent PEH, the sample recruitment aimed to include more subjects. Therefore, after data collection, ANOVA and consistency analyses of BP considered the entire cohort (n=30), while analyses of PEH mechanisms were performed with the 21 participants who presented consistent PEH in MBP. Analyses of $LF_{SBP}$ and BRS included, respectively, 19 and 18 participants due to technical difficulties.

**Results**

Sample characteristics are detailed in Table 1. Participants comprised 24 males (80%) and 6 females (20%). Most of them with overweight (33%) or obesity (50%); with pre (27%) or established hypertension (43%); and not taking medication (73%).

Pre-intervention values of MBP, SVR, CO, HR, SV, $LF/HF_{R-R}$, $LF_{SBP}$ and BRS were similar among the 4 experimental sessions (Table 2). Mean responses to exercise in test and retest are demonstrated in Figure 1. For all variables, responses were similar in the test and retest (no significant interaction in ANOVAs, all $p>0.05$). Additionally, independently of the block (test or retest), MBP, SVR, VS and BRS were significantly lower, while HR and $LF/HF_{R-R}$ were significantly higher after the exercise than the control session (significant session mean effect, all $p<0.05$). Independently of the session (control or exercise), BRS was significantly lower in the test than in the retest. No significance was observed for $LF_{SBP}$.

TEs of all variables are presented in Table 3, and consistency of the responses are shown in Figure 2. For MBP, 21 participants (70.0%) presented PEH in both test and retest, 5 (16.7%) showed PEH only in test or retest, and 4 (13.3%) did not present PEH in neither test nor retest, resulting in a good consistency (k= 0.510, p = 0.005). Regarding the mechanisms, consistency was marginal for SVR (k = 0.152), CO (k = -0.400), SV (k = -0.055) and $LF_{SBP}$ (k = -0.096), and good for HR (k = 0.456), $LF/HF_{R-R}$ (k = 0.438) and BRS (k = 0.458).

**Discussion**

The main findings of the current study are that PEH presented a good within-individual consistency, while the consistencies of its mechanisms were good for HR, $LF/HF_{R-R}$ and BRS, but only marginal for SVR, CO, SV and $LF_{SBP}$.

Based on the mean responses (Figure 1), the proposed exercise was effective in promoting PEH via SVR decrease, as commonly reported in literature (5–9). This response occurred in absence of sympathetic vasomotor modulation changes (no alteration in $LF_{SBP}$), suggesting a sustained vasodilation due to functional sympatholysis (6) and/or local release of vasodilatory substances (27). Additionally, as also reported in literature, the exercise did not change CO since the decrease in SV was compensated by the increase in HR mediated by the higher cardiac sympathovagal balance (higher $LF/HF_{R-R}$) observed in the exercise session. Moreover, the post-exercise tachycardia was not sufficient to abolish PEH, probably due to the reduced cardiac BRS after the exercise. Therefore, the occurrence and mechanisms of PEH observed in the present study are in accordance with previous literature (4,11,12).

As a novelty, for the best of our knowledge, this is the first study to show that mean post-exercise responses are reproducible. All post-exercise hemodynamic and autonomic responses were successfully replicated between the test and retest as all ANOVAs revealed no interaction between session and block factors, indicating similar responses between the repeated tests (25). Nevertheless, although group responses were reproducible, a recent paper about methodological recommendations for PEH studies (28) highlighted that mean group responses do not necessary reflect the individual responses, raising the necessity to examine these responses.

Along this line, the current data (Figure 2) showed a good within-individual consistency of MBP decrease after exercise (k=0.510, i.e. between 0.40 and 0.75) as most of the participants (70%) presented PEH in both blocks (test and retest). This result is in accordance with previous studies that reported consistent BP responses to different physiological stimulus, such as mental stress (29,30) and cold pressor test (30), and expands this consistency to physical stress. Interestingly, differently from MBP response, the consistency of its components, SBP and DBP responses (k=0.379 and k=0.162, data not shown) were only marginal, probably reflecting the marginal consistency of their systemic hemodynamic determinants (i.e. CO and SVR) that are discussed in the next paragraphs.

SVR response after exercise showed a marginal consistency that can be attributed, at least in part, to the inconsistent effect of exercise on peripheral sympathetic modulation assessed by BP variability, as $LF_{SBP}$ response also showed a marginal consistency. In accordance, previous studies also reported inconsistent responses of SVR (29) and muscle sympathetic activity (30) to other physiological stresses (i.e. mental stress and cold pressor test). Additionally, the variable response of SVR after exercise may also reflect an inconsistent effect of previous exercise on other factors, such as local and hormonal vasomotor influences.

Regarding the cardiac responses to exercise, CO also had a high within-individual variation that can be explained by the marginal consistency of SV responses, since SV is one of the CO determinants (31). Although SV determinants are beyond the scope of this study, it is possible to speculate that the inconsistent SV response after exercise might be related to the inconsistent effect of exercise on cardiac afterload as shown by the marginal consistency of SVR response. Alternatively, the inconsistent SV response might also reflect a variable effect of previous exercise on cardiac pre-load, considering its importance in mediating post-exercise SV decrease (32). On the other hand, post-exercise HR response showed good consistent between test and retest, which is in accordance with its responses to other physiological manoeuvres, such as mental stress and head-up tilt (29,30,33), and is coherent with the good consistency observed for cardiac sympathovagal balance (i.e. $LF/HF_{R-R}$). Finally, BRS reduction after exercise also showed a good consistency that might play an important role on the stability of MBP and HR responses to exercise. The change in BRS after exercise is considered essential to allow for the occurrence of PEH, since an unchanged baroreflex function would compensate for BP fall, abolishing PEH (1). Thus, an inconsistent response of BRS would also result in inconsistent responses of BP and HR after the exercise.

Given the exposed, it is possible to speculate that the marginal consistencies of both PEH hemodynamic determinants (i.e. CO and SVR) are related to their interdependence. BP regulation depends on the integrative responses of both cardiac and vascular factors (31). When CO decreases, a compensatory increase on SRV is expected to maintain mean BP at an adequate level for each occasion (31). Based on that, PEH occurrence requires a simultaneous effect of previous exercise on both CO and SRV, decreasing one of them but also blunting the compensatory increase of the other. The current study provided new insight on this topic by demonstrating that PEH is a robust physiological response that occurs consistently after an exercise bout. However, its hemodynamic determinant is inconsistent, and may vary each time the same exercise bout is executed.

Although the current study focused on improving the comprehension about PEH mechanisms, some clinical implications can be proposed. The between-individual cardiovascular responses to different stresses have received emergent interest of the physiologists. Regarding PEH, a previous study (17) reported it was due to a decrease in CO in 50% of the participants and to a reduction in SVR in the other 50% (i.e. between-individual variation), suggesting the possibility to identify the individuals who would present PEH via SVR which may be clinically relevant for hypertensives who usually present an increase in SVR as the cause for BP increase (34). However, the current result put this conjecture in check since it shows that each individual may present PEH by a different hemodynamic mechanism after each bout of the same exercise (high within-individual variation). Thus, future studies should evaluate how experimental design characteristics can be manipulated to attenuate the within-individual variability of PEH hemodynamic and autonomic mechanisms.

Lastly, it is important to mention that the current results are limited to aerobic exercise protocols since the mechanisms (12) of PEH are different after other types of exercise, such as dynamic resistance exercise. Moreover, the marginal consistencies of PEH hemodynamic determinant were mainly observed for SV, CO and SVR that can be influenced by pre-exercise plasma volume and hydration status that were not checked in the current study. Although this lack of control could initially be interpreted as a relevant limitation, it is important to highlight that the current study emulates most of the PEH studies' designs that do not control hydration status, and that a recent study using a similar protocol reported no difference in plasma volume and hydration status before different exercise and control sessions (35). Finally, as a first study on PEH consistency, this study involved a comprehensive sample composed by individuals of both sexes, at different age groups, and with a large variation in BMI and BP status. Within-individual PEH consistency may be different in each of these specific populations, and future studies should address this issue. However, specifically for sex, we performed complementary analyses excluding the women (n=6) and they did not reveal any difference in consistency results for PEH or its mechanisms (data not shown). Additionally, anti-hypertensive medication use (class and dose) may affect PEH magnitude and mechanisms (36,37). However, despite these possible influences on the response to exercise, it is improbable that medication use changes PEH variation between different days (within-individual consistency) if the subjects receive the same dose of medication at the same time of day before the exercise sessions as done in the present study. In additional analyses, consistency results remained the same (data not shown) for all variables when individuals taking anti-hypertensive medication (n=8) were excluded. Future studies, however, may investigated PEH consistency with medication took at different times to evaluate any possible impact.

## Conclusion

PEH is a highly consistent phenomenon that presents low within-individual variation. However, the within-individual consistency of PEH hemodynamic and autonomic mechanisms varies depending on the considered mechanism, with HR, $LF/HF_{R-R}$ and BRS post-exercise responses having good consistencies, while CO, SV, SVR and $LF_{SBP}$ responses present marginal consistency.

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**Conflict of interest**

The authors declare that they have no conflict of interest.

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## Figure legends

**Fig. 1** Mean blood pressure (MBP - panel a), systemic vascular resistance (SVR – panel b), cardiac output (CO – panel c), stroke volume (SV – panel d), heart rate (HR – panel e), natural logarithm of the ratio between low and high frequency components of R-R interval ($lnLF/HF_{R\text{-}R}$ – panel f), low frequency band of systolic blood pressure ($lnLF_{SBP}$ – panel g), and natural logarithm baroreflex sensitivity (lnBRS – panel h) measured after exercise and control sessions conducted in the test and retest blocks of experiments; data are expressed as mean ± standard error; † significantly different from control – session main effect in ANOVA ($p<0.05$). * significantly different from test – block main effect in ANOVA ($p<0.05$)

**Fig. 2** Within-individual consistency in responses of mean blood pressure (MBP - panel a), systemic vascular resistance (SVR – panel b), cardiac output (CO – panel c), stroke volume (SV – panel d), heart rate (HR – panel e), ratio between low and high frequency components of R-R interval ($LF/HF_{R\text{-}R}$ – panel f), low frequency component of systolic blood pressure ($LF_{SBP}$ – panel g), baroreflex sensitivity (BRS – panel h) to exercise evaluated by kappa coefficient (k). * statistically significant ($p<0.05$)


a
p (block) = 0.919
p (session) = <0.001
p (block vs. session) = 0.855
MBP (mmHg)
Control
Exercise
Test
Retest
b
p (block) = 0.474
p (session) = 0.009
p (block vs. session) = 0.510
SVR (U)
c
p (block) = 0.856
p (session) = 0.909
p (block vs. session) = 0.582
CO (L/min)
d
p (block) = 0.522
p (session) = <0.001
p (block vs. session) = 0.527
SV (mL)
e
p (block) = 0.600
p (session) = <0.001
p (block vs. session) = 0.472
HR (bpm)
f
p (block) = 0.449
p (session) = 0.007
p (block vs. session) = 0.669
ln LF/HF R-R
g
p (block) = 0.260
p (session) = 0.160
p (block vs. session) = 0.059
ln LF SBP (mmHg2)
h
p (block) = 0.006
p (session) = 0.014
p (block vs. session) = 0.547
ln BRS (ms/mmHg)


a
Number of participants
21 (70.0%)
k = 0.510
p value = 0.005*
4 (13.3%)
5 (16.7%)
Consistent response
Absent response
Inconsistent response
MBP
b
Number of participants
k = 0.152
p value = 0.466
5 (23.8%)
7 (33.3%)
9 (42.9%)
Consistent response
Absent response
Inconsistent response
SVR
c
Number of participants
k = -0.400
p value = 0.067
0 (0.0%)
9 (42.9%)
12 (57.1%)
Consistent response
Absent response
Inconsistent response
CO
d
Number of participants
k = -0.055
p value = 0.801
4 (19.0%)
6 (28.6%)
11 (52.4%)
Consistent response
Absent response
Inconsistent response
SV
e
Number of participants
k = 0.456
p value = 0.027*
12 (57.1%)
4 (19.0 %)
5 (23.8%)
Consistent response
Absent response
Inconsistent response
HR
f
Number of participants
k = 0.438
p value = 0.031*
9 (42.9%)
6 (28.6%)
6 (28.6%)
Consistent response
Absent response
Inconsistent response
LF/HF R-R
g
Number of participants
k = -0.096
p value = 0.539
0 (0.0 %)
13 (68.4%)
6 (31.6%)
Consistent response
Absent response
Inconsistent response
LF SBP
h
Number of participants
k = 0.458
p value = 0.040*
6 (33.3%)
7 (38.9%)
5 (27.8%)
Consistent response
Absent response
Inconsistent response
BRS

**Table 1.** Characteristics of the participants (n=30; 24 males and 6 females).

| | |
|---|---|
| Age (ys) | 42 ± 11 |
| Height (m) | 1.73 ± 0.06 |
| Weight (kg) | 90.5 ± 18.5 |
| Body Mass Index (kg/m$^2$) | 30.1 ± 5.1 |
| Systolic BP (mmHg) | 123 ± 13 |
| Diastolic BP (mmHg) | 83 ± 11 |
| Mean BP (mmHg) | 97 ± 11 |
| **Blood pressure diagnosis** | |
| Normotensive, n (%) | 9 (30) |
| Pre-hypertensive, n (%) | 8 (27) |
| Hypertensive, n (%) | 13 (43) |
| **Anti-hypertensive Drug therapy** | |
| No medication, n (%) | 22 (73) |
| AT1 receptor blocker, n (%) | 4 (13) |
| Angiotensin-converting enzyme inhibitor, n (%) | 2 (7) |
| Diuretic, n (%) | 1 (3) |
| AT1 receptor blocker + diuretic + dihydropyridine calcium channel blocker, n (%) | 1 (3) |

Continuous values are expressed as mean ± standard deviation. BP = blood pressure. Normotension was defined as systolic and diastolic blood pressure < 130 and 85 mmHg, respectively. Pre-hypertension was defined as systolic and/or diastolic blood pressure between 130-139 and/or 85-89 mmHg, respectively. Hypertension was defined as systolic and/or diastolic blood pressure ≥ 140 and/or 90 mmHg or the use of anti-hypertensive medications.

Table 2. Mean blood pressure (MBP) and its hemodynamic and autonomic mechanisms measured in the pre-intervention periods of the exercise and control sessions of the test and retest evaluations.

| | | TEST | | RETEST | | |
|---|---|---|---|---|---|---|
| | **N** | **Exercise** | **Control** | **Exercise** | **Control** | **p** |
| **MBP (mmHg)** | 30 | 94 ± 12 | 95 ± 11 | 95 ± 11 | 93 ± 10 | 0.647 |
| **CO (mL/min)** | 21 | 4921 ± 1254 | 4837 ± 1111 | 5095 ± 1075 | 4763 ± 936 | 0.269 |
| **SVR (U)** | 21 | 20 ± 5 | 21 ± 5 | 19 ± 4 | 21 ± 5 | 0.138 |
| **SV (mL)** | 21 | 75 ± 22 | 77 ± 20 | 81 ± 23 | 74 ± 19 | 0.164 |
| **HR (bpm)** | 21 | 67 ± 9 | 64 ± 8 | 64 ± 8 | 66 ± 8 | 0.148 |
| **$lnLF/HF_{R-R}$** | 21 | 0.59 ± 1.10 | 0.54 ± 1.04 | 0.55 ± 0.81 | 0.64 ± 1.02 | 0.973 |
| **$lnLF_{SBP}$ ($mmHg^2$)** | 19 | 2.28 ± 0.86 | 2.33 ±1.16 | 2.09 ± 1.04 | 2.39 ±1.05 | 0.649 |
| **lnBRS(ms/mmHg)** | 18 | 1.79 ± 0.54 | 1.76 ± 0.65 | 1.87 ± 0.49 | 1.86 ± 0.49 | 0.717 |

Data are expressed as mean ± standard deviation. CO = cardiac output; SVR = systemic vascular resistance; SV = stroke volume; HR = heart rate; $LF/HF_{R-R}$ = ratio between low and high frequency bands of R-R interval variability; $LF_{SBP}$ = low frequency band of systolic blood pressure; BRS = baroreflex sensitivity; ln = natural logarithm.

**Table 3.** Typical error of measurement calculated using the pre-intervention values of the control sessions conducted in the test and retest blocks expressed in their actual units of measurement (TE) and in percentage of the mean test and retest values (TE%).

| | **TE** | **TE%** |
|---|---|---|
| **MBP (mmHg)** | 2.9 | 3.1 |
| **SVR (U)** | 2.8 | 13.7 |
| **CO (L/min)** | 0.487 | 10.0 |
| **SV (mL)** | 8.3 | 11.1 |
| **HR (bpm)** | 4.3 | 6.4 |
| **ln $LF/HF_{R-R}$** | 0.66 | 94.5 |
| **ln $LF_{SBP}$ ($mmHg^2$)** | 0.64 | 28.2 |
| **ln BRS (ms/mmHg)** | 0.28 | 15.0 |

MBP = mean blood pressure; SVR = systemic vascular resistance; CO = cardiac output; SV = stroke volume; HR = heart rate; ln $LF/HF_{R-R}$ = natural logarithm of the ratio between low and high frequency bands of R-R interval; ln $LF_{SBP}$ = natural logarithm of the low frequency component of systolic blood pressure; ln BRS = natural logarithm of baroreflex sensitivity.