Blood pressure behavior after counter-resistance exercises: a systematic review on determining variables and possible mechanisms

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ABSTRACT

Post-exercise hypotension (PEH) is a phenomenon with high clinical relevance, despite its doubtful aspects concerning the variables that may contribute to its manifestation. The questioning is even greater when counter-exercise is applied with the purpose to cause PEH. Within this context, the aim of this study was to review some variables of the counter-resistance exercise that may be associated with PEH. Moreover, some physiological mechanisms possibly related to this effect were discussed. Fourteen references were found concerning counter-exercise and PEH. Six studies observed hypotensive effect for systolic blood pressure (SBP) and/or diastolic blood pressure (DBP) after the counter-resistance exercise. However, it was observed that some studies did not identify significant differences (p > 0.05) for SBP and DBP (n = 4) or even reported significant increase (p < 0.05) (SBP or DBP) (n = 4). These disagreeing results may be related to the exercise volume and intensity, as well as the monitoring period. Nevertheless, it is possible to identify PEH when the counter-resistance exercise is applied, both in normotensive and hypertensive individuals. Despite of that, the physiological mechanisms responsible for this kind of response still remain obscure.

INTRODUCTION

Some of the main medical normative agencies have published positions in which they considered the counter-resistance exercise prescription adequate for healthy individuals11, as well as for hypertensive12 and cardiac ones13. Some years ago, more specifically until the 1990 decade, the exercise recommendation for individuals with any chronic disease, especially cardiovascular ones, was restricted to aerobic training4. However, the conduction of studies involving counter-resistance exercise identified chronic adaptations which the aerobic training did not contemplate. For instance, the cardiovascular demand for a given load is attenuated in individuals who systematically train muscular strength10. Counter-resistance training may be considered the one dynamically performed, with the use of specific implements (apparel) or free loads (dumbbells), in which the aim is to increase both the capacity (hypertrophic adaptations) as well as the ability (neural adaptations) in order to lift a given load during a determined movement. During this kind of exercise, an expressive increase of the cardiovascular responses is expected, especially if it is performed until fatigue6. Such increase is usually mediated by the vases occlusion through the muscular contraction, by the Valsalva maneuver, and in some cases, by the chemoreceptor response derived from the metabolites accumulation17. Thus, some counter-resistance training variables are directly associated with the cardiovascular responses increase, especially of blood pressure (BP), namely number of series8, recovery interval9, mobilized load in cardiac individuals10, types of exercises11 and muscular mass involved12. Therefore, a closer supervision of the cardiovascular responses in the counter-resistance training is important for individuals who have any cardiovascular limitation.

It is known that after a training session the BP (the systolic and diastolic indices were considered as BP) may decrease below the indices shown in the pre-exertion condition, in an effect called post-exercise hypotension (PEH). The PEH, when not derived from autonomous compromising, which may interfere in the orthostatic tolerance, is considered an excellent intervention for the pressor control, especially in hypertensive individuals12. Thus, the greater the magnitude, and mainly the PEH duration, the more efficient the non-pharmacological strategy of resting BP reduction tends to be13. Therefore, the understanding about the exercise quantity and type for the PEH determination, as well as its action mechanism, is aimed.

The aim of the present study was to review the literature concerning the intervenient variables to the PEH manifestation derived from counter-resistance exercise and understand through it what is established concerning such issue.

PROCEDURE FOR THE ARTICLES SEARCH

The articles search was systematically conducted in the Medline, Scielo, Sportdiscus and Lilacs databases without date limits. In all cases the terminology used was: post-exercise hypotension, resistance exercise, strength exercise, resistive exercise, weight training, blood pressure response and recovery blood pressure combined in quotations, in the title or in the abstract. In the Lilacs and Scielo databases Portuguese terms were also used, namely: hipotensão, pressão arterial, exercício de força e exercício resistido.

In Medline sixteen studies were obtained, while in Scielo four references were obtained. Articles different from the ones mentioned in the previous databases were not found in the Sportdiscus and Lilacs databases. Studies which measured the BP only immediately after exertion; exclusively analyzed orthostatic hypotension or studied exercise chronic effect were not considered valid for the present review. Thus, six references obtained in the Medline base were discarded. Due to the fact that studies involving counter-resistance exercise and BP behavior after exertion were scarce,
POST-EXERCISE HYPOTENSION

PEH documentation is not recent. In the late XIX century, more specifically in 1897, a 90 min-decrease in the BP was described after a fast race in a distance of approximately 360 m(14). Some years later, Schneider and Thuesell(15) showed that, in two minutes after climbing a chair with 45, 72 cm height for five times in 15 s, the blood pressure and the heart rate reduced in relation to the pre-exercise index.

However, only from 1980 relevant investigations on PEH became more solid concerning exercise prescription. Within this context, the after counter-resistance exercise may be understood in two ways. Immediately after its ending, the BP tends to rapidly decrease (i.e., approximately 10 s when the exercise is exhausting)(8). This reduction occurs due to hyperemia in the muscles which was obstructing the blood flow and by the baroreflex action as well(8). Nevertheless, the BP may continue to decline in the minutes subsequent to the exercise ending, exceeding in some occasions, the indices measured before the session, characterizing the PEH. Whenever there is not autonomic dysfunction, the baroreflex mechanism does not allow such reduction to compromise the orthostatic tolerance(13). Thus, the PEH should be more observed in individuals who have high resting BP, such as hypertensive patients(16).

Concerning clinical relevance, the PEH may be understood as a non-pharmacological action strategy for BP reduction. Despite of that, it is crucial that the duration is the longest as possible(16). There is evidence of PEH for longer than 12 h after an exercise(17). Conversely, research on PEH predominantly went back to aerobic exercise. Concerning this activity, the data suggest relation between PEH and exercise duration(18), intensity(19), sex and training status(20), age group(21-22), race(23) and muscular mass(24).

Available results concerning BP behavior after counter-resistance exercise are scarce and even conflicting. For instance, significant reduction in the BP after a counter-resistance exercise session performed by normotensive individuals, in 24 h monitoring was not identified, regardless age group, exercise intensity and training status(25-26). Probably, the clinical condition of the sample was not identified, regardless age group, exercise intensity and training status. The authors hence proposed the analysis of the after-exercise anxiety behavior, which may have influenced the BP response.

The counter-resistance exercise prescription may also be intertwined in the findings on PEH. For instance, a significant decrease was reported only for diastolic BP (DBP) after a circuit exercises session(29). On the other hand, studies performed in our laboratory identified important reductions especially for systolic BP (SBP) after a counter-resistance exercises session, independently of the intensity(20) and performance type (circuit x traditional)(31). In all cases, the BP monitoring period was 60 min, which seems sufficient for the manifestation of the hypotensive effect in individuals with BP at normal indices.

In the following sessions, some aspects possibly related to BP reduction after counter-resistance exercise, are presented and discussed. The main training variables which could be associated with PEH were descriptively treated and presented in table 1. A summary of the main characteristics of the several studies found is presented in table 2.

INFLUENCE OF THE EXERCISE VOLUME

There are three main variables which are closely related with the exercise volume in the counter-resistance exercise: number of series; number of repetitions and quantity of performed exercises. All the variables are closely related and determine the total quantity of performed work. For instance, when the mobilized load is set, a session consisting of several exercises of a single series may represent a work quantity equal to another session with few exercises of multiple series. Therefore, the influence of the exercise volume over the PEH will be only verified when the intensity is previously determined and the exercises, series or repetitions are manipulated.

A study conducted by our group involved medicated hypertensive patients. The load (10RM) was kept in the four counter-resistance exercises (straight supine, leg-press, standing rowing and twist triceps), while the number of series was altered (one or three)(32). The SBP was not lower than the resting one until 60 min after the session of three series, the DBP values were lower than in the resting conditions in the measurements taken 30 and 50 min after the exercise. Conversely, after the single series session, no alteration was observed in the DBP and the SBP showed decrease in relation to the pre-exercise index only in the measurement taken 40 min after the exertion ending.

The majority of the found studies used a session involving several counter-resistance exercises. Actually, the average of used exercises was of five to six exercises. However, the isolated effect of the exercise over the PEH seems to be dependent on other variables, such as intensity or monitoring period. The study by Roltsch et al.(25) illustrated this fact when used the largest quantity of exercises (n = 12), but did not identify any alteration in the BP after exertion. In this case, the follow-up was of 24 h. The study by MacDonald et al.(33) used only an exercise (leg-press), performed during 15 min with load corresponding to 65% of the maximal one. The authors identified significant reduction in the SBP between 10 and 60 min after exercise, with more expressive reduction in the measurement taken 30 min after exertion (20 mmHg). It is worth mentioning that there was no control of the number of series and repetitions; however, when the duration of the activity was observed, a reasonable amount of work is supposed. In this case, the exercise was performed with one leg - when it was in fatigue, the subject continued the exercise with the counterlateral limb. Although the used protocol is considered an activity which has the aim to improve the resistance strength, such strategy is not the most used in training centers, which decreases to a certain extent the external validation of the experiment. On the other hand, a single counter-resistance exercise (bilateral leg extension), performed in three series of 12 repetitions up to exhaustion, did not cause significant hypotension after the session. The authors simply observed a reduction between 2 and 3% of the SBP between 10 and 20 min after the exercise ending(34).

Concerning the number of repetitions, the studies that analyzed the PEH in relation to this variable also proposed to modify the exercise intensity. No reference concerning the isolated study of

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**Table 1**

Descriptive statistics for the counter-resistance exercise and the monitoring period used in the studies

<table>
<thead>
<tr>
<th>Exertions quantity</th>
<th>Revised studies</th>
<th>Average*</th>
<th>Standard deviation*</th>
<th>Minimum</th>
<th>Maximum</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number of repetitions</td>
<td>12</td>
<td>10-15</td>
<td>6-7</td>
<td>4</td>
<td>30</td>
</tr>
<tr>
<td>Intensity (% of 1RM)</td>
<td>7</td>
<td>57-67%</td>
<td>13-15%</td>
<td>40%</td>
<td>70%</td>
</tr>
<tr>
<td>Number of series</td>
<td>13</td>
<td>3</td>
<td>1</td>
<td>1</td>
<td>3</td>
</tr>
<tr>
<td>Monitoring period</td>
<td>14</td>
<td>11 h</td>
<td>21 h</td>
<td>5 min</td>
<td>24 h</td>
</tr>
</tbody>
</table>

* The interval means the minimum and maximum average, once some studies did not use steady indices for number of exercises, repetitions and intensity. The indices were rounded to one decimal case, considering the adjustment for the intensity and number of exercises, repetitions and series variation. The studies that used as percentage intensity of body mass, percentage of maximum repetitions and number of maximum repetitions were not considered. The studies that did not define number of series and repetitions were not considered.
<table>
<thead>
<tr>
<th>Study</th>
<th>Sample</th>
<th>N</th>
<th>Sex</th>
<th>Post-exercise monitoring</th>
<th>Number of exercises</th>
<th>Series</th>
<th>Repetitions</th>
<th>Intensity</th>
<th>Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>Roltsch et al.(25)</td>
<td>Trained normotensive (23 ± 2 years), sedentary (20 ± 2 years) and aerobically trained (24 ± 3 years)</td>
<td>36</td>
<td>M and F</td>
<td>24 h</td>
<td>12</td>
<td>2</td>
<td>8-12</td>
<td>Maximal (8-12RM)</td>
<td>NS</td>
</tr>
<tr>
<td>Bermudes et al.(26)</td>
<td>Sedentary normotensive (44 ± 1 years)</td>
<td>25</td>
<td>M</td>
<td>24 h</td>
<td>10 (circuit)</td>
<td>3</td>
<td>20-35</td>
<td>40% 1RM</td>
<td>NS</td>
</tr>
<tr>
<td>O’Connor et al.(28)</td>
<td>Active normotensive (23 ± 4 years)</td>
<td>14</td>
<td>F</td>
<td>120 min</td>
<td>6</td>
<td>3</td>
<td>10</td>
<td>40, 60 and 80% 1RM</td>
<td>Higher SBP after 1 abd 15 min of the 80% intensity and after 1 min of the 60% intensity</td>
</tr>
<tr>
<td>Hill et al.(29)</td>
<td>Trained normotensive (22-33 years)</td>
<td>6</td>
<td>M</td>
<td>60 min</td>
<td>4 (circuit)</td>
<td>3</td>
<td>Maximum</td>
<td>70% 1RM</td>
<td>Reduction for the DBP in the 60 min.</td>
</tr>
<tr>
<td>Polito et al.(30)</td>
<td>Trained normotensive (M = 20 ± 1 years; F = 21 ± 5 years)</td>
<td>16</td>
<td>M and F</td>
<td>60 min</td>
<td>6</td>
<td>6 and 12</td>
<td>Maximal (6RM) and 50% 6RM (12)</td>
<td>Sequence of 6RM : SBP reduction in the 60 min Sequence with 50% 6RM : SBP reduction up to 40 min and in DBP up to 10 min.</td>
<td></td>
</tr>
<tr>
<td>Simão et al.(31)</td>
<td>Two groups of trained normotensive (G1 = 25 ± 4 years; G2 = 21 ± 4 years)</td>
<td>20</td>
<td>M and F</td>
<td>60 min</td>
<td>G1 = 5; G2 = 6</td>
<td>3</td>
<td>6 and 12</td>
<td>Maximal (6RM) and 50% 6RM (12)</td>
<td>G1 = SBP reduction during 50 min after 6RM and during 40 min after circuit G2 = SBP reduction during 60 min after 6RM and during 40 min after 12 rep; DBP reduction during 10 min after 12 rep</td>
</tr>
<tr>
<td>Mediano et al.(32)</td>
<td>Active hypertensive (61 ± 12 years)</td>
<td>20</td>
<td>M and F</td>
<td>60 min</td>
<td>4</td>
<td>1 and 3</td>
<td>10</td>
<td>Maximal (10RM)</td>
<td>SBP reduction after 40 min in 1 series and in the 60 min in 3 series; DBP reduction in 30 and 50 min in 3 series</td>
</tr>
<tr>
<td>MacDonald et al.(33)</td>
<td>Active normotensive (24 ± 2 years)</td>
<td>13</td>
<td>M</td>
<td>60 min</td>
<td>1</td>
<td>15 min of performance</td>
<td>Maximal</td>
<td>65% 1RM</td>
<td>Reduction in the SBP between 10 and 60 min</td>
</tr>
<tr>
<td>Polito et al.(34)</td>
<td>Trained normotensive (33 ± 11 years)</td>
<td>18</td>
<td>M and F</td>
<td>20 min</td>
<td>1</td>
<td>3</td>
<td>12</td>
<td>Maximal (12RM)</td>
<td>NS</td>
</tr>
<tr>
<td>Fisher(35)</td>
<td>Normotensive (45 ± 2 years) and active hypertensive (48 ± 3 years)</td>
<td>16</td>
<td>F</td>
<td>60 min</td>
<td>5 (circuit)</td>
<td>3</td>
<td>15</td>
<td>50% 1RM</td>
<td>Reduction in the SBP in the 60 min for both groups</td>
</tr>
<tr>
<td>Brown et al.(36)</td>
<td>Active normotensive (21 ± 2 years)</td>
<td>7</td>
<td>M and F</td>
<td>60 min</td>
<td>5</td>
<td>3</td>
<td>8-10 and 20-25</td>
<td>70% 1RM (8-10) and 40% 1RM (20-25)</td>
<td>Increase in the SBP and reduction in the DBP in both intensities after 2 min; with no alteration in the SBP after 5 min and in the DBP after 15 min in both intensities</td>
</tr>
<tr>
<td>Koltyn et al.(37)</td>
<td>Active normotensive (19 ± 3 years) and sedentary (18 ± 2 years)</td>
<td>50</td>
<td>M and F</td>
<td>5 min</td>
<td>7-10</td>
<td>2-3</td>
<td>7-10</td>
<td>30-80% of the body mass</td>
<td>Increase in the SBP for the experimental group and reduction for the control group</td>
</tr>
<tr>
<td>Focht e Koltyn(38)</td>
<td>Trained normotensive and sedentary</td>
<td>84</td>
<td>M and F</td>
<td>180 min</td>
<td>4</td>
<td>3</td>
<td>4-8 or 12-20</td>
<td>80% 1RM (4-8 rep) and 50% 1RM (12-20 rep)</td>
<td>SBP increased immediately after the session with 80% 1RM and DBP decreased after the session with 50%. Both returned to base line in the 20 min measurement</td>
</tr>
<tr>
<td>Raglin et al.(40)</td>
<td>Trained normotensive (20 ± 1 years)</td>
<td>26</td>
<td>M and F</td>
<td>30 min</td>
<td>6-7</td>
<td>3</td>
<td>6-10</td>
<td>70-80% 1RM</td>
<td>NS</td>
</tr>
</tbody>
</table>

Trained = individuals who were practicing counter-resistance training for more than 6 months; active = individuals who practiced other physical activities or who were starting the counter-resistance training; sedentary = individuals who were not engaged in any physical activities program; the age was average; N = total number of subjects; M = male; F = female; RM = maximal repetition; rep = repetitions; SBP = systolic blood pressure; DBP = diastolic blood pressure; NS = non-significant results.
the repetitions number and BP behavior after exercise was found. Thus, at least for now, this gap still remains.

One may conclude that there is a possibility that the number of series influences the duration of the hypotensive effect after counter-resistance exercise. Concerning the exercises quantity and number of repetitions though, it was not possible to make any relevant comments, since these variables were studied in association with others which will be discussed in the following topics.

Nevertheless, what would be the influence of the muscular mass when the remaining variables of exercises sessions volume were constant?

**MUSCULAR MASS INFLUENCE**

Concerning aerobic exercise, there seems to be a close relation between the muscular mass involved and the PEH duration[24]. In this case, the lower limbs exercise (cycle ergometer) tends to cause an extended PEH than the exercises with the upper limbs (arm ergometer). Concerning the counter-resistance exercise, there are not available data for a safe inference. A comparison between sequences performed only for lower and upper limbs would be necessary.

However, a study performed together with our laboratory compared the influence of the muscular mass over the post-exertion BP for the same muscular group[34]. In this experiment, the sample performed three series of 12RM of uni and bilateral leg extension. Probably, due to the reduced number of series, as described in the previous topic, no alteration in the pressor responses was identified. Nevertheless, these results could be different in case the required muscular mass was higher, such as in the leg press exercise.

Thus, no further comments can be made concerning the muscular mass in the PEH caused by the counter-resistance exercise. However, even when the counter-resistance training session is performed with specific muscular mass and exercises volume, the results can still be clashing. One of the possible explanations would be related to the follow-up period of the post-exercise pressor responses.

**MONITORING PERIOD**

Only two studies out of the fourteen found on BP behavior after counter-resistance exercise[25-26] used the 24 h monitoring. In both cases no significant differences were found concerning pre-exercise. The only alteration identified for SBP and DBP was for hypotension during sleep, which is an expected effect. Nevertheless, the authors used normotensive individuals as sample, which may have influenced the results. Such fact is explained by the fact that normotensive individuals present lower PEH than hypertensive ones[36]. Therefore, one may think that if the sample consists of hypertensive individuals, the PEH tends to be more extended. Pescatello et al. confirmed such theory[27], in a study in which reduction of 5 ± 1 mmHg in the SBP for 8,7 h and of 8 ± 1 mmHg in the DBP for 12,7 h after the aerobic exercise performed in different intensities, in borderline hypertensive objects without medication was observed.

The studies that used shorter monitoring periods (60 min) identified PEH in both normotensive and hypertensive individuals[29-33,35]. However, excessively short periods may provide results different from those obtained in a relatively longer period. For instance, some studies[28,36-38] identified increase in the SBP up to 5 min after a counter-resistance exercises session, probably due to the adrenergic action still increased.

Therefore, in order to identify any significant alteration in the BP after counter-resistance exercise, we believe a follow-up period of at least 60 min is necessary. Conversely, longer periods may not be sensitive enough to identify PEH in normotensive individuals. Despite of that, studies with monitoring periods relatively short, using the same muscular mass and exercises volume, revealed contradictory results. Therefore, other variables such as the counter-resistance exercise intensity could interfere in the results.

**EXERCISE INTENSITY**

Concerning the exercise with aerobic nature, some studies indicate that intensity of the activity does not influence the magnitude of the hypotensive effect[17,19]. Such fact is particularly important for hypertensive individuals since it suggests that training more moderate intensities may be effective, avoiding exaggerated cardiovascular responses during exercise.

Yet, few experiments proposed the investigation of the isolated influence of the counter-resistance exercise intensity over the PEH. Some authors[36,38] compared exercises with different intensities, but with different number of repetitions as well, which limits the inferences. Nonetheless, whenever the experimental outlining keeps the work volume steady, it seems that there is a greater chance to observe similar results to the ones obtained with aerobic exercise. In two studies with this purpose[30,31], it was concluded that there may be a significant reduction in the SBP up to 40-60 min, independently of the exertion intensity. In both investigations, the load was reduced to half, while the number of repetitions doubled. Such strategy offered a very intense sequence, with few repetitions, and another one very mild, with more repetitions, though.

On the other hand, the isolated manipulation of the intensity in the study by O’Connor et al.[28] did not altered the post-exertion BP. The authors submitted normotensive women to a protocol of six exercises performed in three series of 10 repetitions, with load corresponding to 40, 60 and 80% of 10RM. The SBP was only higher than the resting one in the measurement taken 15 min after the exercise with intensity of 80%. However, this study proposed the study of the exercise effect over the anxiety state, using the BP measurement as a complementary variable. Thus, the measurement occurred in 15 min. intervals during 2 h, in which the subjects were freed for their daily activities, which reduced the laboratorial control of the resting condition.

It is worth mentioning that all the pointed studies used normotensive subjects as sample. Nevertheless, a study using low intensity was found (50% of the maximal load) and that identified PEH in hypertensive and normotensive women[35]. The session with five exercises performed in circuit (three rows of 15 repetitions) made reduction in the SBP possible in the 60 min. of follow-up, without any significant alteration in the DBP.

Thus, one may conclude that the intensity does not seem to interfere in the SBP and DBP behavior after counter-resistance exercise, since the exercises volume is kept. Yet, how should the PEH behavior be when the sample consists of hypertensive subjects?

**INITIAL INDICES OF THE BLOOD PRESSURE**

The study by Fisher[35], as mentioned before, compared the post-exertion BP responses in hypertensive and normotensive women. The hypertensive group did not use medication and presented BP indices significantly higher than the normotensive group. After the exercise, the SBP was not influenced by its initial index and similarly reduced in both groups.

There seems to be PEH even when the sample is under medication effect. As mentioned before, a study conducted by researchers of our laboratory used as sample hypertensive medicated subjects, with resting pressor indices close to 120 and 75 mmHg (SBP and DBP, respectively)[32]. When the training volume was compared (one series x three series) in four counter-resistance exercises, it was verified that the session performed with higher volume caused PEH for the SBP in the entire follow-up period (60 min). The de-
crease in absolute terms was between 8 and 6 mmHg. Despite of that, considering that the sample consisted of hypertensive subjects, it is possible to infer that this result is clinically relevant.

It is concluded that the PEH derived from the counter-resistance exercise may occur in people with different resting indices for the BP, even when under medication. However, it is worth highlighting that the monitoring time in the two studies mentioned in this section was of 60 min. It is unknown though, the time period in which the normotensive and hypertensive subjects remain under the hypotensive response action derived from the counter-resistance exercise, due to the involved physiological mechanisms.

POST-EXERCISE HYPOTENSIVE POSSIBLE MECHANISMS

Differently from the pharmacological procedures, the physiological mechanisms that may explain the PEH are not totally clear yet. The research that proposed to investigate such mechanisms has not provided consistent data yet.

One of the hypotheses is the decrease of the sympathetic nervous activity (SNA). A study by Floras et al.(39) demonstrated that hypertensive subjects presented, concomitantly with PEH, reduction of SNA during 1 h, after performing 45 min. of moderate aerobic exercise. When the sample consisted of normotensive men, the same exercise did not determine PEH or alteration in the SNA(40). Nonetheless, the normotensive men showed lower SNA than the hypertensive ones in resting; suggesting that the PEH observed in the hypertensive men has relation with the decrease of the central sympathetic activity. Genetically hypertensive rats also presented, after a moderate aerobic exercise session, SNA indices lower than the pre-exercise one(42). However, the authors observed that the cardiac debt was higher than in resting. In this case, the heart rate, the venous return or both could have influenced in the cardiac debt increase, without repercussion in the BP indices after exercise.

Actually, some results with humans verified decrease in the cardiac debt during PEH. Nonetheless, this fact was not confirmed by other studies. In a study involving hypertensive elders for instance, an association between PEH and reduction in the cardiac debt was described, firstly by the reduction in the ejection volume, and later by decrease in the heart rate(27). On the other hand, studies with sample of hypertensive individuals as well, identified reduction in the BP after exertion, with increase(42) or with no alteration in the cardiac debt, though(43). Another experiment involving aerobic exercise (45 min of cycle ergometer at 30, 50 and 75% of the peak VO2) and normotensive subjects identified PEH for SBP and DBP in the two highest intensities, regardless the sex and training status, but with differentiated behavior for the cardiac debt(44). In this study, while the sedentary subjects and the trained women experienced cardiac debt increase, the trained men demonstrated reduction. Moreover, in all exercise intensities, the cardiac debt was higher than in resting, while the systemic vascular resistance decreased.

Since the SNA and the cardiac debt do not completely explain the PEH, an interaction between these mechanisms of central origin and other of peripheral via, such as the vascular resistance, is suggested(40). In this case, a reduction of the vascular resistance seems to occur, with consequent increase of the blood flow after exercise. On the other hand, it is important to consider that the increase of the blood flow may locally or systemically occur. In the first case, the blood flow in increased after exercise only in the required region. Such fact was confirmed in the study by Legramante et al.(43), in which a reduction of the leg peripheral vascular resistance was observed (which was required during a maximal test), with no alteration in the forearm, though. Probably, the hyperventilation may have been the explanation for this result. Cleroux et al.(42) verified that, after 30 min of exercise in cycle ergometer at 50% of the maximal capacity, the peripheral resistance in the forearm was lower than in the resting one. Such fact may find its explanation by the action of endothelial vasodilator substances, such as the prostaglandins and the nitric oxide.

Both prostaglandines and nitric oxide are released by the endothelium and present increased production due to exercise(46-48). The pharmacological inhibition of these substances synthesis reduces the peripheral blood flow(49-52). One may speculate hence, that there is a straight relation between the production of such substances derived from exercise and the PEH. However, some experiments did not confirm this hypothesis. For instance, a recent study(51) did not observe alteration in the blood flow in both control group and the one under inhibition of the prostaglandines. On the other hand, the PEH was identified in both cases. Concerning the nitric oxide, it was verified that the obstruction of such substance did not interfere in the BP and the vascular resistance after exercise reduction(52). Therefore, it is not possible yet to precise which substance is more responsible for the mechanism of the BP reduction after physical activity.

Concerning investigations that proposed to study the counter-resistance exercise, only one(33) investigated a physiological mechanism related with the PEH. The authors analyzed the variation of the natriuretic atrial peptide related to the PEH. Such hormonal substance is an agent with vasodilator properties and relates with the pressor control as well. However, no association between the natriuretic atrial peptide and the PEH was identified. Studies involving counter-resistance exercise and blood flow measurement were not identified either. Such intervention field still needs scientific background, since there is a demand for knowledge about cardiovascular responses and their applicability for individuals with special characteristics, such as cardiac hypertension patients(2).

CONCLUSION

The studies about PEH and counter-resistance exercise are scarce; therefore, one may not affirm which are the optimum exercise intensity and volume in order to optimize the BP reduction after activity. Despite of that, it is possible to speculate that, in monitoring periods close to 60 min, the counter-resistance exercise may provide PEH in normotensive and hypertensive individuals. Concerning intensity, it does not seem to be an interventional variable. Conversely, the work volume performed seems to affect the BP reduction after exercise, especially when several series are performed.

The physiological mechanisms that may explain the PEH are still somehow obscure. Nevertheless, the vascular resistance decrease by endothelial substances seems to play a relevant role in the phenomenon, independently of the cardiac debt and the SNA behavior.

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