The effect of acute slow resistance exercise on post-exercise blood pressure in active normotensive male recreational athlete

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Abstract
Physical activity is a non-pharmacological way to prevent or treat disorders like type 2-diabetes, obesity and hypertension. The aim of this study was to investigate The Effect of Acute Slow Resistance exercise on post-exercise blood pressure in active normotensive male recreational athlete. For this, Twelve recreationally active men were recruited, Brachial systolic blood pressure (SBP) and diastolic blood pressure (DBP) and heart rate were measured immediately before (pre) and after(post) and approximately 15 min(post-15), 30 min (post-30) and 60 min (post-60) after each exercise bout. Three resistance exercise bouts were performed: a high-intensity bout (HI), a low-intensity bout (LI) and a slow low-intensity (SL) resistance exercise bout. In each bout, 6 whole body exercises including: leg press, knee flexion, knee extension, bench press, lat pull down and biceps curl were performed. The SPANOVA test with repeated measures was used to analyze data. Our results showed that Significant time effect for SBP, DBP, MAP and HR (P<0.05). High intensity bout caused more stimulation for HR and HI and SL bout caused PHE (post-exercise hypotension) for DBP and MBP similarly greater than LI bout (P<0.05). In our study all exercise bouts promote systolic and diastolic PEH in active normotensive subjects, while HI and SL exercise decreases DBP more than LI. Perhaps because SL method could promote PEH as much as HI method, although exercise intensity was lower in SL, it is recommended for people with special disabilities who want to control hypertension with low exercise intensity and volume.

Keywords: Blood pressure, heart rate, slow contraction, strength exercise.

INTRODUCTION
Scientific literature has demonstrated exercise training and an acute exercise bout are relevant non-pharmacologic option for the prevention and treatment of blood pressure (BP) disorders, a number of investigators have examined the effects of chronic exercise training on resting blood pressure in hypertensive populations (22). It is generally accepted that the mechanisms underlying the sustained decrease in blood pressure of hypertensive individuals after training are a decrease in the resting heart rate and a decrease in circulating catecholamine (33). However, more recent investigations evaluating acute blood pressure responses in the prolonged post exercise period have addressed that an acute bout of exercise may transiently decrease resting blood pressure in the minutes or hours after exercise (18). Some authors have showed that after an acute exercise bout, BP levels are reduced for minutes or hours in relation to pre-exercise levels (19). This phenomenon (temporary reduction in blood pressure) has been termed post exercise hypotension (PEH), and considered as an important strategy in the control and reduction of BP (13,19).

An acute bout of steady-state aerobic exercise between 40% and 70% of peak oxygen consumption has consistently been shown to produce PEH (14), defined as a drop in blood pressure (BP) from baseline during recovery from exercise. This response typically onsets within 60 min following exercise (11). An acute bout of high-intensity interval exercise (HIIE) has also been shown to produce PEH (26). Resistance exercise (RE) is an activity mode that is commonly indicated as part of the treatment of diseases such as hypertension, aiding in obtaining increased cardiovascular function and increasing muscle mass and improving body composition (15). Some studies have found that an acute bout of resistance exercise will produce
HI (high intensity) method. Perhaps SL resistance exercise like Low-intensity BFR resistance exercise that has been found to stress the cardiovascular system to a greater degree than low-intensity non-BFR resistance exercise as evidenced by increased heart rate and BP and decreased stroke volume (SV) (owing to reductions in venous return) during exercise (29) and PEH after exercise bout (1). To our knowledge, the post-exercise BP response to traditional high intensity exercise training and BFR was investigated. Some researchers compared slow resistance exercise with BFR and traditional high intensity exercise training regarding to metabolic and hormonal and neuromuscular perspective (32).

To our knowledge, the effect of SL resistance exercise on PEH is not clear and any research not performed in this area. For this, the main purpose of this study was to determine whether PEH occur following low-intensity slow resistance exercise. A secondary purpose was to compare this response to both a work-matched low-intensity condition (LI) and a typically recommended traditional high-intensity exercise resistance exercise bout (HI).

**MATERIAL & METHOD**

Subjects

Twelve recreationally active men were recruited for this study. All the subjects were physically active and familiar with resistance training, but they had not participated in a regular resistance training program for six months before the beginning the study. Potential participants were screened with a health history questionnaire prior to participation in this study. Exclusion criteria included any known cardiovascular, pulmonary or metabolic disease (asthma, diabetes, hypertension, dyslipidaemia, etc.), orthopedic problems or self-report as a smoker. Subjects who presented a body mass index ≥ 24 kg/m² and fat mass > 20% were excluded. Complete advice about possible risks and discomfort was given to the participants, and all of them gave their written informed consent to participate. All procedures were in accordance with the declaration of Helsinki and the study was approved by the faculty ethics committee in the University of Shahid Rajaee Teacher Training, Tehran, Iran.

Procedures

Before initiating the tests, the participants underwent an anamnesis, a clinical evaluation and BP, body fat mass, body mass index, weight and
height measurements. Then all of them underwent familiarization session and participated in 1RM test. Afterwards, participants carried out three experimental sessions with a minimum of 72 h intervals: This study employed a randomized, cross-over design. Participants were tested >2 h postprandial and were instructed to avoid caffeine, medications and exercise on the day of testing. The participants were instructed not to ingest alcoholic or caffeinated drinks, not to perform strenuous physical activity in the previous 48 h and to have their last meal 2 h before the beginning of the experimental sessions. All three exercise bouts were performed at 10:00-12:00 AM to control diurnal variation in BP. The laboratory had a mean temperature of 21°C and mean relative air humidity of 40% to 45%.

Blood pressure measurements

Brachial systolic blood pressure (SBP) and diastolic blood pressure (DBP) were measured using a standard mercury sphygmomanometer (ALPK2, Japan), taking the first and the fifth phases of Korotkoff sounds as SBP and DBP values, respectively. BP was taken in duplicate, and if values were within 5 mmHg of each other, the average of the two values was used for analysis. BPs were measured until two values within 5 mmHg were obtained. Mean arterial pressure (MAP) was calculated as \((1/3)\) SBP + \((2/3)\) DBP. After a 5-min rest in the seated position, BP was measured three times during two different visits to the laboratory. On the occasion of each visit, BP was measured. Participants were excluded if the average of the last two values obtained during each visit for SBP and DBP was greater than 139 and 89 mmHg, respectively. Immediately before (pre) and after(post) and approximately 15 min(post-15), 30 min (post-30) and 60 min (post-60) after each exercise bout, BP, and heart rate was assessed.

1RM test

A week before the main testing session, subjects were recruited to performing 1 RM test. Before testing, subjects performed three warm-up sets after 5 min running and 5 min stretch exercises. The subject’s 1RM was determined by allowing three attempts to lift the heaviest weight by trial and error procedure. Subjects used their natural concentric and eccentric repetition speed to perform all warm up and 1 RM attempts and rested 3 minutes between sets. Subjects relaxed 3 min between repetitions and at least 5 min between exercises (5).

Exercise protocols

Initially, the volunteers remained seated in a comfortable chair for 20 min, with BP being assessed each 5 min from the 10th min to obtain average resting values. If the pre-exercise BP of participants were abnormal (SBP>139, DBP>89), the experimental session was postponed to another day. Then, the subjects who were randomly selected for one of the three protocols underwent 15 min warm-up consisting of 5 minutes of slow running, 5 minutes of static stretching, and 5 minutes of dynamic exercise and performed, Three resistance exercise bouts were performed: a high-intensity bout (HI), a low-intensity bout (LI) and a Slow low-intensity (SL) resistance exercise bout. In each bout, 6 whole body exercises including: leg press, knee flexion, knee extension, bench press, lat pull down and biceps curl were performed. For the HI, participants completed three sets of ten repetitions using 70% 1-RM with 1 min rest between all sets and all exercises. For the LI, participants completed one set of 30 repetitions followed by three sets of 15 repetitions using 20% 1-RM with 30 s rest between all sets and all exercises. For the SL resistance exercise, participants performed the same reps, sets and rest as in the LI while in SL contraction speed was lower than two other methods (SL, 3-3 method, 3 s for CON and 3 s for ECC action) Contraction speed has been monitored with the aid of metronome at a defined constant velocity during each method and an experienced assistant who announced the end of CON and ECC action.

Statistical analysis

All data were expressed as mean ± SD and were analyzed using SPSS software (v. 16.0). The Split-Plot Analysis of Variance (SPANOVA) with repeated measures (group × time) was used to analyze data and when the difference presented was significant, the Bonferroni post-hoc test was used for multiple comparisons, and one way ANOVA test was used to determine between group differences. When ANOVA revealed significant, Bonferroni method was used for post-hoc comparisons. For all tests, \(P<0.05\) was considered as significant.

RESULTS

Subject characteristics, including 1-RMs, are shown in Table 1. Table 2 compares the PEH and HR response between bouts (\(P<0.05\)). Significant time effect was found for SBP, DBP, MAP and HR (\(P<0.05\)). Significant post exercise reductions in SBP,
DBP and MAP were seen at majority of times following each protocol (P<0.05). A significant interaction effect (group × time) was seen DBP, MBP and HR variables (P<0.05). A significant between group differences are demonstrated in Table 2 (P<0.05) following all bouts, HR increased from baseline at 15,30 and 60 min post exercise and then decreased at 60 min post exercise with the HR being significantly higher in HI bout than SL and LI two other bouts (P<0.05).

Table 1. Subject characteristics and 1RM

<table>
<thead>
<tr>
<th></th>
<th>Height(m)</th>
<th>Weight(kg)</th>
<th>BMI(m²/kg)</th>
<th>Age(years)</th>
<th>Body fat (%)</th>
<th>1-RM(kg)</th>
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<tr>
<td>Height(m)</td>
<td>1.73±9.6</td>
<td>73.2±10.02</td>
<td>24.0±3.13</td>
<td>24.2±2.18</td>
<td>12.8±3.11</td>
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<tr>
<td>Weight(kg)</td>
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<td>BMI(m²/kg)</td>
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<td>Body fat (%)</td>
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<td>1-RM(kg)</td>
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DISCUSSION

To our knowledge, this is the first study to address BP and HR after resistance exercise with slow method (increasing time under tension). The aim of this study was to investigate the acute effects of three resistance exercises (HI, LI and SLI) on blood pressure and heart rate during recovery after 15, 30 and 60 minutes exercise bout. We tested this hypothesis if increasing time under tension (slow resistance exercise) could stimulate PEH in active Normotensive Male recreational athlete. The results of current study showed that HR increased significantly following all methods, but this increment was higher in HI group than other low intensity two groups (SL and LI). These results are in consistent with other researchers like Rossow et al. (26) that tested and compared low intensity and high intensity resistance exercise bout on HR 30min and 60min post exercise bout. Their results showed that following all bouts, HR increased from baseline at 30 min post exercise and then decreased at 60 min post exercise with the HR being significantly higher in HI than in LI or low-intensity BFR exercise at both time points. Also Rezk et al. (24) in a study compared effect of two types of resistance exercise with different intensities %80 1RM (high) and %40 1RM (low) on post exercise HR, and they found In comparison with the pre-intervention values, HR decreased throughout the post-intervention period in the Control session (P< 0.05), and increased significantly in the exercise sessions. Moreover, HR increase observed 15 min after exercise was greater in the HI (80%) than LI (40%) session (24). It is clear from these studies and our results that HR is dependent to exercise intensity and for this HI resistance exercise could increase it greater than LI resistance exercise even increasing time under tension and BFR can’t compensate intensity intervention.

Table 2. Comparison three bouts of resistance exercises.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Pre-exercise</th>
<th>Post-15min</th>
<th>Post-30min</th>
<th>Post-60min</th>
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<tbody>
<tr>
<td>Brachial SBP (mmHg)</td>
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<tr>
<td>HI</td>
<td>113.42±3.02</td>
<td>111.67±2.60*</td>
<td>108.50±3.35*</td>
<td>110.17±3.09*</td>
</tr>
<tr>
<td>LI</td>
<td>114.92±4.07</td>
<td>113.25±3.62</td>
<td>111.92±3.31*</td>
<td>112.82±3.08</td>
</tr>
<tr>
<td>SL</td>
<td>113.58±3.36</td>
<td>111.17±2.88</td>
<td>109.42±2.35*</td>
<td>111.16±2.40</td>
</tr>
<tr>
<td>Brachial DBP (mmHg)</td>
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<td></td>
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<tr>
<td>HI</td>
<td>73.5±3.11</td>
<td>72.25±3.22</td>
<td>69.75±2.86*</td>
<td>71±3.13</td>
</tr>
<tr>
<td>LI</td>
<td>74.16±3.27</td>
<td>72.83±3.29</td>
<td>71.58±3.02*</td>
<td>72.16±3.24*</td>
</tr>
<tr>
<td>SL</td>
<td>73.25±3.33</td>
<td>71.91±3.25</td>
<td>69.2±2.99</td>
<td>70.16±3.21*</td>
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<tr>
<td>Brachial MAP (mmHg)</td>
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<tr>
<td>HI</td>
<td>85.93±2.50</td>
<td>84.53±2.60</td>
<td>81.84±3.67*</td>
<td>83.21±2.13*</td>
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<tr>
<td>LI</td>
<td>86.87±2.12</td>
<td>85.44±2.08</td>
<td>84.17±2.06*</td>
<td>84.89±2.25*</td>
</tr>
<tr>
<td>SL</td>
<td>85.82±2.24</td>
<td>84.15±2.10</td>
<td>81.64±1.65*</td>
<td>82.99±2.05*</td>
</tr>
<tr>
<td>Heart rate (beats min⁻¹)</td>
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<tr>
<td>HI</td>
<td>64.08±3.11</td>
<td>81.91±3.26*</td>
<td>78.08±3.65*</td>
<td>79.08±2.90*</td>
</tr>
<tr>
<td>LI</td>
<td>63.08±2.53</td>
<td>75.38±3.83*</td>
<td>72.3±3.46*</td>
<td>66.5±2.23*</td>
</tr>
<tr>
<td>SL</td>
<td>64.25±3.84</td>
<td>77.75±4.3*</td>
<td>74.08±3.77*</td>
<td>68.91±2.6*</td>
</tr>
</tbody>
</table>

Values are mean ± SD; n = 12 for each group. SL, low-intensity with slow contraction resistance exercise bout; LI, low-intensity exercise bout; HI, high-intensity exercise bout; SBP, systolic blood pressure; DBP, diastolic blood pressure; MAP, mean arterial pressure; *P<0.05 versus pre-exercise; †P<0.05 versus post-15; ‡P<0.05 versus post-30; #P<0.05 versus high intensity; +P<0.05 versus low intensity; %P<0.05 time effect; £ P<0.05 condition by time interaction.
The results of current study showed that all three methods have reduced blood pressure and could potentially cause PEHE. With regard to literature, the data about PEH and resistance exercise is still scarce and controversial results have been reported (2). Our main finding was that all three type of exercise produced PEH, and this response was observed until 60 min post exercise. The present study found a significant PEH of SBP compared to pre-exercise measurements in the protocols tested. The reductions in SBP levels after a single exercise session are in consistent with the other studies results that observed PEH after resistance exercises. For instance, Rossow et al. (26) conducted a study and compared HI, LI and BFR methods of resistance exercise on SBP and they found that only HI could attenuate SBP after 60 min recovery from exercise. They explained these results by speculating that either BFR resistance exercise protocol was not intense enough or something, as yet unknown, intrinsic to BFR resistance exercise prevented the development of PEH (26). PEH following resistance exercise may be intensity dependent with PEH only elicited after exercise of higher intensities (28). In support of the idea that perhaps BFR protocol was not intense enough, recent work by Suga et al. (2010) found BFR exercise performed at 20% 1-RM, to not cause the same accumulation of intramuscular metabolites as high intensity (65% 1-RM) resistance exercise. In contrast, in our study it was revealed both low intensity resistance exercises could decrease SBP. Although, these decrements were not significantly different among groups in all times except 30 min post exercise bout that was significant differences between HI and LI. It is possible that there are the PEH intensity threshold for resistance exercise was simply not attained by our Li exercise. In a study performed by Arazi et al. (2), subjects performed two types of circuit resistance exercise with different rest intervals time, they concluded that the resistance exercise with RI30s and RI40s led to significant post-exercise decrease in SBP. Rezk et al. (24) also reported significant reduction of SBP after two resistance exercise sessions with different intensities in normotensive young individuals. Mota et al. (20) observed PEH of SBP after circuit model for resistance exercise composed of 13 resistance exercises that were performed with 20 repetitions at 40% 1RM and 30 seconds rest interval between exercises. In contrast to the results of the present study, Veloso et al. (35) observed no change in SBP after three resistance exercise protocols with RI of 1, 2 and 3 minutes between the sets, that consisted of three sets of eight repetitions in six exercises. Simões et al. (28) did not find significant differences between post-exercise measurements and rest measurements of SBP in type-2 diabetic and nondiabetic subjects after doing a resistance exercise session at 23% of 1RM. The discrepancy between the present results and other studies could be due to methodological differences as well as differences between subjects. Veloso et al. (35) used a protocol in which the load decreased at each set, with the purpose of maintaining the same work volume (load x repetitions) in all protocols. And some differences may be related to the amount of muscle mass involved in exercise. In the present study that performed 3 sets of 6 exercises for upper and lower limbs, exercising muscle mass were more than some studies that used only lower or upper limb exercises.

Regarding DBP, compared to pre-exercise values there were significant PEH of DBP observed in all measured moments during recovery period of exercise trials. In our study each protocol decreased DBP in majority of times tested after exercise bout, but interesting finding is that this decrement was same values are detected to HI and SL statistically and greater than LI group. One of the physiological mechanisms that could explain the influence of SL on blood pressure after resistance exercise is the time under tension increment, perhaps increasing concentric and eccentric action time (TUT) compensate low intensity and little volume load in SL method and could make acute PEH response similar to high intensity methods. Additionally, metabolic changes is essential to PEH responses after exercise bout, there may be a reduction in vascular resistance influenced by an accumulation of metabolites and ions (e.g. nitric oxide, prostaglandins, adenosine, H+ and K+) produced in muscle contraction (2), is one of the factors accounting for vasodilation and subsequent decrease in peripheral vascular resistance. As an explanation to our findings some authors like Tanmito et al. (31) found no significant differences between HI and SL methods on lactate responses(metabolic changes). Furthermore Mazzetti et al. (10) found that SL increased blood lactate concentration significantly more than HI resistance exercise. Some studies investigated PEH in DBP, for instance, In the Simão et al. (28) study, significant post-exercise decrease in DBP was also observed 10 minutes after completion of a protocol of 12 repetitions with a load of 50% of 6RM. Rezk et al. (24) also found significant post-exercise decrease in
DBP; however, the duration of PEH was longer (30 minutes) than that found by Simão et al. (28). In contrast, Polito et al. (23) did not observe significant variation in the DBP after resistance exercise (35). Also, Veloso et al. (33) observed no changes in DBP after resistance exercise with 2 min RI between resistance exercise sets but, significant reduction in DBP occurred after 1 and 3 min RI. Resistance exercise protocols typically differ among studies, and these differences may likely be responsible for some of the variations seen in the results. The resulting differences in the degree of metabolic stress produced by differences in exercise intensity, number of sets, rest intervals, contraction speed and stations of resistance exercise may be enough to affect recovery blood pressure.

It is clear that MBP is calculated from SBP and DBP and depending to its formula, DBP has greater effect (1 / 3 SBP + 2 / 3 DBP), for this reason, we concluded that MBP decrement is similar to DBP trend. For testing this anticipation our results showed within group and inter action differences among groups. Like DBP each group showed meaningful reduction in MBP, but this reduction was statistically higher in HI and SL than LI group. Our finding is in consistent with Rossow et al. (26) found that HI could decrease MBP more than LI and interesting finding is that this decrement was same in HI and SL statistically although exercise intensity was lower in SL than HI (20% to 80% of 1 RM).

As a result; in our study all exercises promote systolic and diastolic post-exercise hypotension in active normotensive subjects, while HI and SL exercise decreases DBP more than LI. This is novel findings if it is approved in further studies. Because SL method could promote PEH as much as HI method, although exercise intensity was lower in SL, this is maybe because of more metabolic change made by SL than other high speed methods, however, metabolic changes is not controlled in this study but is documented in literature. This method may be recommended to old and people with special disability like type 2 diabetic or participants in phase 3 or 4 heart rehabilitation program who are in rehabilitation and they must perform low intensity exercises but want more PEH and hemodynamic results.

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REFERENCES


