Recommendations in Post-exercise Hypotension: Concerns, Best Practices and Interpretation

Authors
Leandro Campos de Brito[1], Rafael Yokoyama Fecchio[1], Tiago Peçanha[2], Aluisio Lima[1], John Halliwill[3], Claudia Lucia de Moraes Forjaz[1]

Affiliations
1 Exercise Hemodynamic Laboratory, School of Physical Education and Sport, University of Sao Paulo, Sao Paulo, Brazil
2 Applied Physiology & Nutrition Research Group; Laboratory of Assessment and Conditioning in Rheumatology, Clinical Hospital HCFMUSP, Faculty of Medicine, University of Sao Paulo, Sao Paulo, Brazil
3 Department of Human Physiology, University of Oregon, Eugene, United States

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Correspondence
Mr. Leandro Campos de Brito
University of São Paulo, School of Physical Education and Sport,
Av. Prof. Mello Moraes, 65 - Cidade Universitária,
05508–030 São Paulo,
Brazil
Tel.: + 55/11/30918 792, Fax: + 55/11/30918 792
leandrobrito@usp.br

ABSTRACT

Post-exercise hypotension (PEH) is a clinically relevant phenomenon that has been widely investigated. However, the characteristics of study designs, such as familiarization to blood pressure measurements, duration of PEH assessments or strategies to analyze PEH present discrepancies across studies. Thus identifying key points to standardize across PEH studies is necessary to help researchers to build stronger study designs, to facilitate comparisons across studies, and to avoid misinterpretations of results. The goal of this narrative review of methods used in PEH studies was therefore to gather and find possible influencers in the characteristics of study design and strategies to analyze blood pressure. Data found in this review suggest that PEH studies should have at least two familiarization screening visits, and should assess blood pressure for at least 20 min, but preferably for 120 min, during recovery from exercise. Another important aspect is the strategy to analyze PEH, which may lead to different interpretations. This information should guide a priori study design decisions.

Introduction

Many research groups have been investigating post-exercise hypotension (PEH), which is characterized as a reduction in blood pressure below the values observed either immediately prior to exercise or on a control day (e. g., same conditions but without exercise) [1]. Reasons to study this phenomenon include efforts to understand the capacity of an exercise session to predict the chronic effect and/or to understand the cardiovascular regulation during this window of opportunity [2–4]. Recent meta-analyses have demonstrated that PEH occurs with sufficient magnitude and duration to be clinically relevant [5, 6]. Indeed, PEH has been observed for 16 h after an exercise session [7], and the average blood pressure value for 24-hours post-exercise is lower compared to the same period after a control session [8–10]. However, the magnitude and duration of this response present with significant variability across studies [5, 6], which might be attributable to differing methodological approaches for reporting PEH within these studies.

The existing literature on PEH is full of a wide range of varied protocols, methods and analyses. Common variations include the number of familiarization measurements prior to the actual study; whether subjects are studied supine or seated; employment of different types of blood pressure monitors; and in clinic/lab (clinical) versus ambulatory monitoring. Protocols often vary for the type, duration, and intensity of exercise, and for the timing of post-exercise measurements. In addition, different comparisons are used for data analysis and interpretation: pre vs. post-exercise, exercise session vs. control session, or net effect comparisons of exercise vs. control session. All these conditions must be taken into account for the study design and interpretation of data in studies about PEH. If these characteristics of PEH studies create difficulty for comparisons across studies, then perhaps recommendations about methodological concerns might help to standardize the study of PEH and increase the validity of comparisons. In this narrative review, these aspects are discussed comprehensively.

Development

Concerns and approaches: from design to interpretation

Due to the exciting prospects for the clinical relevancy of PEH and the need for more research on this phenomenon, we were compelled to explore issues related to experimental design, methodology, and data collection, analysis and interpretation of the PEH studies that should be carefully considered to promote scientific rigor and high reproducibility on this topic. As the aim of this review was to stimulate a comprehensive discussion about methodological concerns in PEH studies, a narrative model was more appropriate, and was therefore selected [11]. For this narrative review, the literature search was conducted in the PubMed database using the term "post-exercise hypotension," as well as in reference lists of systematic reviews about this topic and reference lists of the PEH studies. Moreover, this study followed all ethical standards [12].

Concerns with blood pressure measurements

An important aspect regarding any study of blood pressure measures is the fact that subjects should be sufficiently familiarized with the lab environment and blood pressure measurement procedures before the beginning of the experimental protocol.

Given the variability in blood pressure [13–15], clinical guidelines for assessing hypertension recommend the measurement of blood pressure on two or more visits to determine resting blood pressure, and during each visit blood pressure should be measured at least twice [16–18]. This is based on studies comparing blood pressure changes between successive visits when blood pressure decreases substantially from the first to the second visit [19–21], regardless of the interval between the visits (3 days or 14 days) or the initial level of blood pressure (normotensives or hypertensives) [21]. Values obtained on a third visit were similar to those observed on a second visit [21], highlighting the importance of screening visits that include the measurement of blood pressure to familiarize subjects. So that, a stable resting blood pressure can be recorded in the experimental protocol, allowing for greater rigor and reproducibility in the measure of PEH.Despite the importance of familiarization visits to stabilize resting blood pressure in PEH investigations, some studies have relied on only one screening visit [9, 22–47] or none at all [48–52], with blood pressure measurements before the experiments, and many studies simply have not reported whether any blood pressure screening or familiarization was performed [53–69] (▶ **Table 1**). While study designs that incorporate both exercise and control session may mitigate this concern by showing no difference in pre-exercise blood pressure between the sessions, studies with a simple pre- versus post-exercise design (i. e., without a control session and/or one comparison session of exercise) are highly susceptible to this limitation [24, 29, 47, 49].

Since post-exercise blood pressure reduction is influenced by its pre-exercise value [5, 70], an "unreal" elevated pre-exercise value, based on a lack of adequate familiarization, may inflate PEH. As a result, future studies where PEH is the main outcome should include, at least two familiarization visits during which blood pressure is measured in accordance with reliability and familiarization studies [13–15]. Researchers may then have more confidence in resting blood pressure obtained during subsequent experimental sessions.

Timing of post-exercise measurements

The timing of blood pressure measurements after exercise may be one of the main causes for divergent results, impacting the magnitude and determinants of PEH across studies. The variability of experimental designs ranges from evaluating PEH during the first 5 min [67] to first 3 hrs [22] post-exercise, and extends to 24-h ambulatory blood pressure (ABPM) measurements after exercise. In fact, this variability has made it difficult to determine at what time after exercise the greatest PEH occurs and has introduced debate as to how long it lasts.

The magnitude of clinical PEH is important due to its strong correlation with the chronic hypotensive effect of exercise [39, 71, 72], allowing for the detection of responders and non-responders to training. Among 37 studies that analyzed blood pressure in the laboratory setting for at least 20 min after exercise [22, 24–26, 28, 29, 35, 36, 42, 43, 45, 47, 50–56, 62, 64–68, 71, 73–83], 32 studies reported no decrease in blood pressure until that time (i. e., 20 min) [25, 26, 29, 35, 36, 42–44, 47, 50–56, 58, 62, 64–68, 71, 73–77, 79–81], and the other 5 reported PEH was only at 15 min post-exercise [22, 28, 78, 82, 83]. Among 31 studies that followed blood pressure throughout the recovery period for at least 60 min [22, 24–26, 28–30, 42, 43, 47, 50, 51, 53, 55, 58, 62, 66, 73, 76–88], 19 studies reported the greatest blood pressure decrease after 30 min of recovery period [22, 24, 28, 29, 42, 47, 50, 53, 58, 62, 76–78, 80, 81, 83, 84, 87, 88], while 9 studies reported the same magnitude of PEH at all recovery times [43, 51, 55, 66, 73, 79, 82, 85, 86], and 3 studies reported the greatest PEH between 15–20 min after exercise [25, 26, 30]. Finally, all of the 8 studies that measured blood pressure for more than 1 h in the laboratory setting after exercise reported PEH lasting at least 120 min [22, 40–42, 62, 83, 84, 88]. Taking into account the aforementioned information, future studies aiming to evaluate PEH should assess blood pressure between 20 and 120 min after the exercise to increase the chance for identifying PEH.

The duration of PEH is important to reveal whether the hypotensive effect can have a clinical impact reducing the subject's cardiovascular overload and risk for a long period of time [1]. Ambulatory

▶ **Table 1** Concerns and approaches in post-exercise hypotension studies.

Author and Year	Days of Screening	Time of Blood Pressure Measurements Post-exercise	Control or Comparative Session yes (y)/no (n)	Approach (I - Pre vs. Post)/(II - E vs. C)/(III - NE)
Angadi (2015)[22]	1	3hs/every 15 min	Y	II
Aprile (2016)[98]	2	1 h	Y	II
Augeri (2011)[23]	1	ABPM	Y	III
Azevêdo (2017)[53]	NR	1 h/every 15 min	Y	III
Birk (2012)[54]	NR	10 min	Y	I
Bisquolo (2005)[99]	NR	45 min	Y	I, II
Bonsu (2016)[24]	1	1 h/every 10 min	N	I
Brandão (2002)[82]	2	15;30;60;90 min/ ABPM	Y	I, II
Brito (2014)[55]	NR	10;30;50;70;90 min	Y	II
Brito (2015)[107]	2	45 min	Y	I, II, III
Brito (2017)[89]	2	ABPM	Y	II
Carter III (2001)[56]	NR	5 min	Y	I, II
Cavalcante (2017)[25]	1	1 h/every 15 min	Y	I, II, III
Ciolac (2009)[57]	NR	ABPM	Y	II
Cleroux (1992)[84]	3	30;60;90 min	Y	II
Coats (1989)[58]	NR	1 h/every 10 min	Y	III
Collier (2010)[48]	0	40;60 min	Y	I, III
Costa (2016)[73]	2	1 h/every 10 min	Y	I
Cucato (2015)[106]	2	45 min	Y	I, III
Cunha (2016)[26]	1	20;40;60 min	Y	II
Dantas (2016)[27]	1	ABPM	Y	II
Dawson (2008)[59]	NR	60 min	N	I
Dos Santos (2014)[119]	1	1 h/every 15 min	Y	II
Dujic (2006)[49]	0	30;60 min	N	I
Endo (2012)a[29]	1	1 h/every 15 min	N	I
Endo (2012)b[28]	1	1 h/every 15 min	Y	I, II
Esformes (2006)[60]	1	45 min	Y	I, II
Figueiredo (2015)[50]	2	1 h/every 10 min	Y	I, II
Floras (1989)[85]	3	1 h/every 5 min	Y	I
Floras (1992)[93]	3	1 h/every 5 min	Y	I
Forjaz (1999)[86]	2	90 min/every 5 min	Y	I
Forjaz (2000)[8]	2	ABPM	Y	II
Forjaz (2004)[81]	2	15;30;60;90 min ABPM	Y	II, III
Franklin (1993)[51]	0	1 h/every 15 min	Y	I, II
Gagnon (2012)[30]	1	10;30;50;70;90 min	Y	I, II, III
Goessler (2015)[31]	1	ABPM	Y	I, II
Hagberg (1987)[83]	4	1 h/every 10 min	Y	I
Halliwill (1996)[33]	1	60 min	Y	II
Halliwill (2000)[32]	1	1 h	Y	II
Hamer (2006)[61]	NR	30 min	Y	II
Harvey (2005)[34]	1	45;90 min	Y	I
Headley (2008)[76]	2	1 h/every 10 min/ABPM	Y	III
Headley (2017)[100]	1	1 h/every 10 min/ABPM	Y	I, II, III
Hecksteden (2013)[71]	2	10 min/1 h/24 h	N	I
Heffernan (2007)[35]	1	20 min	Y	I, II
Isea (1994)[94]	NR	30 min;1 h;2 h;3 h;4 h	Y	II
Jones (2007)[36]	1	20 min	Y	III
Jones (2008)a[75]	1	5;10;15;20	Y	III
Jones (2008)b[74]	1	5;10;15;20	Y	III
Keese (2012)[62]	NR	2 h/every 10 min	Y	I, II

▶ **Table 1** Continued

Author and Year	Days of Screening	Time of Blood Pressure Measurements Post-exercise	Control or Comparative Session yes (y)/no (n)	Approach (I - Pre vs. Post)/(II - E vs. C)/(III - NE)
Lacombe (2011)[37]	1	60 min	Y	I, III
Legramante (2002)[108]	3	60;90 min	N	I
Lehmkuhl (2005)[38]	1	ABPM	Y	II
Liu (2012)[39]	1	30 min	Y	I, II
Lockwood (2005)[40]	1	30;60;90 min	Y	II
MacDonald (1999)[78]	5	1 h/every 15 min	Y	I, II
Mac Donald (2000)a[77]	5	1 h/every 15 min	Y	I, II
MacDonald (2000)b[80]	5	1 h/every 15 min	Y	I, II
Mach (2005)[52]	0	30;60;90 min	Y	II
McCord (2006)[101]	1	30;60;90 min	Y	III
Moreira (2014)[72]	NR	60 min	N	I
New (2013)[63]	NR	30;60;90 min	Y	I, II
Notarius (2005)[64]	NR	10 min	Y	I, II
Pricher (2004)[42]	1	2 h/every 20 min	N	I
Queiroz (2009)[87]	2	1 h/every 5 min ABPM	Y	I, II, III
Queiroz (2013)a[109]	2	60 min ABPM	Y	I, II
Queiroz (2013)b[110]	2	60 min	Y	I, II, III
Queiroz (2015)[90]	2	45 min/ABPM	Y	I, II, III
Queiroz (2017)[111]	2	ABPM	Y	I, II
Raine (2001)[43]	1	1 h/every 10 min	Y	III
Rezk (2006)[79]	NR	15;30;60;90 min	Y	III
Rossow (2010)[44]	1	30;60 min	Y	I, II
Santaella (2006)[45]	1	30;60 min	Y	III
Santana (2013)[46]	1	60 min	Y	I, III
Somers (1991)[65]	NR	ABPM	Y	I, II
Souza (2016)[66]	NR	2 h/every 10 min	Y	III
Taylor-Tolbert (2000)[7]	4	ABPM	Y	II
Takahashi (2000)[68]	NR	10 min	Y	I, II
Takahashi (2005)[67]	NR	5 min	Y	I, II
Teixeira (2011)[88]	2	30;60;90;120 min	Y	III
Terblanche (2012)[112]	2	ABPM	Y	I, II, III
Tibana (2015)[120]	NR	10;30;60 min	Y	II, III
Wallace (1997)[9]	1	ABPM	Y	II
Wilcox (1982)[69]	NR	30 min	Y	I
Wilkins (2004)[47]	1	2hs/every 20 min	N	I

AMBP – Ambulatory blood pressure; NR – non-reported; Pre vs. Post – Comparing pre values with post-exercise values; E vs. C – Comparing exercise with control sessions; NE – Evaluating net effect.

blood pressure monitoring is the only adequate measure to evaluate this benefit. However, interpretations of ambulatory blood pressure recordings after exercise are sometimes confounded by the use of prolonged averaging periods (e. g., 12- or 24-h mean) [8, 9, 38, 57, 82, 89, 90]. While studies that report a reduction in mean 12- or 24-h blood pressure provide useful information related to the health benefits of exercise, this approach does not lend itself to a determination of the duration of PEH, which has erroneously been reported as lasting 24 h. The problem is that values early in a timeframe can generate the appearance of a blood pressure reduction, which may be resolved by the end of the averaging timeframe. As a result, authors should opt for reporting hour-to-hour analysis, as demonstrated by Pescatello et al. [91], where mean blood pressure stayed lower than pre-exercise values for 13 h.

It is also important to note that post-exercise ambulatory blood pressure analysis allows for investigating the effects of exercise in real life conditions. However, some care is needed for comparing days with similar daily activities, such as conducting the experiment on the same day of the week (e. g., exercise and control sessions conducted on Monday) or at least to avoid conducting experiments on days with very different activities (e. g., a business day and a weekend day). Another important aspect is to start ambulatory blood pressure recordings at the same time of day in all experimental sessions, since average ambulatory blood pressure values are higher when monitoring was started in the morning than in the afternoon [92].

Body position for blood pressure measurements

The body position of subjects before the exercise and during the recovery period after the exercise is an important experimental consideration for PEH studies and impacts the interpretation of the results, especially when considering hemodynamic mechanisms. An extensive review of the literature suggests that studying subjects in the supine position and in the seated position are both common methods [2], but some studies have not reported the adopted body position [93, 94]. In addition, no study has, to our knowledge, investigated PEH in the upright position. This inconsistency across studies limits comparisons. However, for some purposes, each position may be justified.

The supine position favors hemodynamic measurements without the interference of orthostatic stress [95]. However, it may decrease the generalizability of some findings, as outside of the construct of research studies, it is less common for individuals to recover from exercise in the supine position. In contrast, blood pressure measurements performed with subjects in a seated position may be more relevant, since many persons sit after exercise to rest, talk or do any other task. Actually, due to the orthostatic stress promoted by seated position, diastolic blood pressure increases over time when this position is sustained [95]. This is likely to be secondary to decreased venous return, which deactivates the cardiopulmonary reflex, increasing peripheral sympathetic nerve activity, systemic vascular resistance and diastolic blood pressure [96]. In the laboratory setting, this influence of body position is sufficient to flip the hemodynamics which underlies PEH, from a decrease of systemic vascular resistance when recovery occurs in the supine position to a decrease of cardiac output when recovery occurs in the seated position [43, 67, 68].

Therefore, there are valid rationales for studying subjects in either the supine or the seated position in PEH studies, depending on the experiment's objectives. However, authors should report which body position was used in the studies and should use the same body position pre- and post-exercise in both control and exercise sessions. Likewise, authors should discuss results within the context of the chosen body position. These steps may help avoid misinterpretations.

Method of blood pressure measurement

Due to the sometimes-small magnitude of relevant blood pressure reductions associated with recovery from exercise, rigorous methods of measuring blood pressure are essential in the study of PEH. While auscultatory measurement is the most common method for assessing blood pressure [97], manual sphygmomanometry, with its susceptibility to observer bias, two-digit preference and environmental noise, may not be so appropriate, if it was not made by an experienced evaluator, which minimizes but does not exclude observer bias. As a result, many researchers of PEH use automated blood pressure monitoring [22, 25, 26, 28, 29, 32, 34, 40, 42, 44, 46, 47, 49–51, 53, 54, 59, 62, 65, 71, 73, 76, 86, 87, 94, 98–101].

While various groups have established criteria that such devices must fulfill to be validated (e. g., Association for the Advancement of Medical Instrumentation, British Hypertension Society, European Society of Hypertension), the criteria are focused on clinical testing and do not generally require testing specific to exercise or recovery from exercise. Thus, while there are validations of automated oscillometric devices at rest [16, 18], to the best of our knowledge, no study has tried to validate oscillometric devices for use during the recovery period after exercise. Nevertheless, oscillometric devices actually measure mean blood pressure and estimate systolic and diastolic blood pressure through algorithms based on the range of pulse pressure [97]. Thus when pulse pressure changes, the algorithm is affected [102]. As blood pressure reductions after exercise are usually greater for systolic than diastolic blood pressure [5, 6, 70], pulse pressure decreases during exercise recovery, and this change may introduce bias into oscillometric-estimated systolic and diastolic blood pressures. Indeed, when assessed at the same time, auscultatory blood pressure showed a decrease in systolic and diastolic blood pressures after exercise, which was not detected by automated oscillometry that only reveals a reduction of mean blood pressure [87]. However, oscillometric method minimizes observer bias [102] and is less impacted by environmental noise than manual auscultation. For this reason, when employing oscillometric devices, mean blood pressure should be considered.

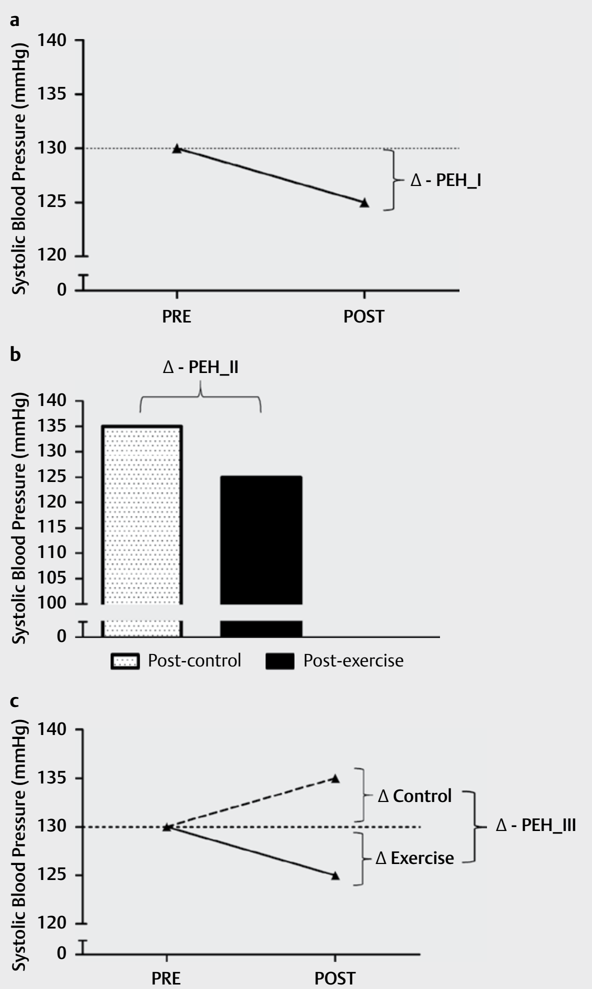
In contrast, at least one automated auscultatory device has been validated for exercise [103] and such device is gaining use in PEH studies [40, 42, 47, 94, 101]. Finger monitors that rely on photoplethysmography (e. g., Finometer) have been validated for the study of PEH [78], and this has encouraged other research groups to use this technique [30, 33, 35–37, 43, 48, 55, 56, 61, 67, 68, 74, 75, 77, 78, 80]. However, while these devices can accurately track beat-to-beat changes in blood pressure, they have limitations for determining absolute values for blood pressure [104].

In brief, automated oscillometric measurements have advantages, mainly in mitigating investigator bias, but their validity is not as well established as automated auscultatory measurements for PEH studies. Considering that auscultatory is also widely used for measurement in PEH studies, its limitation can be minimized by appropriate training and blinding of the observer.

Different approaches to quantifying PEH

Evaluating the occurrence of PEH and quantifying its magnitude requires a comparison of post-exercise blood pressure to some reference, and a variety of blood pressure measurements have been used as this reference value across studies (e. g., measurements taken immediately before the exercise bout, or from a control session without exercise). In addition, the magnitude of PEH has been calculated with different methods, with some incorporating changes over time determined from a control session [105], an example can be seen in ▶ **Fig. 1**.

In many studies [24, 25, 28–31, 34, 37, 39, 42, 44, 46–49, 51, 54, 56, 59, 62–65, 67–69, 71, 73, 77, 78, 80, 82, 83, 85–87, 90, 93, 99, 100, 106–112] (shown in ▶ **Table 1**), PEH has been calculated as simply the difference between post- and pre-exercise blood pressures, which we will define as "PEH_I" (PEH_I = post-exercise blood pressure – pre-exercise blood pressure). Other studies which have included both an exercise and a control session, and have calculated PEH as the difference between post-exercise and post-control blood pressures, what we define as "PEH_II"



▶ **Fig. 1** Demonstrative of post-exercise hypotension (PEH) evaluated by different methods. Systolic blood pressure responses to exercise (continuous line) and control (dotted line) session. (Panel a) - PEH_I post-exercise blood pressure - pre-exercise blood pressure; (Panel b) - PEH_II: post-exercise blood pressure - post-control blood pressure; (Panel c) - PEH_III: [(post-exercise blood pressure – pre-exercise blood pressure) – (post-control blood pressure – pre-control blood pressure)].

(PEH_II = post-exercise blood pressure – post-control blood pressure) [7–9, 22, 25–28, 30–33, 35, 38–40, 44, 50–52, 55–57, 60–65, 67, 68, 77, 78, 80, 82, 84, 87, 89, 90, 94, 98–100, 107, 109–112] (▶**Table 1**). Finally, some studies [23, 25, 30, 36, 37, 43, 45, 46, 48, 53, 58, 66, 74–76, 79, 81, 87, 88, 90, 100, 101, 106, 107, 110, 112] (▶ **Table 1**) have taken a more complex approach, calculating PEH as the net effect, i. e., the difference between responses observed in an exercise session and a control session, which we define as "PEH_III" [PEH_III = (post-exercise blood pressure – pre-exercise blood pressure) – (post-control blood pressure – pre-control blood pressure)].

Thus, researchers should consider the advantages and disadvantages for each approach to data analysis when they are designing their study. PEH_I has the advantage of demanding only one experimental session, with as few as two blood pressure measurements (pre- vs. post-exercise); however, we would strongly suggest the inclusion of more measurements over time rather than the bare minimum of two. In contrast, both PEH_II and PEH_III demand two data collection sessions, performed on different occasions (exercise vs. control). Per session, there is a minimum of one (post-exercise and post-control for PEH_II) or two (pre- and post-exercise/post-control for PEH_III) blood pressure measurements. Again, more measurements across time are desirable.

As studies using PEH_I require less time to execute and fewer data collection sessions, it is not surprising that this approach has been employed in many studies. However, PEH_I does not take into account possible changes in blood pressure that can happen across time independently of exercise (e. g., circadian effects), which can only be controlled by the inclusion of a control session in the study design, as done for PEH_II and PEH_III analysis. The inclusion of a control session as part of the experimental design and analysis in PEH mitigates against any overlying time effect. For example, some studies [8, 81, 90, 106, 107, 109–111] observed no decrease in blood pressure after an exercise bout when relying on PEH_I analysis, but observed an increase in blood pressure from before to after a control session, showing that time alone is associated with increases in blood pressure under their specific experimental conditions. In such cases, PEH_II or PEH_III analysis can demonstrate that the exercise session had a measurable hypotensive effect, blunting the blood pressure increase that was observed with the control session [8, 81, 90, 106, 107, 109–111]. Based on these arguments, whether or not a particular exercise session can promote PEH can only be clearly determined if time effects are controlled by a control session. Then, both PEH_II and PEH_III calculations can be employed to address this issue. On the other hand, if the objective is to compare expected PEH among different exercises protocols, instead of detecting its occurrence, PEH_I may be an option to save time.

PEH_II is often employed when studies rely on ambulatory blood pressure monitoring [7–9, 23, 27, 38, 57, 82, 89, 90, 109, 112] because of the challenges in using an ambulatory blood pressure monitor for more than 24 hrs (i. e., these studies do not include a pre-exercise 24-h recording, just a post-exercise and post-control recording). That said, there are also many studies using this analysis for laboratory-based blood pressure measurements [22, 25, 26, 28, 30, 32, 33, 35, 39, 40, 44, 50–52, 55, 56, 60–64, 67, 68, 77, 78, 80, 84, 94, 98, 99, 107]. The main limitation of PEH_II analysis is that it does not account for day-to-day variation in resting (pre-exercise) blood pressure [113]. Pre-intervention blood pressure might be different between the exercise and control session, confounding the results obtained with PEH_II analysis.

PEH_III analysis overcomes the main limitations of PEH_I and PEH_II, controlling for time effects and day-to-day variation in resting blood pressure. However, it demands two experimental sessions and at least two blood pressure measurements during each session (ideally more). In addition, there are some disadvantages beyond the obvious additional time requirements for conducting studies. The PEH_III value has more degrees of freedom, which decreases its reproducibility and increases the number of subjects needed in these studies [105]. This makes a prior sample size calculation even more important when designing protocols which will use PEH_III analysis.

Therefore, as stated before, all these procedures for calculating PEH are being used in the literature. All of them have advantages and disadvantages, and they may lead to contradictory outcomes. Thus, when designing, analyzing, and interpreting results about PEH, researchers should take into consideration these strengths and limitations. In addition, future research should relate these different approaches with meaningful clinical outcomes.

Reporting Results

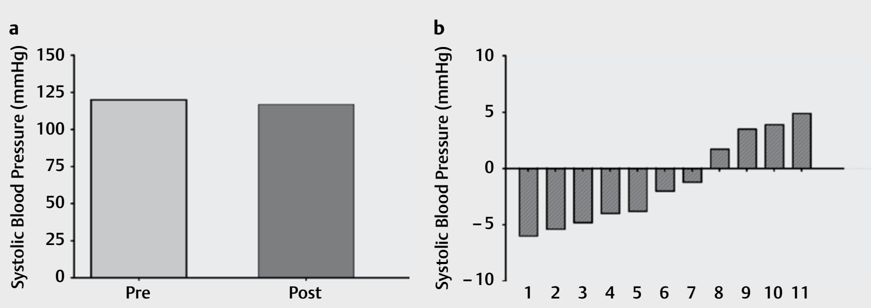
Many studies include results for both systolic and diastolic blood pressure [22–24, 27, 31, 38, 39, 45, 52, 62, 65, 66, 68, 69, 71, 73, 76, 80, 83, 89, 94, 109, 110, 112], whereas others report results for mean blood pressure [30, 32, 40, 42, 47, 48, 54, 56, 67, 74, 99, 101]. It is possible that this is a reflection of researchers with a more clinical orientation versus those who focus on systemic hemodynamics and cardiovascular regulation. Based on the limitations exposed about oscillometric measurement, mean blood pressure should be reported in PEH studies employing this method of measurement. Using auscultatory method, systolic and diastolic represent the primary data, while mean blood pressure is secondary (calculated). Systolic and diastolic blood pressure have been more often related to risk for mortality and disease development [114], reflecting a clinical orientation, while mean blood pressure lends itself to discussions of the determinants of PEH. Thus, the best conduct would be to report all three blood pressure parameters in future studies with auscultatory PEH.

Another rationale for reporting all three parameters, is that some studies only report systolic [33, 36, 37, 46, 51–53, 63, 66, 73] or diastolic blood pressure [64] reductions in response to exercise, rather than finding all blood pressure parameters following the same pattern. This is likely to be more than a statistical underpowering of studies (and a type II error) and may reflect some of the complicated physiology which underlies PEH in some contexts. For example, if one assumes, somewhat simplistically, that changes of systolic blood pressure are more related to cardiac output and changes of diastolic blood pressure are more related to systemic vascular resistance, divergent patterns of PEH between systolic and diastolic blood pressure become reasonable predictions. After performing aerobic exercise, PEH is most often mediated by a decrease of systemic vascular resistance, except in some special conditions in which cardiac output is reduced [2, 3]. In contrast, resistance exercise generally causes PEH by a decrease in cardiac output [79, 109] or a mixture of patterns across subjects [110, 111]. However, studies which find decreased cardiac output can also report a decreased diastolic blood pressure [49, 82, 107]. Likewise for reductions in systemic vascular resistance and systolic blood pressure [33, 34, 36, 43, 44, 58, 60, 63, 75, 79, 81, 84, 94]. Thus, the "simple" attribution of isolated effects on systolic and diastolic blood pressure does not fit all observations. This is likely due to the more integrated nature of the responses, with cardiac and vascular changes being linked by physiological compensation, with superimposed baroreflex regulation of the heart [35] and peripheral vasculature [33]. Again, it is better to report all three blood pressure parameters in future studies on PEH, especially if they portray divergent profiles following exercise.

Reporting individual responses: Do group means describe what is happening?

PEH can be observed in individuals with a broad range of characteristics [8] and after different exercise protocols, including aerobic, dynamic resistance, and isometric exercise [5, 6]. However, within a given study, it is expected that not all subjects present a uniform blood pressure decrease after exercise (as shown in ▶ **Fig. 2**). As a further demonstration, consider ▶ **Fig. 2**, which shows how a study may find no change in mean systolic blood pressure after a session of aerobic exercise (panel a) despite the presence of PEH in the most of the subjects (panel b). Indeed, a previous study that evaluated ambulatory blood pressure after resistance and walking exercises in subjects with peripheral artery disease did not observe any difference in mean values, but individual responses revealed that many subjects presented clinically relevant blood pressure reductions [115]. Along these lines, Forjaz et al. [8] observed no change in the group mean blood pressure for hypertensive subjects and a decrease in the group mean blood pressure for normotensive subjects, yet in both groups there were subjects who increased, decreased, or maintained blood pressure after the exercise session.

As in other areas of research, we recognize that these different individual responses may be of great importance. There may be a



▶ **Fig. 2** Hypothetical data of systolic blood pressure (mmHg) assessed pre and post an exercise session, demonstrating how a group's means can mask important individual responses. Panel a): data presented by means; Panel b) data presented by individual responses.

strong rationale to classify subjects as “responders” and “non-responders” [73, 115]. However, similar to debates deriving from randomized control trials, there is still no consensus on how to define a true “response”: whether it should be based on the presence of clinically relevant change [116] or on clearly measurable change [117]. In fact, the clinically relevant change for PEH has yet to be defined. An option might be when blood pressure response to exercise overcome the error of the blood pressure measurement [118]. However, clearly measurable change is still not clear, since very few studies have adequately investigated the reproducibility of individual PEH patterns [105].

As a result, this is an open area for future investigations that needs to be addressed. Studies employing analysis of group mean data revealed the clinical relevance of PEH, but individual response analysis may be necessary to advance comprehension of this phenomenon, clarifying as well whether there is a role for responders and non-responders.

Analyses of individual responses have been highlighted in the literature, and this approach shows great promise as a tool to identify who is responsive to a specific intervention, based on an acute evaluation. However, the lack of standardization of how PEH is assessed creates a barrier for moving this approach forward.

Conclusions

Based on this narrative review, it is possible to recommend that rigor and reproducibility can be increased in PEH studies by inclusion of familiarization sessions for blood pressure measurements as well following some recommendations (▶ **Table 2**). Investigators should be sure that screening visits are sufficient to familiarize subjects so that pre-exercise values are stable and representative of the individuals resting blood pressure. Apart from familiarization, authors who desire to observe blood pressure decreases are strongly encouraged to assess blood pressure at least for 20 min after exercise, but preferably for 120 min. They also should employ blood pressure measures considering their limitations. Finally, there are several statistical approaches for evaluating PEH, and the pros and cons of each must be considered in parallel to study design, and not after the study has been completed, since the choice impacts sample size determinations and can change the conclusion of study, illuminating or hiding the presence of PEH.

Finally, this is a narrative review that did not intend to completely explore the theme. Some important recommendations have been highlighted, but other researchers might have additional concerns or recommendations. For this reason, a broad discussion by a panel of experts leading to a task force about the methodological concerns in PEH studies should be encouraged.

▶ **Table 2** Recommendations to guide post-exercise hypotension (PEH) studies.

– Perform, at least, two screening visits for blood pressure measurements before the first study day
– Track blood pressure for at least 20 min but preferably 120 min after exercise
– Determine the statistical approach before the study design
– If discussing mechanisms, include presentation of the mean blood pressure
– Avoid drawing conclusions on the duration of PEH from data averaged over long timeframes

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Conflict of Interest

The authors declare that there is no conflict of interest.

References

[1] Kenney MJ, Seals DR. Postexercise hypotension. Key features, mechanisms, and clinical significance. Hypertension 1993; 22: 653–664
[2] Brito LC, Queiroz AC, Forjaz CL. Influence of population and exercise protocol characteristics on hemodynamic determinants of post-aerobic exercise hypotension. Braz J Med Biol Res 2014; 47: 626–636
[3] Halliwill JR, Buck TM, Lacewell AN, Romero SA. Postexercise hypotension and sustained postexercise vasodilatation: What happens after we exercise? Exp Physiol 2013; 98: 7–18
[4] Luttrell MJ, Halliwill JR. Recovery from exercise: Vulnerable state, window of opportunity, or crystal ball? Front Physiol 2015; 6: 204
[5] Carpio-Rivera E, Moncada-Jimenez J, Salazar-Rojas W, Solera-Herrera A. Acute effects of exercise on blood pressure: A Meta-analytic investigation. Arq Bras Cardiol 2016; 106: 422–433
[6] Casonatto J, Goessler KF, Cornelissen VA, Cardoso JR, Polito MD. The blood pressure-lowering effect of a single bout of resistance exercise: A systematic review and meta-analysis of randomised controlled trials. Eur J Prev Cardiol 2016; 23: 1700–1714
[7] Taylor-Tolbert NS, Dengel DR, Brown MD, McCole SD, Pratley RE, Ferrell RE, Hagberg JM. Ambulatory blood pressure after acute exercise in older men with essential hypertension. Am J Hypertens 2000; 13: 44–51
[8] Forjaz CL, Tinucci T, Ortega KC, Santaella DF, Mion D Jr., Negrao CE. Factors affecting post-exercise hypotension in normotensive and hypertensive humans. Blood Press Monit 2000; 5: 255–262
[9] Wallace JP, Bogle PG, King BA, Krasnoff JB, Jastremski CA. A comparison of 24-h average blood pressures and blood pressure load following exercise. Am J Hypertens 1997; 10: 728–734
[10] Wallace JP, Bogle PG, King BA, Krasnoff JB, Jastremski CA. The magnitude and duration of ambulatory blood pressure reduction following acute exercise. J Hum Hypertens 1999; 13: 361–366
[11] Cronin P, Ryan F, Coughlan M. Undertaking a literature review: A step-by-step approach. Br J Nurs 2008; 17: 38–43
[12] Harriss DJ, Macsween A, Atkinson G. Standards for ethics in sport and exercise science research: 2018 update. Int J Sports Med 2017; 38: 1126–1131
[13] Stolt M, Sjonell G, Astrom H, Hansson L. The reliability of auscultatory measurement of arterial blood pressure. A comparison of the standard and a new methodology. Am J Hypertens 1990; 3: 697–703
[14] Clark EG, Glock CY, Schweitzer MD, Vought RL. Studies in hypertension. II. Variability of daily blood pressure measurements in the same individuals over a 3-week period. J Chronic Dis 1956; 4: 469–476

[15] Rosner B, Polk BF. The instability of blood pressure variability over time. J Chronic Dis 1981; 34: 135–139

[16] Malachias MVB, Paulo Cesar Veiga Jardim PCVJ, Almeida FA, Lima EJ, Feitosa GS 7th. Brazilian Guideline of Arterial Hypertension: Chapter 7 - Pharmacological Treatment. Arq Bras Cardiol 2016; 107: 35–43

[17] Mancia G, Fagard R, Narkiewicz K, Redon J, Zanchetti A, Bohm M, Christiaens T, Cifkova R, De Backer G, Dominiczak A, Galderisi M, Grobbee DE, Jaarsma T, Kirchhof P, Kjeldsen SE, Laurent S, Manolis AJ, Nilsson PM, Ruilope LM, Schmieder RE, Sirnes PA, Sleight P, Viigimaa M, Waeber B, Zannad F, Task Force M. 2013 ESH/ESC Guidelines for the management of arterial hypertension: The Task Force for the management of arterial hypertension of the European Society of Hypertension (ESH) and of the European Society of Cardiology (ESC). J Hypertens 2013; 31: 1281–1357

[18] Chobanian AV, Bakris GL, Black HR, Cushman WC, Green LA, Izzo JL Jr., Jones DW, Materson BJ, Oparil S, Wright JT Jr., Roccella EJ. Seventh report of the joint national committee on prevention, detection, evaluation, and treatment of high blood pressure. Hypertension 2003; 42: 1206–1252

[19] Pearce KA, Grimm RH Jr., Rao S, Svendsen K, Liebson PR, Neaton JD, Ensrud K. Population-derived comparisons of ambulatory and office blood pressures. Implications for the determination of usual blood pressure and the concept of white coat hypertension. Arch Intern Med 1992; 152: 750–756

[20] Andre JL, Petit JC, Gueguen R, Deschamps JP. [Variability of arterial pressure and heart rate measured at two periods of 15 min to 15 days intervals]. Arch Mal Coeur Vaiss 1987; 80: 1005–1010

[21] Bovet P, Gervasoni JP, Ross AG, Mkamba M, Mtasiwa DM, Lengeler C, Burnier M, Paccaud F. Assessing the prevalence of hypertension in populations: Are we doing it right? J Hypertens 2003; 21: 509–517

[22] Angadi SS, Bhammar DM, Gaesser GA. Postexercise hypotension after continuous, aerobic interval, and sprint interval exercise. J Strength Cond Res 2015; 29: 2888–2893

[23] Augeri AL, Tsongalis GJ, Van Heest JL, Maresh CM, Thompson PD, Pescatello LS. The endothelial nitric oxide synthase -786 T > C polymorphism and the exercise-induced blood pressure and nitric oxide responses among men with elevated blood pressure. Atherosclerosis 2009; 204: e28–e34

[24] Bonsu B, Terblanche E. The training and detraining effect of high-intensity interval training on post-exercise hypotension in young overweight/obese women. Eur J Appl Physiol 2016; 116: 77–84

[25] Cavalcante BR, Ritti-Dias RM, Soares AH, Lima AH, Correia MA, De Matos LD, Gobbi F, Leicht AS, Wolosker N, Cucato GG. A single bout of arm crank exercise promotes positive emotions and post exercise hypotension in patients with symptomatic peripheral artery disease. Eur J Vasc Endovasc Surg 2017; 53: 223–228

[26] Cunha F, Midgley AW, Pescatello L, Soares PP, Farinatti P. Acute hypotensive response to continuous and accumulated isocaloric aerobic bouts. Int J Sports Med 2016; 37: 855–862

[27] Dantas TC, Farias Junior LF, Frazao DT, Silva PH, Sousa Junior AE, Costa IB, Ritti-Dias RM, Forjaz CL, Duhamel TA, Costa EC. A single session of low-volume high-intensity interval exercise reduces ambulatory blood pressure in normotensive males. J Strength Cond Res 2017; 31: 2263–2269

[28] Endo MY, Kajimoto C, Yamada M, Miura A, Hayashi N, Koga S, Fukuba Y. Acute effect of oral water intake during exercise on post-exercise hypotension. Eur J Clin Nutr 2012; 66: 1208–1213

[29] Endo MY, Shimada K, Miura A, Fukuba Y. Peripheral and central vascular conductance influence on post-exercise hypotension. J Physiol Anthropol 2012; 31: 32

[30] Gagnon D, Lynn AG, Binder K, Boushel RC, Kenny GP. Mean arterial pressure following prolonged exercise in the heat: Influence of training status and fluid replacement. Scand J Med Sci Sports 2012; 22: e99–e107

[31] Goessler KF, Cornelissen VA, de Oliveira EM, de FMG, Polito MD. ACE polymorphisms and the acute response of blood pressure to a walk in medicated hypertensive patients. J Renin Angiotensin Aldosterone Syst 2015; 16: 720–729

[32] Halliwill JR, Minson CT, Joyner MJ. Effect of systemic nitric oxide synthase inhibition on postexercise hypotension in humans. J Appl Physiol (1985) 2000; 89: 1830–1836

[33] Halliwill JR, Taylor JA, Eckberg DL. Impaired sympathetic vascular regulation in humans after acute dynamic exercise. J Physiol 1996; 495: (Pt 1) 279–288

[34] Harvey PJ, Morris BL, Kubo T, Picton PE, Su WS, Notarius CF, Floras JS. Hemodynamic after-effects of acute dynamic exercise in sedentary normotensive postmenopausal women. J Hypertens 2005; 23: 285–292

[35] Heffernan KS, Collier SR, Kelly EE, Jae SY, Fernhall B. Arterial stiffness and baroreflex sensitivity following bouts of aerobic and resistance exercise. Int J Sports Med 2007; 28: 197–203

[36] Jones HG K, Edwards B, Atkinson G. Is the magnitude of acute post-exercise hypotension mediated by exercise intensity or total work done? Eur J Appl Physiol 2007; 102: 33–40

[37] Lacombe SP, Goodman JM, Spragg CM, Liu S, Thomas SG. Interval and continuous exercise elicit equivalent postexercise hypotension in prehypertensive men, despite differences in regulation. Appl Physiol Nutr Metab 2011; 36: 881–891

[38] Lehmkuhl LA, Park S, Zakutansky D, Jastremski CA, Wallace JP. Reproducibility of postexercise ambulatory blood pressure in Stage I hypertension. J Hum Hypertens 2005; 19: 589–595

[39] Liu S, Goodman J, Nolan R, Lacombe S, Thomas SG. Blood pressure responses to acute and chronic exercise are related in prehypertension. Med Sci Sports Exerc 2012; 44: 1644–1652

[40] Lockwood JM, Pricher MP, Wilkins BW, Holowatz LA, Halliwill JR. Postexercise hypotension is not explained by a prostaglandin-dependent peripheral vasodilation. J Appl Physiol (1985) 2005; 98: 447–453

[41] McCord JL, Beasley JM, Halliwill JR. H2-receptor-mediated vasodilation contributes to postexercise hypotension. J Appl Physiol (1985) 2006; 100: 67–75

[42] Pricher MP, Holowatz LA, Williams JT, Lockwood JM, Halliwill JR. Regional hemodynamics during postexercise hypotension. I. Splanchnic and renal circulations. J Appl Physiol (1985) 2004; 97: 2065–2070

[43] Raine NM, Cable NT, George KP, Campbell IG. The influence of recovery posture on post-exercise hypotension in normotensive men. Med Sci Sports Exerc 2001; 33: 404–412

[44] Rossow L, Yan H, Fahs CA, Ranadive SM, Agiovlasitis S, Wilund KR, Baynard T, Fernhall B. Postexercise hypotension in an endurance-trained population of men and women following high-intensity interval and steady-state cycling. Am J Hypertens 2010; 23: 358–367

[45] Santaella DF, Araujo EA, Ortega KC, Tinucci T, Mion D Jr., Negrao CE, de Moraes Forjaz CL. Aftereffects of exercise and relaxation on blood pressure. Clin J Sport Med 2006; 16: 341–347

[46] Santana HA, Moreira SR, Asano RY, Sales MM, Cordova C, Campbell CS, Espindola FS, Sposito AC, Nobrega OT, Simoes HG. Exercise intensity modulates nitric oxide and blood pressure responses in hypertensive older women. Aging Clin Exp Res 2013; 25: 43–48

[47] Wilkins BW, Minson CT, Halliwill JR. Regional hemodynamics during postexercise hypotension. II. Cutaneous circulation. J Appl Physiol (1985) 2004; 97: 2071–2076

[48] Collier SR, Diggle MD, Heffernan KS, Kelly EE, Tobin MM, Fernhall B. Changes in arterial distensibility and flow-mediated dilation after acute resistance vs. aerobic exercise. J Strength Cond Res 2010; 24: 2846–2852

[49] Dujic Z, Ivancev V, Valic Z, Bakovic D, Marinovic-Terzic I, Eterovic D, Wisloff U. Postexercise hypotension in moderately trained athletes after maximal exercise. Med Sci Sports Exerc 2006; 38: 318–322

[50] Figueiredo T, Willardson JM, Miranda H, Bentes CM, Reis VM, Simao R. Influence of load intensity on postexercise hypotension and heart rate variability after a strength training session. J Strength Cond Res 2015; 29: 2941–2948

[51] Franklin PJ, Green DJ, Cable NT. The influence of thermoregulatory mechanisms on post-exercise hypotension in humans. J Physiol 1993; 470: 231–241

[52] Mach C, Foster C, Brice G, Mikat RP, Porcari JP. Effect of exercise duration on postexercise hypotension. J Cardiopulm Rehabil 2005; 25: 366–369

[53] Azevedo LM, de Souza AC, Santos LE, Miguel Dos Santos R, de Fernandes MO, Almeida JA, Pardono E. Fractionated concurrent exercise throughout the day does not promote acute blood pressure benefits in hypertensive middle-aged women. Front Cardiovasc Med 2017; 4: 6

[54] Birk GK, Dawson EA, Batterham AM, Atkinson G, Cable T, Thijssen DH, Green DJ. Effects of exercise intensity on flow mediated dilation in healthy humans. Int J Sports Med 2013; 34: 409–414

[55] Brito Ade F, de Oliveira CV, Santos Mdo S, Santos Ada C. High-intensity exercise promotes postexercise hypotension greater than moderate intensity in elderly hypertensive individuals. Clin Physiol Funct Imaging 2014; 34: 126–132

[56] Carter R 3rd, Watenpaugh DE, Smith ML. Gender differences in cardiovascular regulation during recovery from exercise. J Appl Physiol (1985) 2001; 91: 1902–1907

[57] Ciolac EG, Guimaraes GV, VM DA, Bortolotto LA, Doria EL, Bocchi EA. Acute effects of continuous and interval aerobic exercise on 24-h ambulatory blood pressure in long-term treated hypertensive patients. Int J Cardiol 2009; 133: 381–387

[58] Coats AJ, Conway J, Isea JE, Pannarale G, Sleight P, Somers VK. Systemic and forearm vascular resistance changes after upright bicycle exercise in man. J Physiol 1989; 413: 289–298

[59] Dawson EA, Whyte GP, Black MA, Jones H, Hopkins N, Oxborough D, Gaze D, Shave RE, Wilson M, George KP, Green DJ. Changes in vascular and cardiac function after prolonged strenuous exercise in humans. J Appl Physiol (1985) 2008; 105: 1562–1568

[60] Esformes JI, Norman F, Sigley J, Birch KM. The influence of menstrual cycle phase upon postexercise hypotension. Med Sci Sports Exerc 2006; 38: 484–491

[61] Hamer M, Boutcher SH. Impact of moderate overweight and body composition on postexercise hemodynamic responses in healthy men. J Hum Hypertens 2006; 20: 612–617

[62] Keese F, Farinatti P, Pescatello L, Cunha FA, Monteiro WD. Aerobic exercise intensity influences hypotension following concurrent exercise sessions. Int J Sports Med 2012; 33: 148–153

[63] New KJ, Reilly ME, Templeton K, Ellis G, James PE, McEneny J, Penney M, Hooper J, Hullin D, Davies B, Bailey DM. Free radical-mediated lipid peroxidation and systemic nitric oxide bioavailability: Implications for postexercise hemodynamics. Am J Hypertens 2013; 26: 126–134

[64] Notarius CF, Morris BL, Floras JS. Caffeine attenuates early post-exercise hypotension in middle-aged subjects. Am J Hypertens 2006; 19: 184–188

[65] Somers VK, Conway J, Coats A, Isea J, Sleight P. Postexercise hypotension is not sustained in normal and hypertensive humans. Hypertension 1991; 18: 211–215

[66] Souza AA, Silva RS, Silva TF, Tavares RL, Silva AS. Influence of different doses of coffee on post-exercise blood pressure response. Am J Cardiovasc Dis 2016; 6: 146–152

[67] Takahashi T, Hayano J, Okada A, Saitoh T, Kamiya A. Effects of the muscle pump and body posture on cardiovascular responses during recovery from cycle exercise. Eur J Appl Physiol 2005; 94: 576–583

[68] Takahashi T, Okada A, Saitoh T, Hayano J, Miyamoto Y. Difference in human cardiovascular response between upright and supine recovery from upright cycle exercise. Eur J Appl Physiol 2000; 81: 233–239

[69] Wilcox RG, Bennett T, Brown AM, Macdonald IA. Is exercise good for high blood pressure? Br Med J (Clin Res Ed) 1982; 285: 767–769

[70] Pescatello LS, Franklin BA, Fagard R, Farquhar WB, Kelley GA, Ray CA. American College of Sports Medicine position stand. Exercise and hypertension. Med Sci Sports Exerc 2004; 36: 533–553

[71] Hecksteden A, Grutters T, Meyer T. Association between postexercise hypotension and long-term training-induced blood pressure reduction: A pilot study. Clin J Sport Med 2013; 23: 58–63

[72] Moreira SR, Cucato GG, Terra DF, Ritti-Dias RM. Acute blood pressure changes are related to chronic effects of resistance exercise in medicated hypertensives elderly women. Clin Physiol Funct Imaging 2014; 36: 242–248

[73] Costa EC, Dantas TC, de Farias Junior LF, Frazao DT, Prestes J, Moreira SR, Ritti-Dias RM, Tibana RA, Duhamel TA. Inter- and intra-individual analysis of post-exercise hypotension following a single bout of high-intensity interval exercise and continuous exercise: A pilot study. Int J Sports Med 2016; 37: 1038–1043

[74] Jones H, Pritchard C, George K, Edwards B, Atkinson G. The acute post-exercise response of blood pressure varies with time of day. Eur J Appl Physiol 2008; 104: 481–489

[75] Jones H, George K, Edwards B, Atkinson G. Effects of time of day on post-exercise blood pressure: Circadian or sleep-related influences? Chronobiol Int 2008; 25: 987–998

[76] Headley SA, Germain MJ, Milch CM, Buchholz MP, Coughlin MA, Pescatello LS. Immediate blood pressure-lowering effects of aerobic exercise among patients with chronic kidney disease. Nephrology (Carlton) 2008; 13: 601–606

[77] MacDonald JR, MacDougall JD, Hogben CD. The effects of exercise duration on post-exercise hypotension. J Hum Hypertens 2000; 14: 125–129

[78] MacDonald J, MacDougall J, Hogben C. The effects of exercise intensity on post exercise hypotension. J Hum Hypertens 1999; 13: 527–531

[79] Rezk CC, Marrache RC, Tinucci T, Mion D Jr., Forjaz CL. Post-resistance exercise hypotension, hemodynamics, and heart rate variability: Influence of exercise intensity. Eur J Appl Physiol 2006; 98: 105–112

[80] MacDonald JR, MacDougall JD, Hogben CD. The effects of exercising muscle mass on post exercise hypotension. J Hum Hypertens 2000; 14: 317–320

[81] Forjaz CL, Cardoso CG Jr., Rezk CC, Santaella DF, Tinucci T. Postexercise hypotension and hemodynamics: the role of exercise intensity. J Sports Med Phys Fitness 2004; 44: 54–62

[82] Brandao Rondon MU, Alves MJ, Braga AM, Teixeira OT, Barretto AC, Krieger EM, Negrao CE. Postexercise blood pressure reduction in elderly hypertensive patients. J Am Coll Cardiol 2002; 39: 676–682

[83] Hagberg JM, Montain SJ, Martin WH 3rd. Blood pressure and hemodynamic responses after exercise in older hypertensives. J Appl Physiol (1985) 1987; 63: 270–276

[84] Cleroux J, Kouame N, Nadeau A, Coulombe D, Lacourciere Y. Aftereffects of exercise on regional and systemic hemodynamics in hypertension. Hypertension 1992; 19: 183–191

[85] Floras JS, Sinkey CA, Aylward PE, Seals DR, Thoren PN, Mark AL. Postexercise hypotension and sympathoinhibition in borderline hypertensive men. Hypertension 1989; 14: 28–35

[86] Forjaz CL, Ramires PR, Tinucci T, Ortega KC, Salomao HE, Ignes EC, Wajchenberg BL, Negrao CE, Mion D Jr.. Postexercise responses of muscle sympathetic nerve activity and blood flow to hyperinsulinemia in humans. J Appl Physiol (1985) 1999; 87: 824–829

[87] Queiroz AC, Gagliardi JF, Forjaz CL, Rezk CC. Clinic and ambulatory blood pressure responses after resistance exercise. J Strength Cond Res 2009; 23: 571–578

[88] Teixeira L, Ritti-Dias RM, Tinucci T, Mion Junior D, Forjaz CL. Post-concurrent exercise hemodynamics and cardiac autonomic modulation. Eur J Appl Physiol 2011; 111: 2069–2078

[89] Brito LC, Rezende RA, Mendes C, Silva-Junior ND, Tinucci T, Cipolla-Neto J, Forjaz CL. Separate aftereffects of morning and evening exercise on ambulatory blood pressure in pre-hypertensive men. J Sports Med Phys Fitness 2018; 58: 157–163

[90] Queiroz AC, Sousa JC, Cavalli AA, Silva ND Jr., Costa LA, Tobaldini E, Montano N, Silva GV, Ortega K, Mion D Jr., Tinucci T, Forjaz CL. Post-resistance exercise hemodynamic and autonomic responses: Comparison between normotensive and hypertensive men. Scand J Med Sci Sports 2015; 25: 486–494

[91] Pescatello LS, Fargo AE, Leach CN Jr., Scherzer HH. Short-term effect of dynamic exercise on arterial blood pressure. Circulation 1991; 83: 1557–1561

[92] Wallace JP, Park S, Zakutansky DW, Lehmkuhl LA, Jastremski CA. Time of day to monitor ambulatory blood pressure affects the outcome. Blood Press Monit 2005; 10: 43–50

[93] Floras JS, Wesche J. Haemodynamic contributions to post-exercise hypotension in young adults with hypertension and rapid resting heart rates. J Hum Hypertens 1992; 6: 265–269

[94] Isea JE, Piepoli M, Adamopoulos S, Pannarale G, Sleight P, Coats AJ. Time course of haemodynamic changes after maximal exercise. Eur J Clin Invest 1994; 24: 824–829

[95] Gotshall RW, Aten LA, Yumikura S. Difference in the cardiovascular response to prolonged sitting in men and women. Can J Appl Physiol 1994; 19: 215–225

[96] Mark AL, Mancia G, Cardiopulmonary baroreflex in humans. In Rowell LB, Shepherd JT. (eds) Handbook of Physiology; New York: Oxford: 1996: 795–813

[97] Mancia G, Zanchetti A. One hundred years of auscultatory blood pressure: Commemorating N.S. Korotkoff. J Hypertens 2005; 23: 1–2

[98] Aprile DC, Oneda B, Gusmao JL, Costa LA, Forjaz CL, Mion D Jr., Tinucci T. Post-exercise hypotension is mediated by a decrease in sympathetic nerve activity in stages 2-3 CKD. Am J Nephrol 2016; 43: 206–212

[99] Bisquolo VA, Cardoso CG Jr., Ortega KC, Gusmao JL, Tinucci T, Negrao CE, Wajchenberg BL, Mion D Jr., Forjaz CL.. Previous exercise attenuates muscle sympathetic activity and increases blood flow during acute euglycemic hyperinsulinemia. J Appl Physiol (1985) 2005; 98: 866–871

[100] Headley S, Germain M, Wood R, Joubert J, Milch C, Evans E, Cornelius A, Brewer B, Taylor B, Pescatello LS. Blood pressure response to acute and chronic exercise in chronic kidney disease. Nephrology (Carlton) 2017; 22: 72–78

[101] McCord JL, Halliwill JR. H1 and H2 receptors mediate postexercise hyperemia in sedentary and endurance exercise-trained men and women. J Appl Physiol (1985) 2006; 101: 1693–1701

[102] van Montfrans GA. Oscillometric blood pressure measurement: Progress and problems. Blood Press Monit 2001; 6: 287–290

[103] Cameron JD, Stevenson I, Reed E, McGrath BP, Dart AM, Kingwell BA. Accuracy of automated auscultatory blood pressure measurement during supine exercise and treadmill stress electrocardiogram-testing. Blood Press Monit 2004; 9: 269–275

[104] Gomides RS, Dias RM, Souza DR, Costa LA, Ortega KC, Mion D Jr., Tinucci T, de Moraes Forjaz CL. Finger blood pressure during leg resistance exercise. Int J Sports Med 2010; 31: 590–595

[105] Fecchio RY, Chehuen M, Brito LC, Pecanha T, Queiroz ACC, de Moraes Forjaz CL. Reproducibility (reliability and agreement) of post-exercise hypotension. Int J Sports Med 2017; 38: 1029–1034

[106] Cucato GG, Chehuen Mda R, Ritti-Dias RM, Carvalho CR, Wolosker N, Saxton JM, Forjaz CL. Post-walking exercise hypotension in patients with intermittent claudication. Med Sci Sports Exerc 2015; 47: 460–467

[107] de Brito LC, Rezende RA, da Silva Junior ND, Tinucci T, Casarini DE, Cipolla-Neto J, Forjaz CL.. Post-exercise hypotension and its mechanisms differ after morning and evening exercise: A randomized crossover study. PLoS One 2015; 10: e0132458

[108] Legramante JM, Galante A, Massaro M, Attanasio A, Raimondi G, Pigozzi F, Iellamo F. Hemodynamic and autonomic correlates of postexercise hypotension in patients with mild hypertension. Am J Physiol Regul Integr Comp Physiol 2002; 282: R1037–R1043

[109] Queiroz AC, Kanegusuku H, Chehuen MR, Costa LA, Wallerstein LF, Dias da Silva VJ, Mello MT, Ugrinowitsch C, Forjaz CL. Cardiac work remains high after strength exercise in elderly. Int J Sports Med 2013; 34: 391–397

[110] Queiroz AC, Rezk CC, Teixeira L, Tinucci T, Mion D, Forjaz CL. Gender influence on post-resistance exercise hypotension and hemodynamics. Int J Sports Med 2013; 34: 939–944

[111] Queiroz AC, Sousa JC Jr., Silva ND Jr., Tobaldini E, Ortega KC, de Oliveira EM, Brum PC, Montano N, Mion D Jr., Tinucci T, de Moraes Forjaz CL.. Captopril does not potentiate post-exercise hypotension: a randomized crossover study. Int J Sports Med 2017; 38: 270–277

[112] Terblanche E, Millen AM. The magnitude and duration of post-exercise hypotension after land and water exercises. Eur J Appl Physiol 2012; 112: 4111–4118

[113] Parati G, Ochoa JE, Lombardi C, Bilo G. Assessment and management of blood-pressure variability. Nat Rev Cardiol 2013; 10: 143–155

[114] Hadaegh F, Shafiee G, Hatami M, Azizi F. Systolic and diastolic blood pressure, mean arterial pressure and pulse pressure for prediction of cardiovascular events and mortality in a Middle Eastern population. Blood Press 2012; 21: 12–18

[115] Lima AH, Miranda AS, Correia MA, Soares AH, Cucato GG, Sobral Filho DC, Gomes SL, Ritti-Dias RM. Individual blood pressure responses to walking and resistance exercise in peripheral artery disease patients: Are the mean values describing what is happening? J Vasc Nurs 2015; 33: 150–156

[116] Guyatt GH, Juniper EF, Walter SD, Griffith LE, Goldstein RS. Interpreting treatment effects in randomised trials. BMJ 1998; 316: 690–693

[117] Bouchard C, Blair SN, Church TS, Earnest CP, Hagberg JM, Hakkinen K, Jenkins NT, Karavirta L, Kraus WE, Leon AS, Rao DC, Sarzynski MA, Skinner JS, Slentz CA, Rankinen T. Adverse metabolic response to regular exercise: Is it a rare or common occurrence? PLoS One 2012; 7: e37887

[118] Swinton PA, Hemingway BS, Saunders B, Gualano B, Dolan E. A statistical framework to interpret individual response to intervention: Paving the way for personalized nutrition and exercise prescription. Front Nutr 2018; 5: 41

[119] Dos Santos ES, Asano RY, Filho IG, Lopes NL, Panelli P, Nascimento Dda C, Collier SR, Prestes J. Acute and chronic cardiovascular response to 16 weeks of combined eccentric or traditional resistance and aerobic training in elderly hypertensive women: A randomized controlled trial. J Strength Cond Res 2014; 28: 3073–3084

[120] Tibana RA, de Sousa NM, da Cunha Nascimento D, Pereira GB, Thomas SG, Balsamo S, Simoes HG, Prestes J. Correlation between acute and chronic 24-hour blood pressure response to resistance training in adult women. Int J Sports Med 2015; 36: 82–89