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Fecchio, RY, Brito, LCD, Peçanha, T and Forjaz, CLDM (2020) Post-exercise hypotension and its hemodynamic determinants depend on the calculation approach. *Journal of Human Hypertension*, 34 (10). pp. 719-726. ISSN 0950-9240

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Version: Accepted Version

Publisher: Springer Nature

DOI: <https://doi.org/10.1038/s41371-020-0297-5>

Please cite the published version

<https://e-space.mmu.ac.uk>

1 Title: POST-EXERCISE HYPOTENSION AND ITS HEMODYNAMIC
2 DETERMINANTS DEPEND ON THE CALCULATION APPROACH

3

4 Running title: Post-exercise hypotension calculation approaches

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1 **Abstract**

2 Post-exercise hypotension (PEH) has been assessed by three calculation approaches: I=
3 (post-exercise – pre-exercise); II = (post-exercise – post-control); and III = [(post-exercise
4 – pre-exercise) – (post-control – pre-control)]. This study checked whether these
5 calculation approaches influences PEH and its determinants. For that, 30 subjects
6 underwent two exercise (cycling, 45min, 50%VO₂peak) and two control (seated rest,
7 45min) sessions. Systolic (SBP) and diastolic (DBP) blood pressures, cardiac output (CO),
8 systemic vascular resistance (SVR), heart rate (HR) and stroke volume (SV) were measured
9 pre and post-interventions in each session. The mean value for each moment in each type of
10 session was calculated, and responses to exercise were analyzed with each approach (I, II
11 and III) to evaluate the occurrence of PEH and its determinants. Systolic PEH was
12 significant when calculated by all approaches (I = -5 ± 1 , II = -11 ± 2 and III = -11 ± 2 mmHg,
13 $P<0.05$), while diastolic PEH was only significant when calculated by approaches II and III
14 (-6 ± 1 and -6 ± 1 mmHg, respectively, $P<0.05$). CO decreased significantly after the exercise
15 when calculated by approach I, but remained unchanged with approaches II and III; while
16 SVR increased significantly with approach I, but decreased significantly with approaches II
17 and III. HR was unchanged after the exercise with approach I, but increased significantly
18 with approaches II and III, while SV decreased significantly with all approaches. Thus,
19 PEH and its hemodynamic determinants are influenced by the calculation approach, which
20 should be considered when designing, analyzing and comparing PEH studies.

21

22

1 Summary Table

What is known about topic

- Post-exercise hypotension (PEH) is characterized by a reduction in blood pressure after a single session of exercise.
- Previous studies have demonstrated diverse results regarding the occurrence, magnitude and subjacent hemodynamic determinants of PEH, which has been attributed to differences in populations studied and exercise protocols employed.

What this study adds

- The diverse results obtained by previous studies can also be related to the different approaches used to calculate PEH and its determinants.
- When designing studies, analyzing data and interpreting results about PEH and its determinants, it is essential to take into account the calculation approach employed.

1 INTRODUCTION

2 Post-exercise hypotension (PEH) is characterized by a reduction in systolic (SBP)
3 and/or diastolic (DBP) blood pressure (BP) after a single session of exercise(1). PEH is
4 accepted as clinically relevant due to its significant magnitude (mean decrease of 5/3
5 mmHg for SBP/DBP, respectively)(2) and long lasting duration (i.e. up to 16/12 hours for
6 SBP/DBP, respectively)(3), which may benefit individuals with high BP(4). Moreover,
7 PEH presents a strong and positive association with chronic BP reduction after exercise
8 training(5,6), which suggests its use as a tool to predict BP responsiveness to training.

9 PEH has been reported after different aerobic exercise protocols and in diverse
10 populations (normotensives, pre-hypertensives and hypertensives)(2); however its
11 magnitude varies a lot among the studies(4). In addition, some studies did not report the
12 occurrence of PEH(7,8). This large variation in PEH results is also observed regarding its
13 hemodynamic determinants, since both a reduction in cardiac output (CO)(9–11) or in
14 systemic vascular resistance (SVR)(7,12–15) have been reported in literature. Although this
15 inconsistency has been attributed to differences in the characteristics of population and
16 exercise protocols employed in the studies(16), the approach to calculate PEH may also
17 influence such outcomes.

18 In many studies(5–7,9,17–21), PEH has been calculated simply by the difference
19 between post- and pre-exercise BPs (I: post-exercise BP – pre-exercise BP). Other
20 studies(8,10,13,14,22–24) have compared post-exercise BP with BP measured after a
21 control session without exercise (II: post-exercise BP – post-control BP). Lastly, some
22 studies(11,12,15) have employed a more complex approach, calculating the “net effect” of
23 exercise as the difference between the BP response to an exercise session and the BP

1 response to a control session [III: (post-exercise BP – pre-exercise BP) – (post-control BP –
2 pre-control BP)]. Nevertheless, to the best of our knowledge, no study has directly
3 examined the influence of the calculation approach on the interpretation about the
4 occurrence of PEH and its hemodynamic determinants. This possible influence is supported
5 by the fact that approach I does not control for time changes in BP, and approach II does
6 not consider day-to-day variations in BP.

7 Based on previous background, the current study aimed to investigate whether the
8 occurrence and magnitude of PEH as well as its hemodynamic determinants are influenced
9 by the calculation approach, employing the three above-mentioned approaches (I, II and
10 III).

11

12 **METHODS**

13 **Participants**

14 Male and female subjects, aged between 20 and 60 years old were invited to
15 participate in this study. The exclusion criteria were: 1) diagnosis of any cardiovascular
16 disease, except for hypertension; 2) presence of electrocardiographic abnormalities that
17 suggest cardiovascular disease; 3) resting SBP or DBP higher than 160 and 105 mmHg,
18 respectively; 4) use of medication that directly affects the autonomic nervous system; and
19 5) presence of any health problem that restrains exercise execution.

20 The subjects who fulfilled the study criteria signed an informed written consent to
21 participate. This study was approved by the local Ethics Committee (CAAE
22 43759215.3.0000.5391) and registered at the Brazilian Clinical Trials
23 (www.ensaiosclinicos.gov.br-RBR-3n34).

1

2 **Preliminary evaluation**

3 To confirm the absence of any exclusion criteria, subjects underwent three visits to
4 the laboratory in different days. In the first visit, they were interviewed, and anthropometric
5 and resting BP measurements were performed. In the second visit, resting BP was
6 measured again, and in the third visit, a maximal cardiopulmonary exercise test was
7 conducted. The interview included questions regarding personal data, health condition,
8 familiar health history, current medication treatment and physical activity practice.
9 Anthropometric data consisted of body mass (kg) and height (m) measures (Filizola S.A,
10 Personal, Campo Grande, Brazil), and the calculation of body mass index (BMI).
11 Auscultatory BP was evaluated in triplicate in each visit. Measures were taken after 5 min
12 of sitting rest with a mercury sphygmomanometer (Uniteq, São Paulo, Brazil). SBP and
13 DBP were, respectively, determined by phases I and V of the Korotkoff sounds. The mean
14 of the six measures was used to define the resting BP level of each subject(25). The
15 maximal cardiopulmonary exercise test was performed on a cycle ergometer (Lode Medical
16 Technology, Corival, Groningen, Netherlands) with an initial load of 30 W and increments
17 of 30 W every 3 min until subjects were unable to proceed. A physician evaluated resting
18 and exercise ECG as well as HR and BP responses. Peak oxygen uptake (VO_{2peak}) was
19 defined as the highest value obtained during the test, in averages of 30 s

20

21 **Experimental protocol**

22 After the preliminary procedures, the experimental protocol was started. All
23 subjects underwent two exercise and two control sessions, with an interval of at least two

1 days between them. The order of sessions' execution was randomized. For that,
2 experimental sessions were divided in two blocks, each one being composed by one
3 exercise and one control session. These blocks were performed successively and sessions
4 were randomized within each block. Duplication of each session (exercise and control) was
5 done to improve the precision of measures. Thus, the mean value of the two repeated
6 sessions was considered for analyses.

7 Before all the experimental sessions, the subjects were instructed to keep similar
8 and habitual routines in the previous 24h. They were also instructed to avoid: exercise
9 execution for the previous 48h; alcoholic drinks for the previous 24h; and smoking and the
10 ingestion of foods or drinks that might affect cardiovascular function on days of the
11 sessions. In addition, they were instructed to have a light meal at least two hours before the
12 sessions. The subjects in use of regular medications were instructed to take their
13 medications as prescribed by the physician and at similar times before the experimental
14 sessions.

15 Each subject performed all the experimental sessions in the same time of day.
16 Laboratory temperature was controlled and kept between 20 and 22°C. Each session was
17 composed by pre-intervention, intervention (exercise or control) and post-intervention
18 periods. Assessments were taken during the pre- and post-intervention periods with the
19 subjects resting in sitting position. The pre-intervention assessment started after 20 min of
20 rest, while the post-intervention assessment was taken 45 min after the end of the
21 intervention. In each assessment, auscultatory BP, heart rate (HR) and CO were measured
22 in this sequence. This sequence of measurements was repeated three times with an interval
23 of 3 min between them. The average of the three measurements obtained in each

1 assessment period was calculated for each variable.

2

3 Exercise and control interventions

4 In the exercise sessions, during the intervention period, the subjects exercised on a
5 cycle ergometer for 45 min at 50% of VO_2 peak. The workload necessary to reach this
6 intensity was calculated by the linear regression between workload and VO_2 obtained
7 during the cardiopulmonary exercise test. In addition, exercise intensity was checked by the
8 direct measurement of VO_2 from 15 to 35 min of the exercise. In the control sessions, the
9 subjects remained seated on the cycle ergometer for the same amount of time of exercise
10 sessions, but without performing any effort.

11

12 Measurements

13 BP was measured by the auscultatory method on the dominant arm, using the
14 technique described in the preliminary procedures, and with all measurements taken by the
15 same trained evaluator. Mean BP (MBP) was calculated through the following formula:
16 $(SBP + 2 \times DBP) / 3$. CO was estimated by the indirect method of Fick(26), through the
17 maneuver of CO_2 rebreathing(27), using a gas analyzer (Medical Graphics Corporation
18 CPX/Ultima, Minnesota, USA). HR was measured by radial pulse palpation. SVR was
19 calculated by the quotient between MBP and CO, and stroke volume (SV) by the quotient
20 between CO and HR.

21

22 **Statistical analysis**

23 Considering a statistical power of 90%, an alpha error of 5% and standard

1 deviations of 3 mmHg for SBP and 0.32 l/min for CO, the minimal sample size required
2 was 10 subjects for SBP and 11 subjects for CO in order to detect worthwhile effects of 4
3 mmHg and 0.32 l/min, respectively(28).

4 Prior to data analyses, the mean value for each moment (pre and post) in each type
5 of session (control and exercise) was calculated. Data normality was checked through
6 Shapiro-Wilk test (SPSS, Illinois, USA).

7 First, to confirm similarity in pre-intervention values between the sessions, pre-
8 exercise values were compared with pre-control values by paired *t*-tests. Then, the
9 occurrence of PEH and its hemodynamic determinants were determined by each approach:
10 Approach I - post-exercise values were compared with pre-exercise values; Approach II -
11 post-exercise values were compared with post-control values; and Approach III, the net
12 effects of exercise [(post-exercise values – pre-exercise values) – (post-control values –
13 pre-control values)] were compared with zero. All these comparisons were done by paired *t*
14 tests. Finally, the magnitudes of the responses obtained with each approach were compared
15 using one-way ANOVA for repeated measures and Newman-Keuls post hoc test when
16 necessary. Data was presented as mean ± SE and $p < 0.05$ was set as significant.

17

18 **RESULTS**

19 Thirty-eight subjects were assessed for eligibility, but 4 did not accept to participate
20 in the study. From the remaining 34 subjects, one was excluded during the preliminary
21 evaluation due to ECG abnormalities and another due to orthopedic limitation. Thus, 32
22 subjects started the experimental protocol, but two dropped out due to personal reasons.
23 Therefore, the final sample was composed by 30 subjects (24 men and 6 women) whose

1 characteristics are shown in Table 1.

2 Pre-intervention SBP, DBP, CO, SVR, SV and HR were similar between the
3 exercise and control sessions (Table 2).

4 PEH evaluated by the three calculation approaches are shown in Figure 1. Systolic
5 PEH was significant when evaluated by approaches I (post-exercise: 115 ± 2 vs. pre-
6 exercise: 120 ± 2 mmHg, $p < 0.001$), II (post-exercise: 115 ± 2 vs. post-control: 126 ± 2 mmHg,
7 $p < 0.001$) and III (net effect: -11 ± 2 mmHg vs. zero, $p < 0.001$). Diastolic PEH was not
8 significant with approach I (post-exercise: 82 ± 2 vs. pre-exercise: 82 ± 2 mmHg, $p = 0.186$),
9 but was significant when assessed by approaches II (post-exercise: 82 ± 2 vs. post-control:
10 88 ± 2 mmHg, $p < 0.001$) and III (net effect: -6 ± 1 mmHg vs. zero, $p < 0.001$).

11 Hemodynamic determinants of PEH evaluated by the three methods of calculation
12 are shown in Figure 2. CO decreased significantly after the exercise when analyzed by
13 calculation approach I (post-exercise: 4.43 ± 0.17 vs. pre-exercise: 5.05 ± 0.21 l/min,
14 $p < 0.001$), while remained unchanged when analyzed by approaches II (post-exercise:
15 4.43 ± 0.17 vs. post-control: 4.40 ± 0.18 l/min, $p = 0.808$) and III (net effect: -0.14 ± 0.13 l/min
16 vs. zero, $p = 0.314$). SVR increased significantly after the exercise with approach I (post-
17 exercise: 22.0 ± 0.8 vs. pre-exercise: 19.7 ± 0.7 U, $p < 0.001$), but decreased significantly with
18 approaches II (post-exercise: 22.0 ± 0.8 vs. post-control: 24.2 ± 1.1 U, $p = 0.003$) and III (net
19 effect: -1.5 ± 0.7 U vs. zero, $p = 0.033$). HR remained unchanged with approach I (post-
20 exercise: 68 ± 1 vs. pre-exercise: 66 ± 1 bpm, $p = 0.156$), but increased significantly when
21 analyzed by approaches II (post-exercise: 68 ± 1 vs. post-control: 61 ± 2 bpm, $p < 0.001$) and
22 III (net effect: $+7 \pm 1$ bpm vs. zero, $p < 0.001$). For SV, a significant decrease was found with
23 all approaches (I = post-exercise: 66 ± 3 vs. pre-exercise: 77 ± 4 ml, $p < 0.001$; II = post-

1 exercise: 66 ± 3 vs. post-control: 73 ± 3 ml, $p=0.001$; and III = net effect: -10 ± 3 ml vs. zero,
2 $p<0.001$).

3 When the magnitudes of changes were compared among the three calculation
4 approaches significant differences were observed for all variables ($p\leq 0.05$), except for SV,
5 with approach I providing results significant different from approaches II and III that in
6 turn produced similar results (Table 3).

7

8 **DISCUSSION**

9 The main finding of this study is that the interpretation of results about the
10 occurrence of PEH as well as its subjacent hemodynamic determinants varied according to
11 the calculation approach employed.

12 In the current study, systolic PEH occurred regardless of the approach (I, II and III),
13 but the magnitudes of SBP decrease were different among the approaches, with calculations
14 II and III revealing greater PEH than I. Along this line, diastolic PEH was significant when
15 calculated by approaches II and III, but not by approach I. These divergences may have
16 occurred due to BP changes in the control session. Actually, SBP and DBP increased from
17 pre to post-intervention in the control sessions (SBP: 119 ± 2 vs 126 ± 2 mmHg, $P=<0.001$;
18 and DBP: 82 ± 2 vs 88 ± 2 , $p<0.001$; data not shown). The increase in BP after a control
19 condition has been already reported(11,12), and has been attributed to the circadian
20 variation of BP when experiments were conducted in the morning(29) and/or to a response
21 to the orthostatic stress imposed by the sitting position(30). Without considering the
22 responses to a control session, the effect of exercise blunting the increase in SBP and DBP
23 would not be taken into account. It is also important to consider that under different

1 experimental conditions, BP might change in a different ways after a control period. For
2 example, BP might decrease during a control session performed in evening(11). In that
3 case, a decrease in BP observed with approach I (post-pre-exercise) could reflect the effect
4 of the exercise, the circadian behavior or the additive effect of both.

5 The current study also showed that the calculation approach may influence the
6 interpretation of the systemic hemodynamic determinant of PEH. In the present study,
7 using approach I, BP decrease after the exercise would be attributed to a decrease in CO,
8 while with approaches II and III, a decrease of SVR would be the underlying determinant.
9 Actually, CO decreased after exercise in comparison with pre-exercise, but it was similar to
10 post-control values because CO also decreased in the control session (4.89 ± 0.20 vs
11 4.40 ± 0.18 ml/min, $p<0.001$, data not shown). Similarly, post-exercise SVR increased when
12 compared to pre-exercise values, but it was lower than post-control values, because SVR
13 increased more in the control session from pre-control to post-control (20 ± 5 vs 24 ± 6 ,
14 $p<0.001$; data not shown). In accordance with the present data, previous studies(11,12,31)
15 have also reported a decrease in CO and an increase in SVR during a control session
16 performed in the sitting position, which might be explained by the orthostatic stress
17 promoted by this position that decreases venous return and deactivates the cardiopulmonary
18 reflex(30). Consequently, in the present study, when time effects were considered (i.e.
19 employing a control situation), previous exercise promoted PEH by a SVR decrease.

20 The current results support that different approaches to evaluated PEH may lead to
21 conflicting results, demonstrating the importance of choosing an adequate method for
22 calculating PEH in accordance to the study objectives and, specially, to interpret study's
23 results in accordance with the approach employed. The assessment of PEH through

1 approach I has limitations related to the absence of controlling the time influence, which
2 has been emphasized in the Consolidated Standards of Reporting Trials (CONSORT)(32).
3 Thus, when calculation approach I is employed the real effect of the previous exercise on
4 post-exercise response might be inadequately assessed. This approach might be useful to
5 compare the post-effect of different exercise protocols, but it is not adequate to establish the
6 occurrence, magnitude and determinants of PEH. Time influence is controlled with
7 approaches II and III.

8 Interestingly, in the current study, approaches II and III provided similar results
9 which can be explained by the fact that pre-intervention values were similar in the exercise
10 and control sessions (Table 2). However, it is important to mention that a difference in pre-
11 intervention values can happen due to day-to-day BP variability(33) or to an effect of a
12 previous intervention (e.g. comparing PEH before and after a period of training that
13 changed baseline BP). Differences in pre-intervention values would introduce an important
14 bias to approach II, preventing to attribute post-exercise responses to exercise per se, since
15 they can just reflect the pre-intervention differences. Approach III overcame this limitation.

16 Despite approaches II and III are more robust to assess PEH than approach I,
17 important aspects involving the application of PEH should be mentioned. The use of PEH
18 to identify individuals with greater responsiveness to training has been only explored with
19 approach I(5,6). However, it is possible that employing approaches II and III would
20 improve this prediction, which needs to be investigated. Other aspect that deserves further
21 elucidation is which approach provides results with greater clinical relevance. It is not clear
22 the clinical difference of decreasing BP after exercise in relation to pre-exercise (approach
23 I) or to a control condition (approaches II and III).

1 The current study is not without limitations. The sample was composed by subjects
2 with different characteristics (different BP status, taking or not-taking anti-hypertensive
3 medications, a wide age range and both genders) to generate a comprehensive sample that
4 produces results not restrained to a specific population. The study demonstrated that
5 calculation approach can influence the interpretation about PEH occurrence, magnitude and
6 determinants. However, the specific influence in specific populations and study conditions
7 should be addressed by future research.

8 In conclusion, the current study empirically demonstrated that different approaches
9 to calculate PEH may lead to conflicting interpretations regarding its occurrence,
10 magnitude and hemodynamic determinants. Therefore, study design, data analyses and
11 interpretation of results about PEH and its determinants must take into account the
12 calculation approach employed. Future studies should explore the clinical impact of the
13 results obtained with each one of these different calculation approaches, since they provide
14 different outcomes.

15

16 **Acknowledgements**

17 The authors want to acknowledge the volunteers of the current study. This study
18 received financial support from CNPq and CAPES (0001).

19

20 **Conflict of interest**

21 The authors declare no conflict of interest.

22

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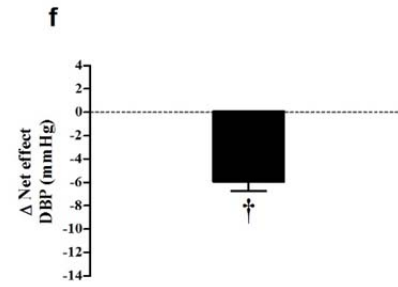
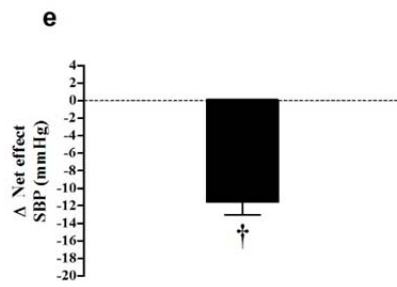
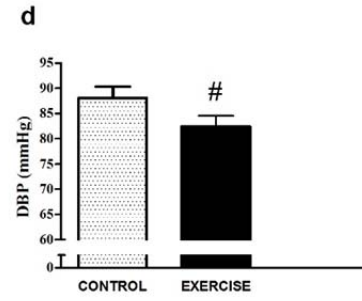
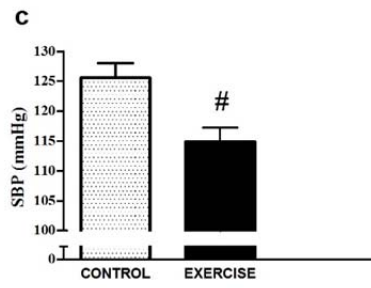
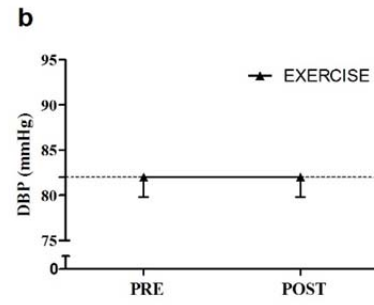
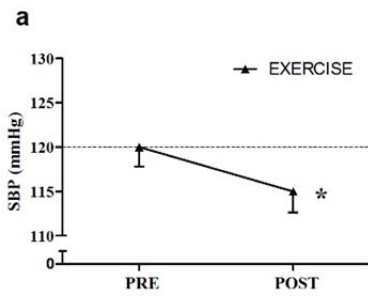
1 **Figure legends**

2 **Figure 1.** Systolic (SBP) and diastolic blood pressures (DBP) responses to exercise
3 calculated by the following three approaches: I: post-exercise vs. pre-exercise (**panel a and**
4 **b**); II: post-exercise vs. post-control (**panel c and d**); and III: [(post-exercise – pre-
5 exercise) – (post-control – pre-control)] vs. zero (**panel e and f**). *Post-exercise
6 significantly different from pre-exercise ($p < 0.05$). #Post-exercise significantly different
7 from post-control ($p < 0.05$). †Net effect significantly different from zero ($p < 0.05$). Values
8 are mean \pm SE.

9

10 **Figure 2.** Cardiac output (CO), systemic vascular resistance (SVR), heart rate (HR) and
11 stroke volume (SV) responses to exercise calculated by the following three approaches: I:
12 post-exercise vs. pre-exercise (**panel a and b**); II: post-exercise vs. post-control (**panel c**
13 **and d**); and III: [(post-exercise – pre-exercise) – (post-control – pre-control)] vs. zero
14 (**panel e and f**). *Post-exercise significantly different from pre-exercise ($p < 0.05$). #Post-
15 exercise significantly different from post-control ($p < 0.05$). †Net effect significantly
16 different from zero ($p < 0.05$). Values are mean \pm SE.

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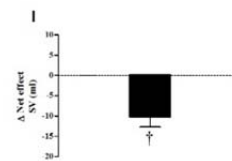
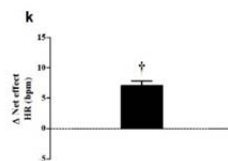
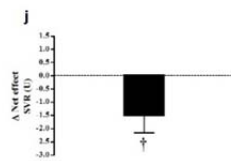
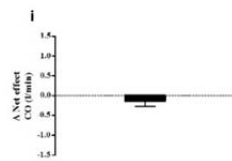
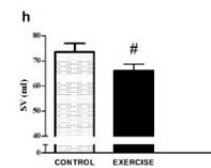
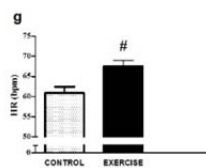
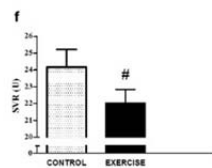
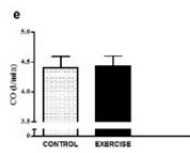
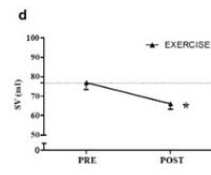
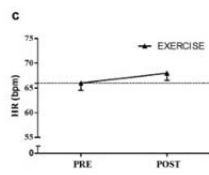
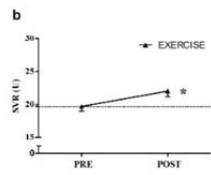
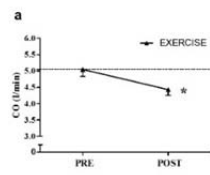


Table 1. Characteristics of the subjects (n=24 males and 6 females).

Characteristic	Value
Age (ys)	42 ± 2
Height (m)	1.73 ± 0.01
Weight (kg)	90.5 ± 3.4
Body Mass Index (kg/m ²)	30.1 ± 0.9
Systolic BP (mmHg)	123 ± 2
Diastolic BP (mmHg)	83 ± 2
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 (%)	5 (17)
Angiotensin-converting enzyme inhibitor, n (%)	2 (7)
Diuretic, n (%)	2 (7)
Dihydropyridine calcium channel blocker, n (%)	1 (3)
Treatment Strategy	
Not using anti-hypertensive medication	5 (17)
Monotherapy	7 (23)
Polytherapy	1 (3)

Continuous values are expressed as mean ± SE. 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 Blood pressure and its hemodynamic determinants measured in the pre-intervention periods of the exercise and control sessions (mean value of the two sessions of

	Exercise	Control	P value
SBP (mmHg)	120 ± 2	119 ± 2	0.469
DBP (mmHg)	82 ± 2	82 ± 2	0.751
CO (l/min)	5.05 ± 0.21	4.89 ± 0.20	0.219
SVR (U)	19.7 ± 0.7	20.3 ± 0.9	0.198
HR (bpm)	66 ± 1	67 ± 2	0.671
SV (ml)	77 ± 4	74 ± 3	0.222

the same type – exercise or control).

Values are mean ± SE; P value = significance levels in paired t-test; SBP = systolic blood pressure; DBP = diastolic blood pressure; CO = cardiac output; SVR = systemic vascular resistance; HR = heart rate; SV = stroke volume.

Table 3. Magnitudes of changes of blood pressure and its hemodynamic determinants after the exercise calculated by three different calculation approaches: I = post-exercise – pre-exercise; II = post-exercise – post-control; and III = (post-exercise – pre-exercise) – (post-control – pre-control).

Variable	I	II	III	P-value
SBP (mmHg)	-5 ± 1	-11 ± 2†	-11 ± 2†	< 0.01
DBP (mmHg)	1 ± 0	-6 ± 1†	-6 ± 1†	< 0.01
CO (l/min)	-0.62 ± 0.12	0.03 ± 0.11†	-0.14 ± 0.13†	< 0.01
SVR (U)	2.3 ± 0.6	-2.2 ± 0.7†	-1.5 ± 0.7†	< 0.01
HR (bpm)	1 ± 1	7 ± 1†	7 ± 1†	< 0.01
SV (ml)	-11 ± 2	-7 ± 2	-10 ± 3	0.180

Values are mean ± SE. P value = significance levels in one-way ANOVA. SBP = systolic blood pressure; DBP = diastolic blood pressure; CO = cardiac output; SVR = systemic vascular resistance; HR = heart rate; SV = stroke volume. †Significantly different (p < 0.05) from approach I.