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Mechanisms and Exercise Characteristics Influencing Postexercise Hypotension

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Author's contribution

The sole author designed, analyzed, interpreted, and prepared the manuscript.

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Review Article

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ABSTRACT

Arterial blood pressure (BP) can be reduced below pre-exercise levels after a single bout of exercise. This post-exercise reduction in BP is termed "postexercise hypotension" (PEH). The aim of this review was to present current studies exploring the mechanisms of PEH and discuss potential factors influencing the magnitude and the duration of PEH. The mechanisms underlying PEH point to centrally mediated decreases in sympathetic nerve activity (due to decreased neural afferent input to the nucleus tractus solitarius and baroreceptor resetting to lower BP) and to local vasodilatory mechanisms (histamine binding to H1 and H2 receptor). The exercise characteristics (intensity, duration, and mode) and the participant characteristics (such as fitness status, baseline BP, body adiposity, gender, and hydration status) influence PEH. Earlier studies using *aerobic* exercise in normotensive and hypertensive individuals, suggested that PEH is a low-threshold event; however, most recent studies seem to agree, that at least in the first few post exercise hours, PEH is exercise intensity dependent. Studies using intermittent

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aerobic, resistance, and concurrent exercise (combined aerobic and resistance) also showed promising results on PEH. Equivocal results regarding the duration and magnitude of PEH are possibly associated with the muscle mass activated, the weight-load, and/or the number of repetitions and sets used in the different studies. Although most studies suggest that moderate to high intensity exercise induces greater PEH in normotensive and hypertensive individuals, exercise prescription should be individualized and caution should be taken in patients with multiple risk factors and chronic diseases.

Keywords: Postexercise hypotension; aerobic exercise; resistance exercise; hypertension; baroreflex; blood pressure; exercise intensity.

1. INTRODUCTION

Arterial pressure, the product of cardiac output and total peripheral resistance, can be reduced below pre-exercise levels after a single bout of moderate exercise [1]. This post-exercise reduction in blood pressure is termed “postexercise hypotension” (PEH) and can last up to a few hours. PEH occurs in normotensive as well as in hypertensive individuals [1]. Therefore, PEH could be an effective non-pharmacological strategy for individuals in prehypertensive or hypertensive states.

The extent and the duration of PEH is influenced by several factors, such as exercise type, intensity, duration, pre-exercise blood pressure levels, fitness/training status, and sex [2]. Knowledge of the factors influencing PEH is potentially useful in designing first line strategies against hypertension and providing a better understanding of blood pressure regulation during exercise in health and disease. Therefore, the aim of this review was to present potential factors influencing post-exercise hypotension and discuss controversial results.

Following a brief summary of the neural factors controlling blood pressure during exercise, this paper will provide a review of current studies exploring the mechanisms of PEH. Finally, this paper will discuss controversial results on the intensity, type, and mode of exercise affecting PEH.

2. CONTROL OF BLOOD PRESSURE DURING EXERCISE

2.1 Neural Control

The cardiovascular adjustments to exercise are controlled by the activities of “feedforward” mechanisms, such as the central command (i.e. signals from the motor cortex or subcortical nuclei responsible for recruiting motor units), and by “feedback” mechanisms, such as the arterial and cardiopulmonary baroreceptors, the chemoreceptors, and a reflex originating in the skeletal muscle, known as the “exercise pressor reflex” [3-6] (see Fig. 1).

Briefly, arterial baroreceptors, originating in the carotid sinus and the aortic arch, project in the nucleus tractus solitarius (NTS) and respond to brief changes in blood pressure. During exercise, input of baroreceptors to the NTS, causes a reset of the baroreceptors operating curve, “allowing” the rise in blood pressure. The second mechanism, the exercise pressor reflex, originates in the skeletal muscles and consists of group III and group IV afferent nerve

endings. Group III fibers respond to mechanical stimuli (changes in muscle length and pressure), whereas, group IV fibers respond mainly to alterations in the concentration of metabolites (lactic acid, H^+, diprotonated phosphate, etc) [3,6]. Integration of these neural signals occurs in the NTS. More specifically, afferent signals convey information to the caudal ventrolateral medulla (via glutaminergic synapses) and release the neurotransmitter substance P, providing an inhibitory (GABAergic) input to neurons in the rostral ventral lateral medulla (RVLM) [6-8]. These actions result in an exacerbation of the sympathetic outflow to the heart, evoking increases in heart rate, cardiac output and/or peripheral resistance [6,8]. Consequently, blood pressure and blood flow to the active muscles is enhanced. At the same time, sympathetic vasoconstriction in inactive muscles (e.g. splanchnic and renal circulations) assists in maintaining blood pressure at an acceptable level during exercise [4,9].

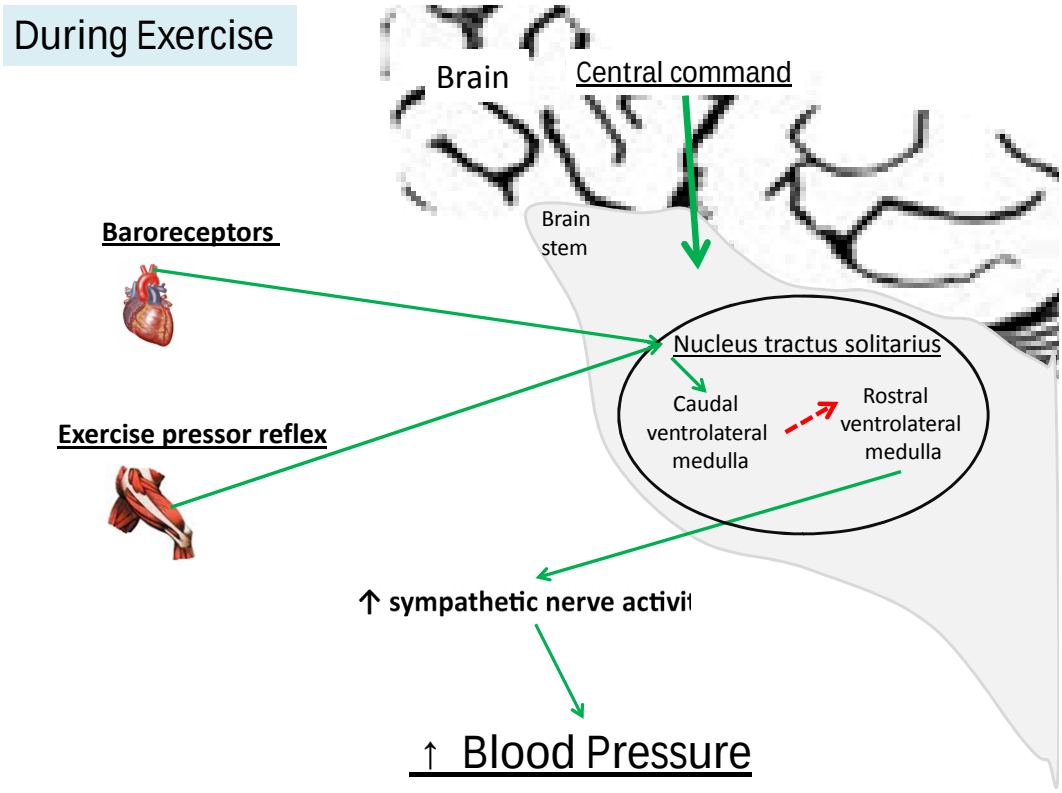

Fig. 1. A simplified illustration of the blood pressure control during exercise

Input from baroreceptors and from mechanical and metabolic fibers arising from the active muscles (the exercise pressor reflex) contact second-order neurons in the nucleus tractus solitarius (NTS). More specifically, the afferent signals convey to the caudal ventral lateral medulla (CVLM). The neuronal output from CVLM provides an inhibitory GABAergic input to the rostral ventral lateral medulla (RVLM). In turn, the output of the RVML neurons increases sympathetic nerve activity, resulting in increases in heart rate, cardiac output, and blood pressure during exercise [3,4,6-8]

2.2 Local Mechanisms

Within the active muscles, local mechanisms, such as endothelium-mediated dilation, byproducts of metabolism, erythrocyte-dependent regulation, etc, induce vasodilation, facilitating the distribution of flow to the active area [10-13].

3. MECHANISMS OF POSTEXERCISE HYPOTENSION

The mechanisms underlying postexercise hypotension point to centrally mediated decreases in sympathetic nerve activity and peripheral/local vasodilatory mechanisms. These mechanisms work independently and synergistically controlling blood pressure during and after exercise. Despite extensive research, the exact mechanisms are still not completely understood.

3.1 Neural Mechanisms

During exercise, the activation of the muscle afferents induces the release of substance P, which stimulates the GABA interneurons of the NTS and activates the neurokinin-1 receptor (NK1-R) [14]. Prolonged activation of the NK1-R during exercise, results in an internalization of the receptor [7]. At the cessation of exercise, this internalization of NK-1, diminishes the NTS GABA interneurons input [14,15] and baroreflex is resetting to lower operating levels. These actions induce an inhibition of sympathetic neurons and a reduction in total peripheral resistance [16]. The final outcome is a reduction in blood pressure after exercise [7].

3.2 Peripheral Mechanisms

In the early post-exercise phase, hypotension has been linked in part, to vasodilation induced by histamine binding to H1 and H2 receptors (located on vascular endothelial cells and vascular smooth muscle cells, respectively). Administration of an H1 receptor antagonist (fexofenadine hydrochloride) to normotensive young men and women markedly reduced vasodilation after exercise (cycling at 60% VO2peak for 1 hour) and blunted postexercise hypotension at 30min after the exercise bout [11]. However, at 60 and 90min after the exercise bout vasodilation became minimal. On the other hand, administration of an H2-receptor antagonist (ranitidine) dampen the increase in vascular conductance and blunted the decrease in mean arterial pressure at 60min after the exercise bout (cycling at 60% VO2 peak for 1 hour) [12]. These studies suggest that there is a time course for differential vasodilation, where the H1-receptor is involved in the early post exercise phase and the H2 in a more delayed and sustained phase [12]. Importantly, the postexercise H1- and H2-receptor–mediated skeletal muscle vasodilation also benefits glucose regulation in healthy humans [17], providing an additional benefit of exercise.

Other studies investigated the involvement of nitric oxide in PEH [18-21]. Although early evidence in rats suggested that there is an association of nitric oxide and postexercise vasodilation through reductions in α-adrenergic receptor sensitivity [22], studies by Halliwill and colleagues in humans did not find a direct involvement of nitric oxide synthase in PEH. More specifically, administration of a nitric oxide synthase inhibitor (L-NMMA) did not prevent postexercise hypotension indicating that PEH was not attributed to the increased nitric oxide (NO) production during exercise (60 min of upright cycling at 60% VO2peak) [23]. In contrast, data from Campell et al. showed that administration of L-NMMA attenuated the decrease in arterial stiffness after maximal exercise (assessed by femoral pulse wave velocity) [18]. Differences in the intensity (i.e. submaximal exercise vs. incremental maximal testing) and the duration of the exercise bout could partially explain the discrepant results, since the NO production has been reported to be affected by exercise intensity [20]. In addition, differences in the participant characteristics could also partially explain the equivocal results, since NO production during the postexercise recovery has been observed in individuals carrying the allele I of the angiotensin converting enzyme (ACE) gene [21].

Further studies that will examine the direct effects of NO-mediated vasodilation in PEH are needed.

The hydration status of the participant especially when exercising in a warm/hot environment and the postural position of the participant (i.e. the presence of venous pooling and reduced preload) also affect PEH [10,13,24-26]. In addition, changes in baroreflex sensitivity and/or metaboreflex function due to excess adiposity (obesity) can also affect the neural control during exercise and recovery. Dipla et al. [28] showed that obese children exhibit less vasodilation during the early postexercise recovery period than lean children [28]. The involvement of excess postexercise oxygen consumption (EPOC) to PEH has also been investigated [30]. However, data by Williams, Pricher, & Halliwill, (2005) did not find a link between PEH and EPOC [30]. Lastly, the gender effect on PEH has been examined by a few studies. These studies suggested that although PEH is evident in both males and females, the mechanisms involved in hypotension might be different. In endurance trained women hypotension during the postexercise period was depended mainly on vasodilation, whereas, in endurance trained men hypotension was the result of reductions in cardiac output. Further studies that will examine the exact mechanisms for these differences are needed. An illustrative summary of possible mechanisms influencing PEH is presented in (Fig. 2).

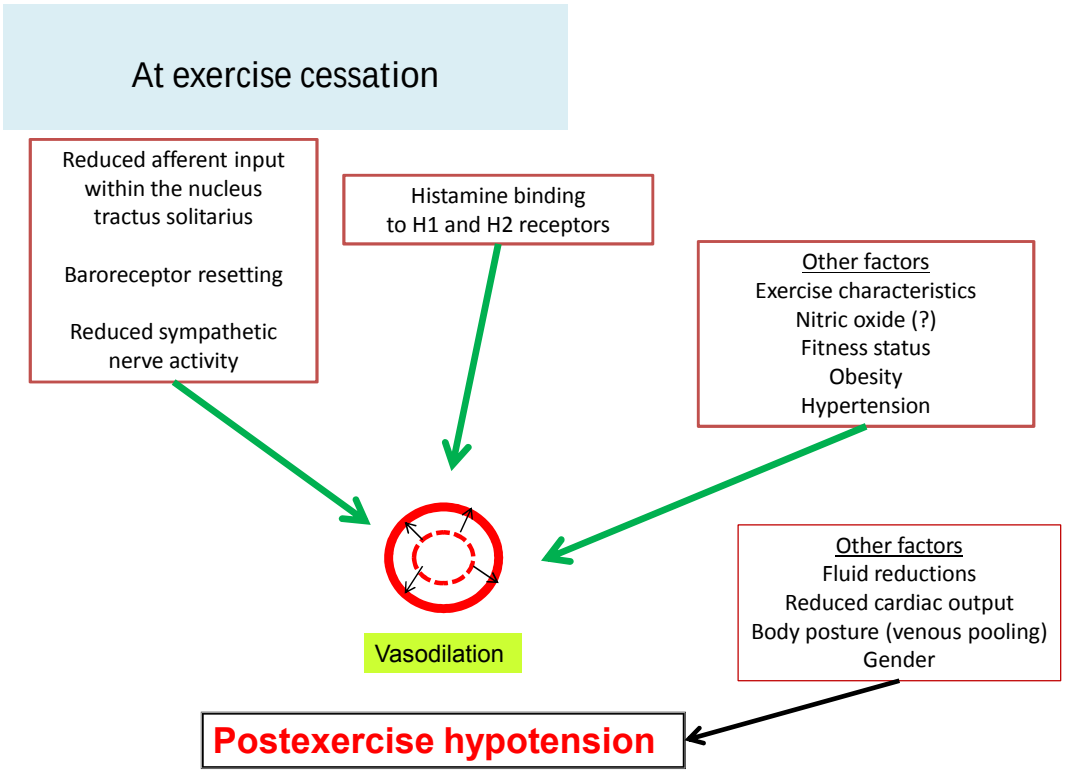

Fig. 2. Possible mechanisms influencing postexercise hypotension

During exercise recovery, decreases in sympathetic nerve activity due to decreased afferent input to the nucleus tractus solitarius (from the exercise pressor reflex) and baroreceptor resetting to lower pressure, as well as and histamine receptor activation cause vasodilation and promote hypotension. The exercise characteristics, the environmental temperature, and the participant characteristics also play a role in the post-exercise hypotension [10-12,14,16,24]

4. EXERCISE CHARACTERISTICS AND POSTEXERCISE HYPOTENSION

The exercise characteristics affect the duration and the magnitude of PEH. First, studies using aerobic exercise will be presented in this review. Results regarding the effects of exercise intensity and muscle mass involved on PEH will be discussed. Next, studies using resistance exercise will be presented, followed by studies using other exercise modes. (Table 1) summarizes the outcome of the studies.

4.1 Aerobic Exercise

4.1.1 Exercise intensity

Studies investigating the effects of exercise intensity on post-exercise hypotension have produced controversial results. Some studies report that lower levels of physical exertion can produce similar results to vigorous exercise on PEH [1,31,32], whereas, other studies support that higher exercise intensity is associated with greater reductions in blood pressure [33]. More specifically, earlier studies in hypertensive individuals reported that aerobic exercise at 30%-<60% of VO2peak was as effective as exercise at 60%-75% VO2peak over the course of nine hours post-exercise [32,34]. In addition, Marceau et al. [35] reported that when sedentary individuals with mild to moderate hypertension underwent 10 weeks of supervised exercise training at either 50% or 70% of VO2max, comparable decreases in average 24-hour blood pressure were observed. In contrast, to these studies supporting that PEH is a low-threshold event [1], Forjaz et al. [33] showed that 45min on a cycle ergometer at 30% of VO2 peak were not sufficient to induce reductions in blood pressure after exercise in normotensive individuals, whereas exercise of similar duration at 50% and 75% of VO2peak decreased significantly systolic and diastolic pressure below pre-exercise levels. The magnitude of decrease, however, was greater in the higher intensity exercise. The superiority of higher compared to lower intensity exercise on promoting PEH was also observed in mildly hypertensive older women [20]. In addition, Quinn [36] showed that high intensity exercise (at 75% VO_2peak) elicited greater blood pressure reductions than lower intensity (at 50% VO2peak) in men and women with stage I hypertension. Moreover, the participants in that study maintained the significant reductions in blood pressure for 13 and 11 hours (in systolic and diastolic pressure, respectively) after the 75% bout compared to 4 hours (in both systolic and diastolic pressure) after the 50% bout. In support of these findings, Eicher et al. [37] also showed that in males with stage I hypertension the post-exercise reduction in blood pressure was linked to aerobic exercise intensity (40%, 60%, and 100% of V02peak) in a “dose response” fashion. Santana et al. [20,21] proposed that the greater release of nitric oxide in the higher exercise intensities is possibly involved in the intensity dependent effect on PEH. However, Halliwill and colleagues did not find a direct involvement of nitric oxide synthase on PEH in humans, as previously mentioned [23].

Another factor that should be taken into consideration when discussing the effects of exercise intensity on PEH, is whether the magnitude or the duration of PEH has been investigated. Smelker et al. [38] examined whether the duration of PEH is intensity dependent in men with stage I hypertension. Participants exercised at different intensities (cycling at a power output corresponding to 70, 80, 90, and 100% of the ventilatory threshold); post-exercise blood pressure was measured at different time points. The authors reported that although PEH was evident following all exercise intensities, a dependency on exercise intensity was apparent only in the first 30 to 60min after the exercise bout, whereas, at 120min a similar reduction in blood pressure was observed in all exercise intensities.

Furthermore, Jones et al. [39] questioned that the intensity *per se* affects PEH and investigated whether the total work performed over the exercise bout is the main contributor to the magnitude of PEH. Participants in that study completed three sessions: 30min of cycling at either moderate or high intensity cycling (40% or 70% VO2peak), and cycling at 40% VO2 peak for a time required to match the total work done in the high intensity trial. The authors concluded that the acute post-exercise reduction in BP was similar following high intensity short duration exercise and moderate intensity longer duration exercise that was matched for total work performed.

4.1.2 Effects of muscle mass involved

MacDonald et al. [40] investigated the effects of muscle mass involved during aerobic exercise on PEH, in recreationally active, borderline hypertensive men. Their data showed that 30 min of arm ergometry (at 65% VO2 peak) or 30min of leg ergometry (at 70% VO2 peak) did not directly influence the magnitude of PEH, but the duration of the response was more prolonged following exercise with greater muscle mass involved (i.e. leg exercise). This effect was possibly associated with a greater histamine receptor activation following dynamic exercise involving greater muscle mass [41].

4.1.3 Intermittent vs. continuous aerobic exercise

In normotensive endurance trained men, high intensity interval exercise resulted in a similar magnitude of decrease in blood pressure to steady-state aerobic exercise (cycling) [42], although differential changes in stroke volume and total peripheral resistance were observed in the two exercise modes. Only a few studies used intermittent exercise in hypertensive individuals, since continuous aerobic exercise has been traditionally, recommended [1]. However, recent data showed that intermittent exercise (1min at 80% of heart rate reserve with 2min at 50%) exerted beneficial effects in medicated hypertensive patients, increasing the percentage of time with normal ambulatory blood pressure values over 24 hours after the exercise session [43]. A study by Lacombe et al. [44] also showed that older prehypertensive adults experienced similar PEH following equicaloric bouts of intermittent (5×2:2min at 85% and 40% of VO2 peak) and steady state (21min at 60% maximal oxygen uptake) exercise, despite differences in the regulation of blood pressure during exercise between the two exercise modes (i.e. larger alterations in heart rate variability and baroreceptor sensitivity elicited by the intermittent exercise) Future studies should, however, examine the effects of isoeffort or isostrain exercise when comparing the effects of intermittent and steady state exercise, to isolate the effects of the exercise mode (continuous vs. intermittent) from those of the effects of exercise intensity.

4.2 Aerobic vs. Resistance Exercise

Various studies have been conducted to clarify the relationship between the type of exercise and the magnitude of PEH. Most studies exploring PEH, however, used aerobic exercise, since the occurrence of PEH following resistance exercise was initially controversial [1]. The two exercise modalities are characterized by distinct acute cardiovascular effects and differential flow patterns. Aerobic and dynamic exercise is characterized by a widened pulse pressure (increased systolic and minimal changes in diastolic blood pressure), an increase in cardiac output, and a decrease in total peripheral resistance. In contrast, static (isometric) exercise induces large increases in intramuscular pressure, resulting in decreased blood flow to the active skeletal muscle. In an attempt to maintain perfusion, the exercise pressor reflex is activated, and sympathetic tone increases, resulting in marked increases in blood

pressure. The different mechanical and metabolic responses between aerobic and resistance exercise result in different chronic adaptations. Endurance exercise training results in improved muscle oxidative capacity and an increase in capillarization (via increases in angiogenetic peptides, such as the vascular endothelial growth factor (VEGF), whereas resistance exercise training leads to increases in muscle size and strength and increased insulin sensitivity [45-47].

Most studies using resistance exercise, showed promising results on PEH following an acute exercise session. Equivocal results regarding the duration and magnitude of PEH, are possibly associated with the exercise protocol used, the muscle mass activated, the weight-load, and the number of repetitions and sets used in the different studies. In trained normotensive men, high-volume multiple-set resistance training sessions were required to induce PEH [48]. More specifically, when either arm or leg exercise were performed, using 6 or 10 sets of 10 repetitions at a 12RM workload, an hour after exercise, PEH was observed only after 10 sets of leg exercise [48]. When a work-matched low-intensity (at 20-40% of 1-RM) and a high-intensity (70-80% of 1-RM) resistance exercise bout were used, higher intensity was required to induce PEH in normotensive young men [49,50]. The effectiveness of high-intensity (80% of 1-RM) over low-intensity (50% of 1-RM) resistance exercise was also demonstrated in a recent study performed in hypertensive individuals [51]. High intensity resistance exercise of sufficient duration (approximately 25 min), has also been demonstrated as more efficient at promoting PEH in normotensive middle-aged individuals with type II diabetes [52] than low intensity training. There is evidence, however, of low intensity resistance exercise (40% of 1-RM) being at least as effective as high intensity resistance exercise (80% of 1-RM) in promoting PEH in normotensive individuals [53].

Despite the promising results of resistance exercise on PEH, studies in hypertensive individuals are limited. Mota et al. [54] compared the effects of 20min treadmill running (at 70–80% of heart rate reserve) vs. 20 min of resistance exercise (20 repetitions at 40% of 1 RM), on the blood pressure responses in hypertensive individuals (40-44 years old) treated with antihypertensive medication. The authors observed a hypotensive response after both exercise modalities during the 7 postexercise hours (free-living). The highest reduction in mean arterial pressure was observed at 30 and 45min of recovery. The reduction in systolic pressurε was longer after aerobic than resistance exercise. However, resistance exercise evoked lower diastolic pressure values than aerobic at the early post exercise phase (15min of recovery).

The effects of duration of rest intervals on PEH during strength training has been examined by de Salles et al. [55] using 3 sets of 10 repetitions per exercise at 70% of 10RM, with 1 vs. 2 minutes' rest interval between sets. The investigators proposed that longer duration intervals were associated with a greater PEH at 60 min after the session in older men (64-69 years). In contrast, when the effects of active vs. passive intervals between exercise series (3 sets, 15 repetitions, 60% load of 15 repetition maximum (RM), with an interval of 90 s between sets) [56] on PEH were investigated in hypertensive women (60-65 years), active intervals resulted in significantly higher PEH.

The fitness/training status of the participants and the pre-exercise blood pressure levels also seem to affect the PEH response [57]. Moraes et al. [58] showed that when the stimulus is new, an acute moderate-intensity session with three sets of 12 repetitions (at 60% of 1RM, seven exercises), can result in a reduction of blood pressure by an average of approximately 22/8mm Hg (systolic/diastolic arterial pressure, respectively); however, this exercise load is

not sufficient to induce PEH after 12 weeks of training in stage 1 hypertensive patients not using antihypertensive medication.

4.3 Concurrent Exercise and Other Exercise Modalities

Keese et al. [59] showed that a concurrent exercise session (6 resistance exercises of 2 sets at 80% 1RM, plus 20 minutes of cycle ergometer at 65% of VO2peak) was as effective as an aerobic session of similar duration (50min on a cycle ergometer at 65% of VO2peak). However, the aerobic session and the concurrent exercise session exhibited decreases in systolic pressure that lasted longer (120 vs. 80min) than a session of resistance exercise alone (8 exercises, 3 sets at 80% of 1RM). In agreement with these results, Teixeira et al. [60] did not find a potentiating effect of concurrent exercise (30min, cycle ergometer at 75% of VO2 peak plus resistance exercise involving 3 sets of 6 exercises, 20 repetitions at 50% of 1RM) in PEH compared to aerobic exercise only. However, both aerobic and concurrent sessions induced greater PEH than resistance exercise alone.

Again, the intensity of the concurrent training is an important factor that should be taken into consideration, when planning an exercise session to induce greater hypotensive benefits. Keese et al. [61] investigated the effect of intensity of the concurrent exercise in normotensive young men (2 sets of 6 exercises at 80% 1RM followed by 30 min of cycle ergometer exercise at 50%, 65%, and 80% of VO2 peak). Their results showed that although the magnitude of systolic blood pressure reduction was similar after all concurrent exercise sessions, PEH lasted approximately 1h longer following the higher intensity sessions.

Results from studies using other exercise modalities such as water exercise are limited. In normotensive untrained women an acute session of chest-deep water- or land-walking (at 40% of VO2 peak for 45 minutes) induced decreases in systolic and diastolic pressure, whereas, in trained normotensive women, the effects on PEH were less pronounced [62]. In individuals with prehypertension and hypertension the magnitude of PEH during free-living conditions was not different after an acute session of land exercise and water exercise, however, the duration of PEH was longer after the land exercise than the water exercise (24h vs. 9h, respectively) [63].

Table 1. The effects of single bouts of aerobic exercise (AE), resistance exercise (RE) and concurrent exercise (CE, aerobic and resistance exercises) protocols on post-exercise hypotension (PEH)

Study	Participants	Exercise protocol	Effects on post-exercise hypotension
MacDonald J et al. [31]	10 active normotensives	AE: 30-min cycling at 50% and 75% VO_{2peak}	PEH after both protocols; No effect of exercise intensity
Pescatello LS et al. [32]	6 mildly hypertensive men and 6 normotensives	AE: 30-min cycling at 40% and 70% of VO_{2peak}	In Hypertensives: PEH after both protocols. No effect of exercise intensity; In Normotensives: No PEH
Forjaz CL et al. [2]	23 normotensives	AE: 45-min cycling at 30%, 50%, and 75% of VO_{2peak}	PEH after 50 and 75% of VO_{2peak}; Greater and longer PEH after more intense exercise

Study	Participants	Exercise protocol	Effects on post-exercise hypotension
Santana HA et al. [20]	23 elderly mildly hypertensive women	AE: High-intensity exercise AE: Moderate-intensity 20 min exercise at 90% of VT	PEH after both protocols; Effect of exercise intensity on PEH
Pescatello LS et al. [34]	49 men with high normal to stage 1 hypertension	AE: Cycling at 40% and 60% of VO_{2peak}	PEH after both protocols; At 5h post-exercise: PEH greater in 60% vs. 40%; At 9h post-exercise: No effect of intensity
Quinn TJ et al. [36]	16, stage 1 and 2 non-medicated hypertensive men and women and 16 normotensive	AE: 30-min cycling at 50% and 75% of VO_{2peak}	In Hypertensives: PEH after both protocols. Greater and longer PEH after 75% vs. 50%; In Normotensives: No PEH after both protocols
Eicher JD et al. [37]	45 men (44 yrs. old) with BP 145/85	AE: Cycling at 40%, 60%, and 100% of VO_{2peak}	Less ↑ in ambulatory SBP and greater ↓ in ambulatory DBP after protocols; Effect of exercise intensity on PEH
Smelker CL et al. [38]	10 subjects with stage 1 hypertension	AE: 25-min cycling at 70%, 80%, 90%, and 100% of VT	PEH after all protocols; Significant effect of exercise intensity on PEH
Jones H et al. [39]	7 normotensive physically active males (28 yrs. old)	AE: 30-min cycling at 40% and 70% of VO_{2peak}, and at 40% with same total work to 70%	PEH *only* after 40% and 70% protocols matched for work; Effects of exercise intensity and total work
MacDonald JR et al. [40]	9 recreationally active, borderline hypertensives	AE: 30-min arm-cranking at 65% VO_{2peak} and 30-min cycling at 70% VO_{2peak}	PEH after both protocols; No effect of exercise modality on magnitude of PEH; Effect of exercise modality on PEH duration
Rossow L et al. [42]	25 endurance-trained men and women	AE: High-intensity interval AE: Steady-state aerobic	PEH after both protocols; No effect of exercise protocol on PEH magnitude; No gender effect on PEH, irrespective of protocol
Ciolac EG et al. [43]	52 treated hypertensives	AE:Continuous exercise (60% of HR reserve) AE: Interval exercise (2 min at 50% and 1 min at 80% of HR reserve)	PEH after Continuous exercise; PEH after Interval exercise (↓ SBP and tended to ↓ DBP)
Lacombe SP et al. [44]	13 pre-hypertensive men	AE: Continuous exercise (21-min at 60% VO_{2peak}) AE: Interval exercise (5 reps of 2-min at 85%	PEH after both protocols; No effect of exercise protocol on PEH magnitude

Study	Participants	Exercise protocol	Effects on post-exercise hypotension
		VO_{2peak} with 2-min 40% VO_{2peak})	
Liu S et al. [57]	17 young (28 yrs. old) and 18 middle-aged (52 yrs. old)	AE: 120-min at 60% VO_{2peak} AE: 120-min at 80% VO_{2peak}	PEH after both protocols; No effect of exercise protocol on PEH magnitude
Polito MD et al. [48]	24 trained men (23 yrs. old)	RE: Arm exercise, 6 sets of 10 reps at 12RM RE: Leg exercise, 10 sets of 10 reps at 12RM	Arm exercise: No PEH; Leg exercise: PEH only with 10 sets; Effect of muscle mass on PEH
Rossow LM et al. [49]	10 young, normotensive men	RE: 20% 1RM RE: 70% 1RM RE: 20% 1RM, BF restricted	PEH occurred only after high-intensity resistance exercise
Duncan MJ et al. [50]	16 resistance-trained men (23 yrs. old)	RE: 40% 1RM RE: 80% 1RM	PEH (↓ SBP) occurred only after high-intensity resistance exercise
Brito EF et al. [51]	10 hypertensive elderly individuals (65 yrs. old)	RE: 50% 1RM RE: 80% 1RM	↑ PEH and forearm vascular resistance after high-intensity vs. low-intensity resistance exercise
Simoes GC et al. [52]	Middle-aged type II diabetics and nondiabetics	RE: 3×16 reps, 43% 1RM RE: 3×30 reps, 23% 1RM	PEH after 43% of 1RM; No PEH after 23% 1RM; Effect of exercise intensity on PEH
Rezk CC et al. [53]	17 normotensives	RE: 40% 1RM RE: 80% 1RM	In low-intensity: PEH; In high-intensity: PEH (↓SBP and ↔DBP)
Mota MR et al. [54]	15 medicated hypertensives (43 yrs. old)	AE: 20-min, 70-80% of HR reserve) RE: 20 reps, 40% 1RM	PEH (↓SBP and DBP) after both protocols
de Salles BF et al. [55]	17 normotensive older men (68 yrs. old)	RE: 3×10 reps, 70% 10RM with 1 and with 2 min rest interval	PEH after both protocols; Greater PEH magnitude for the 2-min rest protocol
Brito AF et al. [56]	21 elderly hypertensive women (61 yrs. old)	RE: Arm 3 × 15 reps at 60% of 15RM with active and passive rest RE: Leg 3 × 15 reps at 60% of 15RM with active and passive rest	In leg exercises: Greater PEH in active vs. passive rest (↓SBP and DBP); In arm exercises: Greater PEH in active vs. passive rest (↓SBP only)
Keese F et al. [59]	21 healthy men (21 yrs. old)	RE: 3 sets at 80% 1RM AE: 50-min, 65% VO_{2peak} CE: Resistance and	↑ duration of PEH after AE and CE vs. RE; No differences in PEH after AE and CE

Study	Participants	Exercise protocol	Effects on post-exercise hypotension
		aerobic	
Teixeira L et al. [60]	20 healthy individuals	RE: 3 × 20 reps at 50% 1RM AE: 30-min, 75% VO_{2peak} CE: Resistance and aerobic	PEH (↓SBP and DBP) after all protocols; ↑ magnitude of PEH (↓SBP) after AE and CE vs. RE; ↑ duration of PEH (↓DBP) after AE vs. CE and RE
Keese F et al. [61]	21 healthy men (21 yrs. old)	CE: Three protocols of CE (2 sets for 6 exercises at 80% 1RM and 30-min cycling at 50%, 65%, or 80% of VO_{2peak})	PEH after all CE protocols; ↑ PEH in CE protocol with higher intensity of aerobic exercise
Rodriguez D et al. [62]	23 untrained (n = 12) and trained (n = 11) normotensive women	45-min walking at 40% of VO_{2peak} in water and on land	Untrained: PEH (↓SBP, DBP, and MAP) after water walking, but no PEH after land walking; Trained: PEH (↓SBP only) after water and land walking; Water walking promotes PEH
Terblanche E et al. [63]	21 men and women (52 yrs. old)	Water exercise Land exercise	PEH after both water and land protocols; The magnitude of PEH is similar for land and water exercises; The duration of PEH longer for land exercise

↓=decrease; ↑=increase; ↔=no change; VO_{2peak}=peak oxygen consumption; PEH=post-exercise hypotension; SBP=systolic blood pressure; DBP=diastolic blood pressure; MAP=mean arterial pressure; HR=heart rate; VT=ventilatory threshold; RM=repetition maximum; BF=blood flow; RE=resistance exercise; AE=aerobic exercise; CE=combined (aerobic and resistance) exercise

5. CONCLUSION AND RECOMMENDATIONS

Summarizing, although PEH has been observed after aerobic exercise performed at intensities ranging from 40% to>70% of VO2 peak, most studies seem to agree that at least in the first few post exercise hours PEH is intensity dependent. Future studies should be conducted to further unravel the exact mechanisms of the intensity dependency of postexercise hypotension. Hypotensive effects of exercise performed around the anaerobic threshold appears to be more effective. Concurrent exercise, as well as resistance exercise of moderate to high intensity and greater muscle mass involved show promising results on PEH. However, exercise prescription should be individualized and caution should be taken in patients with multiple risk factors and other chronic diseases.

CONSENT

Not applicable.

ETHICAL APPROVAL

Not applicable.

COMPETING INTERESTS

Author has declared that no competing interests exist.

REFERENCES

1. 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(3):533-53.
2. 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(5-6):255-62.
3. Rowell LB. Reflex control of the circulation during exercise. Int J Sports Med. 1992;13(Suppl 1):25-7.
4. Rowell LB, O'Leary DS. Reflex control of the circulation during exercise: Chemoreflexes and mechanoreflexes. J Appl Physiol. 1990;69(2):407-18.
5. Fadel PJ. Dynamic arterial baroreflex function during high intensity exercise in humans: Insights into sympathetic control. J Physiol. 2008;586(Pt 11):2667-8.
6. Mitchell JH, Kaufman MP, Iwamoto GA. The exercise pressor reflex: Its cardiovascular effects, afferent mechanisms, and central pathways. Annu Rev Physiol. 1983;45:229-42.
7. Chen CY, Bechtold AG, Tabor J, Bonham AC. Exercise reduces GABA synaptic input onto nucleus tractus solitarii baroreceptor second-order neurons via NK1 receptor internalization in spontaneously hypertensive rats. J Neurosci. 2009;29(9):2754-61.
8. Potts JT. Exercise and sensory integration. Role of the nucleus tractus solitarius. Ann N Y Acad Sci. 2001;940(221-36.
9. Pricher MP, Holowatz LA, Williams JT, Lockwood JM, Halliwill JR. Regional hemodynamics during postexercise hypotension. I. Splanchnic and renal circulations. J Appl Physiol. 2004;97(6):2065-70.
10. Halliwill JR, Sieck DC, Romero SA, Buck TM, and Ely MR. Blood pressure regulation X: What happens when the muscle pump is lost? Post-exercise hypotension and syncope. Eur J Appl Physiol. 2014;114(3):561-78.
11. Lockwood JM, Wilkins BW, and Halliwill JR. H1 receptor-mediated vasodilatation contributes to postexercise hypotension. J Physiol. 2005;563(Pt 2):633-42.
12. McCord JL, Beasley JM, Halliwill JR. H2-receptor-mediated vasodilation contributes to postexercise hypotension. J Appl Physiol. 2006;100(1):67-75.
13. McCord JL, Pellinger TK, Lynn BM, and Halliwill JR. Potential benefit from an H1-receptor antagonist on postexercise syncope in the heat. Med Sci Sports Exerc. 2008;40(11):1953-61.
14. Kajekar R, Chen CY, Mutoh T, and Bonham AC. GABA(A) receptor activation at medullary sympathetic neurons contributes to postexercise hypotension. Am J Physiol Heart Circ Physiol. 2002;282(5):H1615-24.

15. Schreihofer AM, Guyenet PG. The baroreflex and beyond: control of sympathetic vasomotor tone by GABAergic neurons in the ventrolateral medulla. Clin Exp Pharmacol Physiol. 2002;29(5-6):514-21.
16. Kulics JM, Collins HL, DiCarlo SE. Postexercise hypotension is mediated by reductions in sympathetic nerve activity. Am J Physiol. 1999;276(1 Pt 2):H27-32.
17. Pellinger TK, Dumke BR, Halliwill JR. Effect of H1- and H2-histamine receptor blockade on postexercise insulin sensitivity. Physiol Rep. 2013;1(2):e00033.
18. Campbell R, Fisher JP, Sharman JE, McDonnell BJ, Frenneaux MP. Contribution of nitric oxide to the blood pressure and arterial responses to exercise in humans. J Hum Hypertens. 2011;25(4):262-70.
19. Krause M, Rodrigues-Krause J, O'Hagan C, Medlow P, Davison G, Susta D, et al. The effects of aerobic exercise training at two different intensities in obesity and type 2 diabetes: implications for oxidative stress, low-grade inflammation and nitric oxide production. Eur J Appl Physiol. 2014;114(2):251-60.
20. Santana HA, Moreira SR, Asano RY, Sales MM, Cordova C, Campbell CS, et al. Exercise intensity modulates nitric oxide and blood pressure responses in hypertensive older women. Aging Clin Exp Res. 2013;25(1):43-8.
21. Santana HA, Moreira SR, Neto WB, Silva CB, Sales MM, Oliveira VN, et al. The higher exercise intensity and the presence of allele I of ACE gene elicit a higher post-exercise blood pressure reduction and nitric oxide release in elderly women: An experimental study. BMC Cardiovasc Disord. 2011;11:71.
22. Patil RD, DiCarlo SE, Collins HL. Acute exercise enhances nitric oxide modulation of vascular response to phenylephrine. Am J Physiol. 1993;265(4 Pt 2):H1184-8.
23. Halliwill JR, Minson CT, Joyner MJ. Effect of systemic nitric oxide synthase inhibition on postexercise hypotension in humans. J Appl Physiol. 2000;89(5):1830-6.
24. Halliwill JR, Buck TM, Lacewell AN, Romero SA. Postexercise hypotension and sustained postexercise vasodilatation: what happens after we exercise? Exp Physiol. 2013;98(1):7-18.
25. 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(5):e99-e107.
26. Dujic Z, Ivancev V, Valic Z, Bakovic D, Marinovic-Terzic I, Eterovic D, et al. Postexercise hypotension in moderately trained athletes after maximal exercise. Med Sci Sports Exerc. 2006;38(2):318-22.
27. Dipla K, Nassis GP, Vrabas IS. Blood pressure control at rest and during exercise in obese children and adults. J Obes. 2012;2012:147-385.
28. Dipla K, Zafeiridis A, Koidou I, Geladas N, Vrabas IS. Altered hemodynamic regulation and reflex control during exercise and recovery in obese boys. Am J Physiol Heart Circ Physiol. 2010;299(6):H2090-6.
29. Legantis CD, Nassis GP, Dipla K, Vrabas IS, Sidossis LS, Geladas ND. Role of cardiorespiratory fitness and obesity on hemodynamic responses in children. J Sports Med Phys Fitness. 2012;52(3):311-8.
30. Williams JT, Pricher MP, Halliwill JR. Is postexercise hypotension related to excess postexercise oxygen consumption through changes in leg blood flow? J Appl Physiol. 2005;98(4):1463-8.
31. MacDonald J, MacDougall J, Hogben C. The effects of exercise intensity on post exercise hypotension. J Hum Hypertens. 1999;13(8):527-31.
32. Pescatello LS, Fargo AE, Leach CN Jr., and Scherzer HH. Short-term effect of dynamic exercise on arterial blood pressure. Circulation. 1991;83(5):1557-61.

33. 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(1):54-62.
34. Pescatello LS, Guidry MA, Blanchard BE, Kerr A, Taylor AL, Johnson AN, et al. Exercise intensity alters postexercise hypotension. J Hypertens. 2004;22(10):1881-8.
35. Marceau M, Kouame N, Lacourciere Y, Cleroux J. Effects of different training intensities on 24-hour blood pressure in hypertensive subjects. Circulation. 1993;88(6):2803-11.
36. Quinn TJ. Twenty-four hour, ambulatory blood pressure responses following acute exercise: impact of exercise intensity. J Hum Hypertens. 2000;14(9):547-53.
37. Eicher JD, Maresh CM, Tsongalis GJ, Thompson PD, Pescatello LS. The additive blood pressure lowering effects of exercise intensity on post-exercise hypotension. Am Heart J. 2010;160(3):513-20.
38. Smelker CL, Foster C, Maher MA, Martinez R, Porcari JP. Effect of exercise intensity on postexercise hypotension. J Cardiopulm Rehabil. 2004;24(4):269-73.
39. Jones H, George K, Edwards B, and Atkinson G. Is the magnitude of acute post-exercise hypotension mediated by exercise intensity or total work done? Eur J Appl Physiol. 2007;102(1):33-40.
40. MacDonald JR, MacDougall JD, Hogben CD. The effects of exercising muscle mass on post exercise hypotension. J Hum Hypertens. 2000;14(5):317-20.
41. Barrett-O'Keefe Z, Kaplon RE, Halliwill JR. Sustained postexercise vasodilatation and histamine receptor activation following small muscle-mass exercise in humans. Exp Physiol. 2013;98(1):268-77.
42. Rossow L, Yan H, Fahs CA, Ranadive SM, Agiovlasitis S, Wilund KR, et al. Postexercise hypotension in an endurance-trained population of men and women following high-intensity interval and steady-state cycling. Am J Hypertens. 2010;23(4):358-67.
43. Ciolac EG, Guimaraes GV, D Avila VM, 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(3):381-7.
44. 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(6):881-91.
45. Kon M, Ohiwa N, Honda A, Matsubayashi T, Ikeda T, Akimoto T, et al. Effects of systemic hypoxia on human muscular adaptations to resistance exercise training. Physiol Rep. 2014;2(6).
46. Hudlicka O, Brown M, Egginton S. Angiogenesis in skeletal and cardiac muscle. Physiol Rev. 1992;72(2):369-417.
47. McDonagh MJ, and Davies CT. Adaptive response of mammalian skeletal muscle to exercise with high loads. Eur J Appl Physiol Occup Physiol. 1984;52(2):139-55.
48. Polito MD, and Farinatti PT. The effects of muscle mass and number of sets during resistance exercise on postexercise hypotension. J Strength Cond Res. 2009;23(8):2351-7.
49. Rossow LM, Fahs CA, Sherk VD, Seo DI, Bemben DA, and Bemben MG. The effect of acute blood-flow-restricted resistance exercise on postexercise blood pressure. Clin Physiol Funct Imaging 2011;31(6):429-34.
50. Duncan MJ, Birch SL, Oxford SW. The effect of exercise intensity on postresistance exercise hypotension in trained men. J Strength Cond Res. 2014;28(6):1706-13.
51. Brito Ade F, de Oliveira CV, Santos Mdo S, and Santos Ada C. High-intensity exercise promotes postexercise hypotension greater than moderate intensity in elderly hypertensive individuals. Clin Physiol Funct Imaging. 2014;34(2):126-32.

52. Simoes GC, Moreira SR, Kushnick MR, Simoes HG, Campbell CS. Postresistance exercise blood pressure reduction is influenced by exercise intensity in type-2 diabetic and nondiabetic individuals. J Strength Cond Res. 2010;24(5):1277-84.
53. 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(1):105-12.
54. Mota MR, Pardono E, Lima LC, Arsa G, Bottaro M, Campbell CS, et al. Effects of treadmill running and resistance exercises on lowering blood pressure during the daily work of hypertensive subjects. J Strength Cond Res. 2009;23(8):2331-8.
55. de Salles BF, Maior AS, Polito M, Novaes J, Alexander J, Rhea M, et al. Influence of rest interval lengths on hypotensive response after strength training sessions performed by older men. J Strength Cond Res. 2010;24(11):3049-54.
56. Brito AF, Alves NF, Araujo AS, Goncalves MC, Silva AS. Active intervals between sets of resistance exercises potentiate the magnitude of postexercise hypotension in elderly hypertensive women. J Strength Cond Res. 2011;25(11):3129-36.
57. Liu S, Thomas SG, Sasson Z, Banks L, Busato M, Goodman JM. Blood pressure reduction following prolonged exercise in young and middle-aged endurance athletes. Eur J Prev Cardiol. 2013;20(6):956-62.
58. Moraes MR, Bacurau RF, Simoes HG, Campbell CS, Pudo MA, Wasinski F, et al. Effect of 12 weeks of resistance exercise on post-exercise hypotension in stage 1 hypertensive individuals. J Hum Hypertens. 2012;26(9):533-9.
59. Keese F, Farinatti P, Pescatello L, Monteiro W. A comparison of the immediate effects of resistance, aerobic, and concurrent exercise on postexercise hypotension. J Strength Cond Res. 2011;25(5):1429-36.
60. Teixeira L, Ritti-Dias RM, Tinucci T, Mion Junior D, and Forjaz CL. Post-concurrent exercise hemodynamics and cardiac autonomic modulation. Eur J Appl Physiol. 2011;111(9):2069-78.
61. 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(2):148-53.
62. Rodriguez D, Silva V, Prestes J, Rica RL, Serra AJ, Bocalini DS, et al. Hypotensive response after water-walking and land-walking exercise sessions in healthy trained and untrained women. Int J Gen Med. 2011;4:549-54.
63. Terblanche E, Millen AM. The magnitude and duration of post-exercise hypotension after land and water exercises. Eur J Appl Physiol. 2012;112(12):4111-8.

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