Accepted Manuscript

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PII: S1933-1711(18)30291-2

DOI: https://doi.org/10.1016/j.jash.2018.10.006

Reference: JASH 1215

To appear in: *Journal of the American Society of Hypertension*

Received Date: 14 February 2018

Revised Date: 5 October 2018

Accepted Date: 16 October 2018

Please cite this article as: Brito LC, Fecchio RY, Peçanha T, Andrade-Lima A, Halliwill JR, Forjaz CLM, Post-exercise Hypotension as a Clinical Tool: A “Single Brick” in The Wall, *Journal of the American Society of Hypertension* (2018), doi: https://doi.org/10.1016/j.jash.2018.10.006.

Post-exercise Hypotension as a Clinical Tool: A "Single Brick" in The Wall

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Conflict of interest
There is no conflict of interest

Funding
None

Acknowledgement
We would like to thank the Fundação de Amparo à Pesquisa do Estado de São Paulo (FAPESP 2014/21667-6).

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Abstract

After an exercise session, a reduction of blood pressure (BP) is expected, a phenomenon called post-exercise hypotension (PEH). PEH as a predictor of chronic training responses for BP has been broadly explored. It suggests that when PEH occurs after each exercise sessions, its benefits may summate over time, contributing to the chronic adaptation. Thus, PEH is an important clinical tool, acting as a "single brick" in the wall, and building the chronic effect of decreasing BP. However, there is large variation in the literature regarding methodology and results, creating barriers for understanding comparisons among PEH studies. Thus, the differences among subjects' and exercise protocols' characteristics observed in the studies investigating PEH must be considered when readers interpret the results. Further, understanding on these factors of influence might be useful for avoiding misinterpretations in future comparisons and how the subjacent mechanisms contribute to the BP reduction after exercise.

Keywords: Blood pressure; aerobic exercise; resistance exercise; cardiovascular; acute exercise.

Introduction

Hill's initial observations on the behavior of blood pressure (BP) after performing an exercise session in 1898 [1] generated some attention of researchers of that period. However, the topic was largely ignored until 1981, when Fitzgerald, who was hypertensive, brought the effect of exercise on BP into a clinical perspective in the scientific community by describing his own BP decrease after jogging [2].

Since then, many researchers have dedicated their careers to understand the BP behavior after an exercise session, a phenomenon termed by Kenney and Seals [3] as post-exercise hypotension (PEH). PEH is characterized by reduction in systolic and/or diastolic BP after a single bout of exercise to below a control level without any clinical symptomatic hypotension. Based on this concept PEH, has been analyzed as a reduction in BP below the values observed either immediately prior to exercise or on a control day (e.g. same conditions but without exercise) [3]. The current consensus is that PEH holds clinical relevance, due to the magnitude of the pressure reductions and their duration, lasting for many hours [3-5]. In addition, BP responses observed in the first hour after exercise have importance beyond the immediate pressure reduction [6], as it provides insight to future BP adaptations to training [7-9]. It may reflect a vulnerable state in cases developing severe PEH leading to syncope [6], or may be a window of opportunity to understand influencing factors (such as gender or level of physical fitness, among others) behind the physiological changes after exercise [10, 11]. Based on these important implications, subjects' and exercise protocols' characteristics may be faced as crucial factors of influence for data interpretation in PEH investigations. In addition, the mechanisms responsible for PEH are not completely understood and many factors may influence them.

POST-EXERCISE HYPOTENSION AS A CLINICAL TOOL

Clinical relevance of post-exercise hypotension

Regular physical exercise has been widely accepted as an important strategy to control BP [12, 13], a single session of exercise is able to promote PEH, which is more pronounced and sustained in pre-hypertensive and hypertensive individuals than in the normotensive population [14]. Kenney and Seals in 1993 [3] called attention to PEH, which must be sustained at an enough level lasting many hours to be considered clinically relevant.

Recently, a meta-analysis that included 65 studies showed average systolic pressure reductions of approximately 6 and 8 mmHg after exercise in pre-hypertensives and hypertensives, respectively [15]. Moreover, systolic/diastolic PEH was observed after aerobic (6/4 mmHg) and resistance (3/3 mmHg) exercises, and for clinical (laboratory conditions) (5/3 mmHg) and ambulatory measurements (4/2 mmHg) [15]. Cardoso et al. [16] reported ambulatory systolic BP reductions after aerobic exercise, with magnitudes varying between 2 and 12 mmHg and lasting up to 16 hours, while diastolic BP reduced between 1 and 9 mmHg and lasted up to 12 hours. Regarding resistance exercise, Cardoso et al. [16] reported systolic BP decreases varying between 3 and 12 mmHg with duration up to 10 hours, while diastolic BP reduction ranged between 3 and 7 mmHg and lasted up to 10 hours. However, at that time there were only five studies. It is important to highlight that previous studies have reported a mean reduction of 24-hour systolic BP after an aerobic exercise [14, 17], and a recent meta-analysis with 30 studies showed small-to-moderate reductions in mean 24-hour systolic/diastolic BP after an exercise session (2/1 mmHg) [18].

PEH offers to pre-hypertensive and hypertensive individuals the benefit of having their BP transiently lowered during daytime when BP usually presents its highest levels [19]. Importantly, a reduction in resting arterial pressure of 3 mmHg is associated with 8% less chance of stroke mortality and 5% less mortality from coronary heart disease [20]. Thus, the occurrence of PEH, by acutely reducing pressure, may directly lead to risk reduction in the earliest stages of an exercise training program, if exercise is performed every day. This may serve as an important bridge until chronic reductions in pressure, attributable to routine training, have occurred.

Post-exercise hypotension: a predictor of chronic blood pressure responses to exercise training

Recently, studies have demonstrated that the acute and the chronic responses of BP are well-correlated in pre-hypertensive subjects [7, 8], as shown in the Figure 1. Across patients, both systolic and diastolic BP exhibit a strong positive correlation between the fall after a single session of aerobic exercise and the reduction observed after 8 weeks of aerobic training. Other studies have reported similar associations in healthy adult women [9], elderly hypertensive women [21, 22], and patients with coronary artery disease [23]. Similar results have also been reported with resistance [9,

21] and concurrent resistance and aerobic exercises [22]. One exception to this pattern, is that patients with chronic kidney disease, who have normal BP that does not decline in response to exercise training, exhibit PEH which is not correlated with training responses [24]. This discrepancy may be related to the low baseline BP or pharmaceutical therapy. Nonetheless, collectively these findings indicate that the acute BP response after a single session of exercise may predict the chronic effect of exercise training on BP in most relevant patient groups. In other words, individual patients presenting larger PEH would exhibit greater BP reductions after a period of training.

Clinically, this association may be useful for individualizing exercise prescription for a training program and it might help avoiding the wide variability observed between subjects [25, 26]. According to Hagberg et al. [27], exercise training fails to produce BP reductions in approximately 25% of hypertensive individuals. It is possible that acute exercise studies be able to identify responders and non-responders to a specific exercise protocol without the cost of a long-term period of training. A fast identification of such non-responders might help to redirect the treatment towards other non-pharmacological or pharmacological alternatives in these individuals. Furthermore, by monitoring the PEH response to a given acute exercise protocol, protocols might be adjusted until a promising acute effect is generated, guiding the subject toward a more effective exercise protocol for training.

*** insert the figure 1 here**

Subacute effects of exercise on blood pressure: a single brick in the wall of training adaptations

As discussed above, daily sessions of exercise might have a clinically relevant impact by reducing mean BP levels across a protracted timeline [14, 17]. But it has been suggested that these acute effects have even greater impact, by being responsible for the chronic adaptations to exercise training. Nobrega [28] applied the term temporal summation to this concept, suggesting that the repeated “sub-acute effects of exercise” (i.e. the post-exercise hypotension from individual sessions of exercise) would superimpose on each other over time, generating the benefits (i.e. adaptations) of exercise training. In this perspective, as shown in the Figure 2, PEH may be thought of as a “single brick” that when added to others (i.e. other PEH episodes) constitutes the basis of a “large building” (e.g. chronic reduction in BP). This concept reinforces the

importance of performing daily sessions of exercise for optimized BP control, since the interruption of such regularity would break the temporal summation of PEH. While this narrative is not currently supported by direct experimental evidence (no study has attempted to prevent PEH to see if that would prevent chronic adaptations to training), it may be useful in explaining to patients the benefits of committing to daily exercise.

*** Insert the figure 2 here**

Factors of influence on PEH

PEH's occurrence and magnitude may be influenced by many factors that are related to subject's or the exercise protocol's characteristics. Regarding subject's characteristics, the last meta-analysis [15] suggests a greater PEH in men when compared to women, and an inverse association between PEH and age. In addition, although normotensive and hypertensive subjects present PEH, those whom have higher BP values before exercise present a greater PEH [14, 15, 18], as well the absence of anti-hypertensive medication in hypertensives [15]. Besides hypertension, the presence of metabolic diseases, such as diabetes mellitus and obesity impairs PEH [15]. Status of training level is important, since sedentary subjects present a greater PEH than athletes [15]. Finally, some studies suggested a genetic influence in PEH, since subjects with allele I of the angiotensin enzyme conversion polymorphism present a greater PEH [29, 30], but the meta-analyses did not identify any genetic influence on PEH studies [15].

Regarding exercise protocol's characteristics, aerobic and resistance exercise has been shown to promote PEH. For aerobic exercises, longer duration and larger muscle groups involved in exercise promote a greater reduction in BP during the recovery period [15, 31, 32]. Higher intensities are also known for promoting greater PEH [14, 15, 33], although when an isocaloric exercise is performed, the difference is overcame [34]. In addition, the last meta-analysis did not find difference in PEH whether aerobic exercise is performed continuously, intermittently or increasingly [15]. Finally, recent studies have observed that BP reduction is greater after a session of exercise performed in the afternoon [35, 36] and evening [37, 38] than in the morning in comparison to pre-exercise levels. In contrast, when a control day is considered for analysis, the net effect of PEH is greater after morning than evening exercise due to the morning exercise blunts the circadian pattern of increasing BP observed in the control day beyond to decrease BP in the exercise day [38]. Regarding the last meta-analysis, it did not find

differences in PEH between exercise sessions conducted at different times of day [15]. Factors of influence in PEH after resistance exercise have been less studied, but it seems that higher volume (more exercises, larger muscle mass, more sets and repetitions) produces a greater PEH [18], while the role of exercise intensity is still not clear [39, 40].

Mechanisms of PEH

Although many studies investigated the mechanisms responsible for PEH, they are not completely understood. It is mainly due to the high variance among subject's characteristics, body position during recovery, type of exercise and other factors [4, 41]. Furthermore, it is worth noting the knowledge about mechanisms of PEH was mainly generated from aerobic exercise. The reduction of systemic vascular resistance has been frequently reported [4, 41] and attributed to a central blunting of sympathetic outflow through the reset of the operation point in baroreflex control [42], which decreases sympathetic activity to the vascular beds, facilitating vasodilation [43, 44]. Furthermore, vasoconstriction responsiveness to sympathetic activation has also been reported to be reduced after a session of exercise [45]. These cardiovascular autonomics mechanisms seem to be involved in maintenance of sustained vasodilation after a session of exercise. Additionally, local mechanism has also been attributed to, such as histamine release in active skeletal muscle acting on H1 and H2 receptors [10]. Otherwise, other vasodilators, such as prostaglandins [46], nitric oxide [47], and opioids do not seem to be involved in PEH [48], although the opioid effect is controversial [49].

Besides of these most common mechanisms, in some special cases when recovery is conducted in the seated position or in subjects who present vascular dysfunction, a decrease of cardiac output is the responsible for PEH [41]. This reduction is mainly due to a decrease of stroke volume, supported by a decrease of pre-load [17], leading to a shift of blood volume [50, 51]. The decrease in stroke volume is not compensated by the increase of heart rate, usually observed after an exercise session, probably due to an alteration in central baroreflex control [52].

Thus, based on previous explanation, exercise may reduce pre-load and stimulate vasodilation during the post-exercise period. The result of these effects is the PEH, but the main mechanism responsible for the decrease of BP may depend on factors such as subject's characteristics, type of exercise, and body position during recovery among others.

Conclusions

PEH is important beyond being an acute BP response to exercise, as it may decrease BP for many hours, reducing acute cardiovascular risk, and may also provide information about future chronic adaptations. Although this approach shows great promise as a tool to identify who is responsive to a specific intervention relative to others, the readers should be careful since studies that support this theory were mostly conducted with healthy subjects and using few types of exercises. In addition, it is important to keep in mind that many factors, related to subjects' and exercise protocols' characteristics, influence PEH occurrence, magnitude and duration. Similarly, mechanisms responsible for PEH also seem to be influenced by these characteristics.

Figure Legends

Figure 1 - Systolic and diastolic blood pressure correlation between acute and chronic effect of exercise. Adapted from Liu et al (2012) and Heckesteden et al. (2013).

Figure 2 – Illustration of temporal summation of post-exercise hypotension leading to chronic blood pressure reductions with a regular exercise program.

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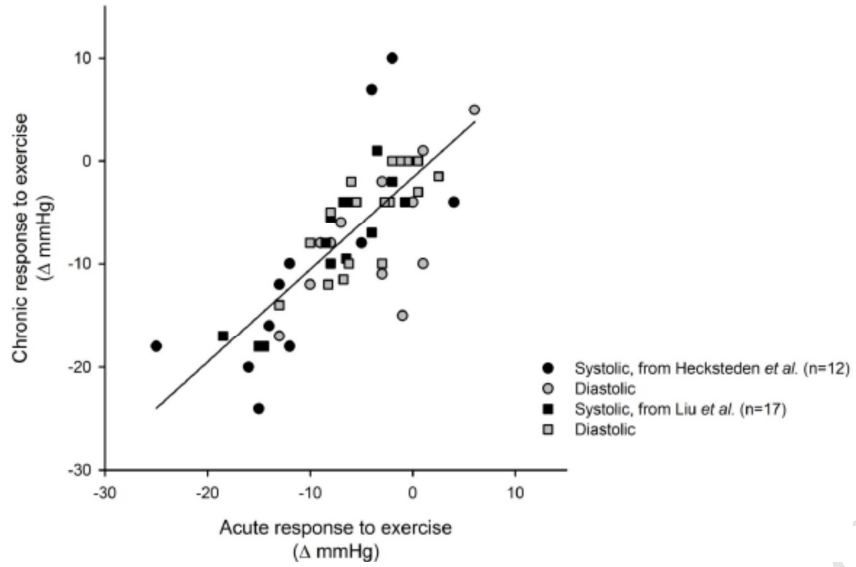

Figure 1

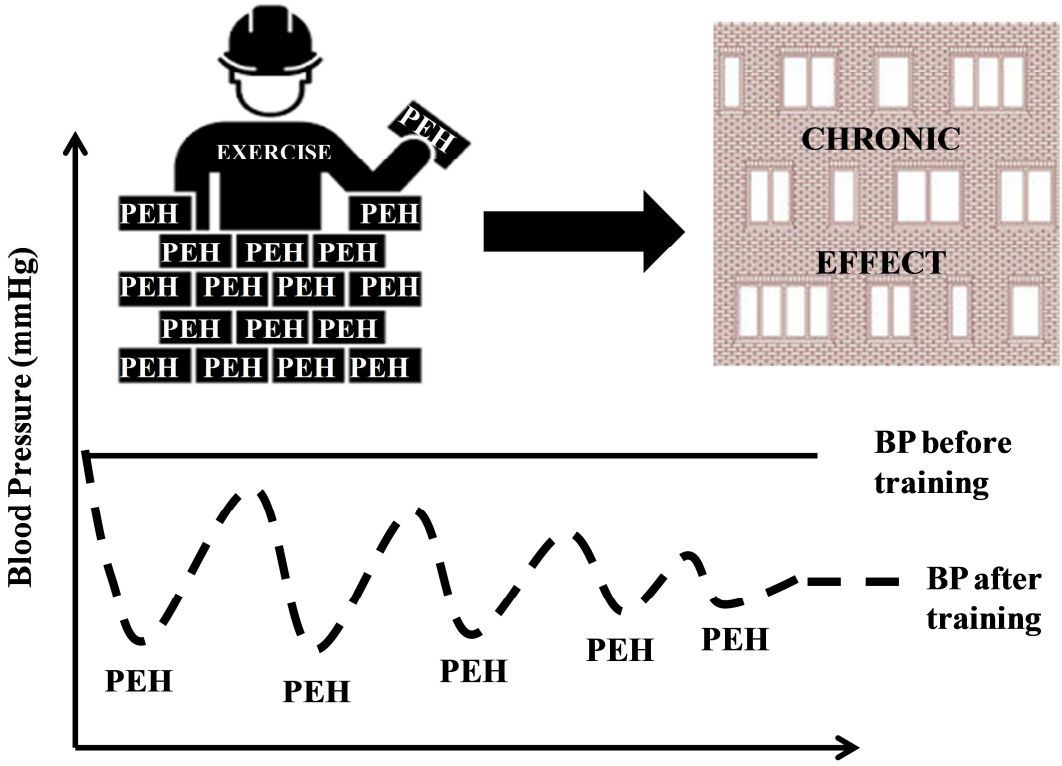

Figure 2

Post-exercise Hypotension as a Clinical Tool: A "Single Brick" in The Wall

Highlights:

1 – Post-exercise hypotension is encouraged to be used as a predictor of chronic blood pressure responses.

2 – Factors of influence have to be taken into account to use the PEH as clinical tool.