A Review on Post-exercise Hypotension in Hypertensive Individuals

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Abstract

Post-exercise hypotension (PEH) may follow a session of physical exercises. This effect has high clinical relevance for hypertensive individuals. Although there are several studies on the subject, an analysis is still lacking on a state-of-art approach considering different types of exercises.

Using a review of literature, the aim of this paper was to verify the relationship between aerobic exercise and resistance exercise in PEH in hypertensive people.

For the purpose of this study, Scielo and Medline databases were surveyed, and the main inclusion criteria were studies on the subject in English language and a sample of hypertensive adults. One hundred and twenty-six studies were found. However, only 32 papers, 5 of which on resistance exercise and 27 on aerobic exercise were used for this study. Although the studies surveyed used different prescription models for resistance exercise, PEH was observed mainly in the laboratory setting. After aerobic exercise, it was noted that PEH occurred for longer periods. However, there are differences of opinion as to the best intensity and duration of the exercise to be prescribed.

Therefore, in hypertensive individuals declines in arterial pressure following aerobic exercise are apparently greater than those observed with resistance exercise. Nevertheless, in order to achieve sounder conclusions, further studies on ambulatory blood pressure monitoring should be conducted.

Introduction

Arterial hypertension (AH) affects a significant part of the adult population worldwide1, and is a risk factor for heart diseases and renal failure2. Lifestyle changes, such as reducing alcohol consumption and tobacco smoking, developing adequate eating habits, and controlling body weight, are suggested as non-drug measures for preventing and treating AHF3,4. Engaging in regular exercise is also recommended as a means to reduce resting blood pressure (BP) values4.

Regardless of the potential reduction in resting BP levels as a result of regular exercising (chronic effect), these may drop to below resting values in the minutes following a bout of exercises, which is termed post-exercise hypotension (PEH)5. One of the first scientific data on the subject was described in 1981 by William Fitzgerald6, whose BP systematically declined after aerobic exercise training (25-minute jogging at 70% of the maximal heart rate).

PEH can be considered an important strategy to help control resting BP, especially in hypertensive individuals7. Aerobic physical exercise is the most commonly surveyed and recommended modality to promote PEH in hypertensive and normotensive individuals8. However, data on the effects of the duration and intensity of exercise in hypertensive subjects are still controversial.

Besides aerobic exercising, resistance training is also recommended for hypertensive individuals as a form of increasing muscle strength9. However, although showing promising results, research on the occurrence of PEH in hypertensive individuals engaged in this type of exercise is scarce10-12. Moreover, there are differences in experimental protocols, such as weight load, number of repetitions, and quantity of exercises.

Thus, considering the potential gaps identified in understanding the effect of diverse exercises on PEH in hypertensive individuals, it is of utmost importance to investigate the subject. Within this context, the aim of this study was to review literature on the effects of aerobic and resistance exercises on PEH in hypertensive individuals. Additionally, physiological mechanisms involved were presented and commented on.

Search methods and inclusion of studies

Two independent researchers surveyed Medline and Scielo databases. To search references on aerobic exercise in the Medline database, the terms dynamic exercise and aerobic exercise were used separately and associated with hypotension, hypertension, post-exercise, postexercise, blood pressure, and acute response in the title or in the abstract. To search references on resistance exercise, the terms resistance exercise and strength exercise were used separately, followed by the terms cited above. Investigation in the Scielo database was done using the terms described separately in the title.

Inclusion criteria for the articles were: 1) papers reporting arterial BP responses to aerobic or resistance exercise for a minimum of 30 minutes; 2) duration of exercise training equal to or more than 15 minutes (for aerobic exercise); 3) papers published in English up to September 2009; 4) a sample consisting of hypertensive adults, i.e., mean BP of the study group equal to or greater than 140 mmHg for systolic blood pressure (SBP) or 90 mmHg for diastolic blood pressure (DBP)2.
After filtering by the keywords, 126 studies remained. Of these, the following papers were excluded: experiments on the effects of chronic exercise, animal models, BP responses during exercise, samples consisting of subjects with normotension/borderline hypertension, orthostatic tolerance, literature review, and exercise duration under 15 minutes (Figure 1). Thus, this study consisted of 32 reference papers, 5 of which addressing resistance exercise (a sample of 72 individuals), and 27 addressing aerobic exercises (a sample of 639 individuals). Table 1 displays the main findings of the studies on resistance exercises, whereas Table 2 shows the main findings on aerobic exercises.

**Hypotension following resistance exercises**

A significant difference was observed among the protocols for resistance exercises. Variation was relative to: performance (conventional\(^{8,10,12}\) and circuit-training\(^{9,11}\)); intensity (ranging from low\(^{8,9,11}\) to high\(^{10,12}\)), number of repetitions (between 8 and 20), and duration of the rest interval (between 30 and 120 seconds). On the other hand, variation in the number of exercises was small (among 4 and 7), and most worked on large muscle groups. Therefore, generally speaking, a comparison between the groups is jeopardized by the diversity of protocols used for exercise prescription.

In all these studies, a decline in SBP values following exercise was noted, especially within the first hour after the activity, regardless of the protocol employed. In their study, Melo et al\(^ {8}\), served that besides lasting over the entire period in the laboratory setting, when participants were released to resume their daily activities the decline in SBP persisted for 10 hours, resembling other studies conducted on aerobic exercises\(^{13-16}\). It is noteworthy that in this study, the individuals were taking medication to control BP (Captopril). This finding

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**Figure 1 - Paper selection process flowchart.**
Table 1 – Behavior of blood pressure after a session of resistance exercise

<table>
<thead>
<tr>
<th>Paper</th>
<th>Sample</th>
<th>Sex</th>
<th>n</th>
<th>Intensity</th>
<th>Volume</th>
<th>Interval</th>
<th>Method of measurement</th>
<th>Duration of monitoring</th>
<th>Results</th>
<th>Mechanism identified</th>
</tr>
</thead>
<tbody>
<tr>
<td>Moraes et al</td>
<td>Sedentary (44±2.5 years of age)</td>
<td>M</td>
<td>10</td>
<td>50% 1RM</td>
<td>7 exercises; 3 sets; 12 reps. (Circuit)</td>
<td>30 sec between exercises; 2 min between circuits</td>
<td>ABPM</td>
<td>24h</td>
<td>↓SBP at 45 and 60 min; ↓DBP and MBP at 5, 10 and 60 min</td>
<td>Kallikrein release</td>
</tr>
<tr>
<td>Hardy and</td>
<td>Sedentary (50±10.2 years of age)</td>
<td>M</td>
<td>24</td>
<td>8-12RM</td>
<td>7 exercises; 3 sets; 8-12 reps.</td>
<td>1 min between sets</td>
<td>ABPM</td>
<td>23h</td>
<td>↓SBP for 60 min</td>
<td></td>
</tr>
<tr>
<td>Tucker</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Melo et al</td>
<td>Sedentary* (46±1 years of age)</td>
<td>F</td>
<td>11</td>
<td>40% 1RM</td>
<td>6 exercises; 3 sets; 30 reps.</td>
<td>45 sec between sets; 90 sec between exercises</td>
<td>ABPM</td>
<td>24h</td>
<td>↓SBP and DBP for 10 h relative to control day; ↓SBP from 15 to 90 min; ↓DBP from 45 to 75 min</td>
<td></td>
</tr>
<tr>
<td>Fisher*</td>
<td>Resistance-trained individuals (47±2.5 years of age)</td>
<td>F</td>
<td>7</td>
<td>50% 1RM</td>
<td>5 exercises; 3 sets; 15 reps.</td>
<td>30 sec between exercises; 2 min between circuits</td>
<td>Auscultatory</td>
<td>60 min</td>
<td>↓SBP for 60 min relative to control day</td>
<td></td>
</tr>
<tr>
<td>Mediano et al</td>
<td>Regular exercisers* (61±12 years of age)</td>
<td>M/F</td>
<td>20</td>
<td>10RM</td>
<td>4 exercises; 1 set or 3 sets; 10 reps.</td>
<td>2 min between sets and exercises</td>
<td>Auscultatory</td>
<td>60 min</td>
<td>↑1X10</td>
<td></td>
</tr>
</tbody>
</table>

*Individuals on medication; Sedentary - not engaged in regular physical activity; F - feminine; M - masculine; N - size of the sample; RM - repetition maximum; Reps. - repetitions performed; ABPM - ambulatory blood pressure monitor; SBP - systolic blood pressure; DBP - diastolic blood pressure; MBP - mean blood pressure.

Table 2 – Behavior of blood pressure after a session of aerobic exercise

<table>
<thead>
<tr>
<th>Paper</th>
<th>Sample</th>
<th>Sex</th>
<th>n</th>
<th>Exercise</th>
<th>Intensity</th>
<th>Duration</th>
<th>Method of measurement</th>
<th>Duration of monitoring</th>
<th>Results</th>
<th>Mechanism identified</th>
</tr>
</thead>
<tbody>
<tr>
<td>Forjaz et al</td>
<td>Sedentary (36±2 years of age)</td>
<td>M/F</td>
<td>23</td>
<td>Cycle ergometric</td>
<td>50% VO₂ max</td>
<td>45 min</td>
<td>ABPM</td>
<td>24h</td>
<td>NS</td>
<td>?</td>
</tr>
<tr>
<td>Blanchard et al</td>
<td>Sédentaires (44.2±1.4 years of age)</td>
<td>M</td>
<td>47</td>
<td>Cycle ergometry</td>
<td>40% (low) and 60% (moderate) VO₂ max</td>
<td>40 min</td>
<td>ABPM</td>
<td>14h</td>
<td>↓SBP in low and moderate; ↓DBP in low; 1X10</td>
<td>?</td>
</tr>
<tr>
<td>Ciolac et al</td>
<td>Sédentaire* (46.5±8.2 years of age)</td>
<td>M/F</td>
<td>50</td>
<td>Cycle ergometry</td>
<td>60% HRR</td>
<td>40 min</td>
<td>ABPM</td>
<td>24h</td>
<td>↓SBP, DBP and MBP during 24h relative to control day</td>
<td>?</td>
</tr>
<tr>
<td>Ciolac et al</td>
<td>Sédentaire* Continuous group (48±7 years of age)</td>
<td>M/F</td>
<td>52</td>
<td>Cycle ergometry</td>
<td>Continuous exercise (60% HRR) and interval exercise (50% HRR for 2 min and 80% HRR for 1 min)</td>
<td>40 min</td>
<td>ABPM</td>
<td>24h</td>
<td>↓SBP, DBP and MBP in continuous exercise for 24h; ↓SBP and MBP in interval exercise for 24h (both relative to control day)</td>
<td>?</td>
</tr>
<tr>
<td>Moraes et al</td>
<td>Sedentary (44±2.5 years of age)</td>
<td>M</td>
<td>10</td>
<td>Cycle ergometry</td>
<td>70% HRR</td>
<td>35 min</td>
<td>ABPM</td>
<td>24h</td>
<td>Kallikrein release</td>
<td></td>
</tr>
<tr>
<td>Pescatello et al</td>
<td>? (44±4 years of age)</td>
<td>M</td>
<td>6</td>
<td>Cycle ergometry</td>
<td>40 and 70% VO₂ max</td>
<td>30 min</td>
<td>ABPM</td>
<td>13h</td>
<td>↓SBP at 1, 2, 7, 11 hours; ↓DBP between 2-12h; ↓MBP over the entire period</td>
<td>?</td>
</tr>
<tr>
<td>Pescatello et al</td>
<td>? (38±1±1.8 years of age)</td>
<td>F</td>
<td>7</td>
<td>Cycle ergometry</td>
<td>60% VO₂ max</td>
<td>30 min</td>
<td>ABPM</td>
<td>24h</td>
<td>↓SBP, DBP and MBP up to 7 hours relative to resting values (at 1, 2, 5 and 7 hours)</td>
<td>? PVR↑ CO</td>
</tr>
</tbody>
</table>
Table 2 Continued - Behavior of blood pressure after a session of aerobic exercise

<table>
<thead>
<tr>
<th>Study</th>
<th>Intervention</th>
<th>Gender</th>
<th>Age (years)</th>
<th>Exercise intensity</th>
<th>Duration</th>
<th>Method</th>
<th>Post-exercise hypotension</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pescatello et al.</td>
<td>Sedentary (43.8±1.3 years of age)</td>
<td>M</td>
<td>49</td>
<td>Cycle ergometry</td>
<td>40% and 60% VO_{peak}</td>
<td>30 min</td>
<td>ABPM</td>
</tr>
<tr>
<td>Pescatello et al.</td>
<td>Sedentary (43.8±1.3 years of age)</td>
<td>M</td>
<td>50</td>
<td>Cycle ergometry</td>
<td>40% and 60% VO_{peak}</td>
<td>30 min</td>
<td>ABPM</td>
</tr>
<tr>
<td>Syme et al.</td>
<td>(?43.8±1.3 years of age)</td>
<td>M</td>
<td>50</td>
<td>Cycle ergometry</td>
<td>40% and 60% VO_{peak}</td>
<td>30 min</td>
<td>ABPM</td>
</tr>
<tr>
<td>Guistry et al.</td>
<td>(?44.7±1.9 years of age)</td>
<td>M</td>
<td>22</td>
<td>Cycle ergometry</td>
<td>60% VO_{peak}</td>
<td>30 min</td>
<td>ABPM</td>
</tr>
<tr>
<td>Guistry et al.</td>
<td>(?43±2.2 years of age)</td>
<td>M</td>
<td>23</td>
<td>Cycle ergometry</td>
<td>40% VO_{peak}</td>
<td>30 min</td>
<td>ABPM</td>
</tr>
<tr>
<td>Brownley et al.</td>
<td>(?33.6±2.4 years of age)</td>
<td>M/F</td>
<td>11</td>
<td>Cycle ergometry</td>
<td>60%-70% HR_{max}</td>
<td>30 min</td>
<td>ABPM</td>
</tr>
<tr>
<td>Rondon et al.</td>
<td>Sedentary (68.9±1.5 years of age)</td>
<td>M/F</td>
<td>1-16 8-23</td>
<td>Cycle ergometry</td>
<td>50% VO_{peak}</td>
<td>45 min</td>
<td>ABPM</td>
</tr>
<tr>
<td>Cléroux et al.</td>
<td>(?44±2 years of age)</td>
<td>M/F</td>
<td>13</td>
<td>Cycle ergometry</td>
<td>50% VO_{peak}</td>
<td>30 min</td>
<td>Auscultatory</td>
</tr>
<tr>
<td>MacDonald et al.</td>
<td>Active (23±4 years of age)</td>
<td>M/F</td>
<td>8</td>
<td>Cycle ergometry</td>
<td>70% VO_{peak}</td>
<td>30 min</td>
<td>Intra-arterial measurement</td>
</tr>
<tr>
<td>MacDonald et al.</td>
<td>Sedentary (24±5±1 years of age)</td>
<td>M/F</td>
<td>11</td>
<td>Cycle ergometry</td>
<td>70% VO_{peak}</td>
<td>30 min</td>
<td>Intra-arterial measurement</td>
</tr>
<tr>
<td>Wallace et al.</td>
<td>(?48.4±12.5 years of age)</td>
<td>M/F</td>
<td>25</td>
<td>Treadmill</td>
<td>Intermittent exercise -50% VO_{max}</td>
<td>50 min (5X10min)</td>
<td>ABPM</td>
</tr>
<tr>
<td>Wallace et al.</td>
<td>(?48.4±11.7 years of age)</td>
<td>M/F</td>
<td>21</td>
<td>Treadmill</td>
<td>Intermittent exercise - 50% VO_{max}</td>
<td>50 min (5X10min)</td>
<td>ABPM</td>
</tr>
<tr>
<td>Taylor-Tolbert et al.</td>
<td>Sedentary (60±2 years of age)</td>
<td>M</td>
<td>11</td>
<td>Treadmill</td>
<td>Intermittent exercise - 70% VO_{max}</td>
<td>45 min (3X15min)</td>
<td>ABPM</td>
</tr>
<tr>
<td>Quinn</td>
<td>Sedentary (M=41.3±8.9 years of age) (F=43.6±7.4 years of age)</td>
<td>M/F</td>
<td>16</td>
<td>Treadmill</td>
<td>50% and 75% VO_{max}</td>
<td>30 min</td>
<td>ABPM</td>
</tr>
<tr>
<td>Wilcox et al.</td>
<td>(?50 years of age)</td>
<td>M</td>
<td>10</td>
<td>Treadmill</td>
<td>Intermittent exercise</td>
<td>50 min (5X10min)</td>
<td>Auscultatory</td>
</tr>
<tr>
<td>Bennett et al.</td>
<td>(?48 years of age)</td>
<td>M</td>
<td>7</td>
<td>Treadmill</td>
<td>Intermittent exercise</td>
<td>50 min (5X10min)</td>
<td>Auscultatory</td>
</tr>
</tbody>
</table>
has significant external validity, since resistance exercise combined with medication may potentiate the duration of the reduction in BP levels.

As to DBP levels, only three studies\(^{3,11,12}\) reported PEH despite using different protocols. Moraes et al\(^{3}\) observed a decline in DBP levels at isolated time points: 5, 10, and 60 minutes following weight training. This fact reduces the clinical relevance of PEH since the BP decline is not sustained. Using the conventional method, Mediano et al\(^{22}\), also observed reductions in BP at 30 and 50 minutes following the activity. However, they used a high-intensity exercise protocol (10MR), and participants were on medication. The prescription of high-intensity exercises reduces the study’s external validity, since hypertensive individuals should follow a light-to-moderate intensity program of exercises because it elicits lower increases in BP values during the activity\(^{1}\).

In addition to these, another study\(^{8}\) reported a decline in DBP (45 and 75 minutes after exercise) in a controlled setting. However, the DBP values continued reduced for 10 hours after the exercise session compared with a non-exercising control day. Findings such as those observed in this study\(^{8}\) are relevant, since they allow, for instance, that hypertensive individuals be exposed to high blood pressure values, for both SBP and DBP, for shorter periods of time. However, regardless of the results reported by the authors (Melo et al\(^{3}\)), it is important to keep in mind that the decline in BP did not occur relative to the resting values recorded on the day of exercise training, but only in relation to the day when the individuals did not exercise (control day). Thus, considering that PEH is the reduction in BP values relative to pre-exercise resting values, further investigation is warranted to assess BP levels during longer periods.

Finally, a study\(^{9}\) conducted with physically fit individuals did not observe any decline in DBP levels. Considering that chronic training may induce hemodynamic adjustments in the body\(^{17}\), this may potentially interfere in the PEH of physically fit individuals. In this sense, the benefit from regular activity would have been already obtained by the chronic effect and, thus, would not be observed after a single bout of exercise. However, no studies were found on the influence of hypertensive individuals’ fitness status on PEH; therefore, greater inferences would be merely speculative. Likewise, studies\(^{18,19}\) involving physically fit normotensive subjects reported no significant drops in DBP following a session of resistance exercises.

Regarding the physiological mechanisms involved in BP decline after resistance exercise, only two studies\(^{13,20}\) were found; one of them\(^{11}\) was conducted with hypertensive individuals, whereas the other\(^{20}\), with normotensive individuals. Moraes et al\(^{3}\) observed that PEH is related to the release of kallikrein, a vasodilator agent\(^{11}\). However, this substance has been rarely studied in the context of PEH, and its actual contribution to reducing BP is still unclear. Rezk et al\(^{20}\) evaluated peripheral vascular resistance (PVR) and cardiac output (CO) in normotensive individuals after a bout of resistance exercise with intensities ranging from 40% to 80% of 1MR. The lowest intensity exercise elicited PEH for both SBP and DBP, although reductions of CO and maintenance of PVR were observed. Conversely, the highest intensity exercise elicited PEH only for SBP, which was associated mainly with a decrease in CO, since PVR increased after exercise. Nevertheless, the sample consisted of healthy young individuals, and this does not allow us to safely extrapolate data for hypertensive people.

**Hypotension following aerobic exercises**

For aerobic exercising, the majority of studies used cycle ergometric protocols\(^{13,15,23,25,32,39,41-43}\), others used ergometric treadmills\(^{16,22,26,27,33-38}\), and only one used water running\(^{16}\).
Regarding PEH and aerobic exercise, only two studies\textsuperscript{15,26} reported no declines in SBP and DBP, compared to resting and control day values. Additionally, a few other studies\textsuperscript{11,22,33,34,36-40,42,43} recorded PEH in short periods of time (30 to 120 minutes) for both SBP and DBP. Reductions in BP for short periods of time have a lower impact on the cardiovascular health of a hypertensive individual, since the duration of the decline in BP is the most important factor in this phenomenon\textsuperscript{9}. Nonetheless, of these 11 studies, eight\textsuperscript{22,33,34,36-40} used the auscultatory method, whereas two\textsuperscript{42,43} used the intra-arterial measurement method, which hinders BP monitoring outside the laboratory setting.

Within this context, approximately 50% of the studies\textsuperscript{13-16,23,24,27-32,35,41} involving aerobic exercise reported reductions in SBP and DBP levels sustained over long periods. However, only two studies\textsuperscript{14,15} recorded declines in SBP compared to pre-exercise values, a fact that characterizes the PEH phenomenon. The findings of some studies\textsuperscript{13,16,23,27,31,32,35} were significant compared with a non-exercising control day, but not compared to resting values. Moreover, a few studies\textsuperscript{28-30,41} recorded increased SBP values compared to resting values, but these were smaller compared to those of the control day.

On the other hand, six studies observed PEH for DBP\textsuperscript{14,15,24,28-30}. Reductions compared only with the control day were recorded in eight studies\textsuperscript{13,16,23,27,31,32,35,41}, and, differently from SBP values, not one study recorded an increase compared with DBP pre-exercise values. It is important to compare BP with the control day value, since this allows an analysis of the behavior of pressure values without exercise. However, as has already been mentioned, the concept of PEH consists in the reduction in BP levels relative to resting values and, thus, it would be interesting to quantify and qualify the behavior of the sample on the control day to allow greater inferences to be drawn.

There is an apparent lack of consistency between study findings regarding exercise intensity and a decline in BP following exercise. Thus, PEH can be observed following bouts of low\textsuperscript{23,27,36,39} and moderate\textsuperscript{15,22,30,31} and high-intensity\textsuperscript{35,38} exercise. In this sense, a few studies\textsuperscript{13,14,16,24,28,29,14,41} that attempted to directly compare exercise intensity and decline in BP have yielded contradictory results. A few studies\textsuperscript{34,36} showed that high-intensity exercises (70 to 75% VO\textsubscript{2max}) promote greater reductions in pressure values than mild-intensity (50% VO\textsubscript{2max}) activities. Conversely, other studies\textsuperscript{13,14,24,28,29,41} have reported no differences regarding BP responses and exercise intensity.

It is noteworthy that some studies\textsuperscript{22,23} compared continuous and interval exercise, suggesting that continuous exercise has a slight advantage over interval exercise. Therefore, literature lacks a consensus as to the relationship between exercise intensity and PEH in hypertensive individuals. Nevertheless, the American College of Sports Medicine\textsuperscript{1} recommends the prescription of low-to-moderate intensity exercise (40-60% VO\textsubscript{2max}), exactly because it elicits lower increases in BP during the activity and, thus, provide greater safety.

Another variable that can interfere with PEH is the duration of the exertion; however, a reduction in BP levels was observed in both, shorter duration sessions (15 to 20 minutes)\textsuperscript{30,41} and longer sessions (50 minutes)\textsuperscript{22,27,37,40}. Bennett et al\textsuperscript{47} evaluated the BP response to intermittent exercise, in which individuals exercised during ten minutes alternately with three-minute rest periods. This routine was repeated five times and the authors verified that the duration of the activity influenced the reduction in BP levels. However, a study\textsuperscript{26} employing a very similar methodology, did not observe any reduction in BP levels after exercise. Within this context, studies comparing directly the BP response and the duration of exercise sessions yield contradictory results, since there is evidence that the longer the duration of the exercise session, the greater the reductions in pressure values\textsuperscript{16}. On the other hand, other findings suggest no difference in PEH following shorter exercise sessions (ten minutes) or ones of longer duration (30 minutes)\textsuperscript{16}. In their study with hypertensive individuals, Guidry et al\textsuperscript{48}, observed a slight advantage of longer-duration exercise bouts (30 minutes vs 15 minutes) in DBP. Therefore, there seems to be no relationship between exercise duration and PEH; this calls for further studies within this context, since two\textsuperscript{15,46} of the three papers investigating the influence of the duration of effort on PEH used a sample of borderline hypertensive individuals.

The mechanisms involved in the reduction of BP following aerobic exercise may be different from those involved in resistance exercise. Curiously, Rueckert et al\textsuperscript{59} observed a biphasic pattern of PEH, in which the initial fall in BP is determined by the reduction of PVR, followed by the reduction in CO. Moreover, some studies\textsuperscript{23,24} observed that the PEH is determined only by the reduction of CO, which is relative to the reduction in the systolic volume. Furthermore, Hagberg et al\textsuperscript{41} noted that among senior individuals, PEH is probably mediated by the reduction in CO, since aging leads to arterial stiffness. Consequently, there is lower capacity for vessel dilation\textsuperscript{47}.

On the other hand, a few studies\textsuperscript{15,39} demonstrated that the fall in BP levels is influenced by the reduction in PVR, which can be associated with the release of vasodilator drugs, such as nitric oxide\textsuperscript{44,48} prostaglandins\textsuperscript{49,50} and adenosine\textsuperscript{51}. However, studies on the effect of blocking the synthesis of nitric oxide and prostaglandins still reported decline in BP levels\textsuperscript{52,53}. Moreover, the reduction in PVR can be associated with a decrease in sympathetic activity, observed in both human\textsuperscript{14,53} and animal models\textsuperscript{56}. Franklin et al\textsuperscript{57} observed that thermoregulation is another factor that could be associated with PEH. Nevertheless, a few studies suggest that thermoregulation\textsuperscript{7} and splenic\textsuperscript{58} cutaneous\textsuperscript{59} and cerebral\textsuperscript{60} circulation contribute very little to PEH. Finally, another mechanism that might influence PEH is baroreflex function, which seems to increase after an exercise session\textsuperscript{61,62}. This mechanism acts in two ways: it lowers PVR and heart rate in response to an increase in BP and, it increases these variables in response to a decrease in blood pressure values\textsuperscript{49}.

Therefore, there is no evidence as to the exact mechanism involved in PEH, as apparently several factors contribute to the occurrence of this phenomenon. It is noteworthy that a large part of the studies involving mechanisms related with the decline in BP used samples of normotensive individuals or animal models. Consequently, these findings should be analyzed very carefully, since the extrapolation of their results for the hypertensive population is jeopardized.
Monitoring of arterial blood pressure following exercise

Regarding the monitoring of BP after aerobic exercise and weight-training, two studies9,12 used only the auscultatory method after resistance exercise, whereas eight studies22,33,34,36-40 used the same method after aerobic exercise. In these studies, investigators monitored BP between 30 and 120 minutes after exercise, and the measurements were made mostly with ten-minute intervals. Moreover, two studies62,63 used direct measurements of BP. These procedures are interesting, as they allow the investigator to analyze the cardiovascular behavior in the absence of intervening variables, although they diminish the external validity of the findings.

On the other hand, three studies8,10,11 involving weight-training and seventeen studies11,13-16,23-32,35,41 involving aerobic exercises used ambulatory blood pressure monitoring (ABPM). However, in a few cases, the information from the ABPM may be misleading. For instance, one study12 reported a significant decrease in BP over the average 24 hours of monitoring. Nevertheless, the significant decline in BP occurred only in the measurements taken during the night. This may lead the reader to conclude that the exercise promoted a decline in BP throughout the entire 24-hour period.

Besides the 24-hour assessment, ABPM can also be analyzed in two different periods, vigil and sleep2,3. Thus, ABPM can be employed to detect refractory hypertension, for instance. This method is very useful, since it allows the comparison of BP values in the medical office and during the period of vigil, which in turn may help detect “white-coat hypertension”64.

Conclusion

Apparently, aerobic exercises promote a greater and longer reduction in BP levels than resistance exercises. However, further studies applying resistance exercise in hypertensive individuals are necessary. There is not one prescription model for weight-training exercise that can promote greater reductions in the blood pressure values of hypertensive individuals. However, considering the individuals’ safety, the exercise intensity should be around 50% of 1MR, with a minimum of one-minute intervals (between sets and exercises), using especially the great muscle groups. Moreover, long series of exercises that leads to exhaustion should be avoided, as they can induce greater BP increases.

In most studies, the prescription of aerobic activities ranged from 50% to 60% of the VO2max, performed continuously during 30 to 45 minutes. It is significant that there are still findings that conflict relative to the best intensity and duration of this modality of exercise.

The exact mechanism involved in PEH remains unknown, but there is a compensatory mechanism. That is, the reduction in BP levels is due to the reduction in PVR or CO, but when no reduction occurs in one of these variables, there is compensation in the other in order to lower BP levels. Finally, further studies are required to investigate BP behavior regarding pre-exercise values for longer periods of time, in order to broaden our knowledge of this subject.

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