ABSTRACT

Introduction: Powerlifting (PWL) is a worldwide method, frequently used in resistance training programs. However, the relationship between cardiovascular responses and PWL is still unclear in the literature. Objective: To evaluate acute cardiovascular overload and post-exercise hypotension (PEH) after acute powerlifting exercise session in subjects with experience in the modality. Methods: Nine powerlifting athletes (34 ± 5 years) participated voluntarily in this study. The following exercises were used in the session: squat, bench press and deadlift (95% of 1 RM, 2-5 repetitions). The anthropometric parameters and blood pressure (systolic, diastolic and mean) were evaluated immediately, 5', 10', 30', 60' and 24 hours after the exercise session with a non-invasive automatic pressure monitor. Results: Significant differences (p<0.05) were found between rest and immediately after exercise on systolic (135 ± 6 vs. 153 ± 10 mmHg) and mean (102 ± 3 vs. 108 ± 3 mmHg) blood pressures, but no difference was found at diastolic (85 ± 3 vs. 85 ± 4 mmHg) blood pressure. Additionally, the increase in systolic pressure did not reach values considered as a risk of cardiovascular overload. Significant PEH was found after 60 minutes (systolic: -12 ± 12%, diastolic: -5 ± 6% and mean: -7 ± 5%) and 24 hours after PWL session (systolic: -5 ± 4%, diastolic: -8 ± 4% and mean: -7 ± 3%). Conclusion: Our data demonstrated that a PWL session does not increase systolic blood pressure up to the risk range and promotes PEH after 60 minutes of exercise and that this cardiovascular response persisted after 24 hours post-exertion in powerlifting athletes.

Keywords: hypotension; resistance training; exercise.
INTRODUCTION

Scientific evidences suggest that regular resistance exercise (RE) is an important strategy to control systemic blood pressure, in both normotensive and hypertensive subjects. Among the effects of physical activity on the cardiovascular system, post-exercise hypotension (PEH) has been studied in hypertensive subjects with clinically relevant implications. There are several studies utilizing RE to promote BP reduction at acute and chronic approaches. The mechanisms involved at PEH have been attributed to reduced peripheral vascular resistance, reduced sympathetic activity, and diminished systolic volume and changes in the sensitivity of adrenergic cardiac and endothelial factors.

The American College of Sports and Medicine and the American Heart Association stated that RE in association with an aerobic based exercise program are efficient to prevent, treat and control the arterial hypertension, however, the RE and PEH effect still unclear. In 2015 our group demonstrated that PEH occur independently of exercise intensity without expressed cardiovascular overload during the session, however, to the best of our knowledge, there are few studies on literature evaluating different manipulation of volume and intensity on RE and the magnitude of PEH.

In this perspective, powerlifting method consists of practice by exercises considered basic, such squat, bench press and deadlift, utilizing load near of maximal repetition test (1RM). Haslam et al. shown that higher loadings lead to larger increases in blood pressure and heart rate, additionally, the exercises utilized on powerlifting exercise session is composed of movements can lead to high values of BP and HR. According to ACSM recommendations high intensity of RE (over 80% of 1RM) had been used by athletes, recreational exercisers and fitness center practitioners such strategy to increase of muscular strength, however, there are a gap on literature about influence exercise intensities using load near of 100% of 1RM. In this way, the purpose of this study was evaluated the acute cardiovascular overload and PEH after acute powerlifting exercise session on subjects with experience PWL modality.

METHODS

After approval by the Ethics Committee for Human Research of São Judas Tadeu University (n° 90801), nine subject with experience on powerlifting exercise session on subjects with experience PWL modality. The inclusion criteria were: obesity with inability to exercise, involvement by muscle or tendon injury on the last month, being under treatment of infectious disease, using any type of medication that alters the cardiovascular, hormonal and / or metabolic responses, being submitted to a weight loss diet and/or any other medical contraindication to physical exercise. The inclusion criteria were: recent hospitalization, symptomatic cardiorespiratory disease1,2. Among the effects of physical activity on the cardiovascular system, post-exercise hypotension (PEH) has been studied in hypertensive subjects with clinically relevant implications3-9. The American College of Sports and Medicine1 and the American Heart Association2 stated that RE in association with an aerobic based exercise program are efficient to prevent, treat and control the arterial hypertension.

The systolic blood pressure (SBP), diastolic blood pressure (DBP), mean arterial blood pressure (MAP) and HR were measured before during, and immediately after each training session using an automated non-invasive BP monitor (Microlife ZAC1-IPC, Microlife, Widnau, Switzerland). Heart pressure product (HPP) was evaluated according to the following equation: RPP = HR*SBP. According to previously publication of our group, the measurement was performed after the subjects completed each set (a total of three); the objective of this measurement was to guarantee that BP did not fall during the exercise session. All BP measurements were taken on the left arm. Individual cuffs were labeled with the ranges of arm circumferences. Pre-exercise BP did not exceed 160 and 100 mmHg for SBP and DBP, respectively. During exercise, HR was continuously measured and recorded on a beat-by-beat basis using a Polar Vantage NV (Polar Electro, Oulu, Finland) HR recorder. Volunteers were also instructed to avoid the Valsalva maneuver during the entire movement, following American College of Sports Medicine guidelines. To evaluate the occurrence of PEH, BP, and HR were also measured on rest, in exercise
peak (immediately on final of exercise session) and in the sitting position (resting) at 5, 10, 30, 60 minutes and 24 hours after exercise session. To obtain MVO₂, we used a mathematical function based on a high correlation between heart pressure product and MVO₂. The following equation expressed in mlO₂/100gVE/min were used: MVO₂ = (DP x 0.0014) - 6.37.

**Statistical analyses**

All statistical analyses were performed using SPSS software (v. 15.0; IBM, Armonk, NY, USA). Analysis of comparisons between groups over the periods was performed with one-way analysis of variance with repeated measures, followed by Kruskal–Wallis or Bonferroni’s post-hoc test when appropriate. The D’Agostino–Pearson test was applied to Gaussian distribution analysis. Statistical significance was established at p<0.05. Data is expressed as mean ± standard deviation. Additionally, the magnitude of the training effect was calculated using the mathematical formula that considers the final average the final average value subtracted from the initial average value in relation to the variation of initial mean: [Pre-Post ES = Posttest mean – Pretest mean / Pretest SD]²¹.

**RESULTS**

The anthropometric parameters are described on Table 1. The hemodynamic analysis of powerlifting exercise session at rest, exercise peak and recovery are presented at Figure 1. Significant increase (p<0.01) was observed from rest to exercise peak in SBP (Panel 1A: rest 135 ± 6 mmHg; exercise peak: 153 ± 10 mmHg; 13 ± 4 %), MBP (Panel 1C: rest 102 ± 3 mmHg; exercise peak: 108 ± 3 mmHg; 6 ± 3 %), HR (Panel 1D: rest 77 ± 11; exercise peak: 160 ± 20 bpm; 50 ± 10 %), RPP (Panel 1E: rest: 10442 ± 1583 bpm*mmHg; exercise peak 24561 ± 4040 bpm*mmHg; 56 ± 10 %) and MVO₂ (Panel 1F: rest: 8.24 ± 2.27 mlO₂/100gVE/min; exercise peak 28.01 ± 1.88 mlO₂/100gVE/min; 69 ± 11%) but not to DBP (Panel 1B: 84 ± 3 mmHg; exercise peak 85 ± 3 mmHg; 0.99 ± 5 %). Additionally, the effect size in relationship rest versus exercise peak was large to SBP (2.65), MBP (2.18), HR (7.5), RPP (8.92), MVO₂ (8.92), but small to DBP (0.32).

During recovery, differences were found only on SBP (122 ± 9 mmHg) and MBP (95 ± 3 mmHg) at 60 min in relation at rest with trivial effect size to both parameters (-2.11 and -1.07) respectively. No differences were presented on others parameters.

The 24h analyses recovery could be observed at Figure 2. Statistical reductions (p<0.001) were found in all parameters 24h after powerlifting exercise session, at SBP (rest 135 ± 6 mmHg; 24h 128 ± 6 mmHg; -5 ± 3 %), DBP (rest 84 ± 3; 24h 78 ± 3 mmHg; -8 ± 4 %), MBP (rest 102 ± 3 mmHg; 24h 95 ± 3,2 mmHg; -7 ± 3 %) on Panel A, and HR (Panel B: rest 77 ± 3bpm; 24h 70 ± 2 bpm; -8 ± 9 %), RPP (Panel B: rest 10442 ± 1583 bpm*mmHg; 24h 9115 ± 1013 bpm*mmHg; -14 ± 12 %) and MVO₂ (rest: 8.24 ± 2.27 mlO₂/100gVE/min; 24h: 6.39 ± 1.42 mlO₂/100gVE/min; -29 ± 24 %) on Panel B. In all parameters the effect size of 24h post exercise to SBP (-1,05), DBP (-1,97), MBP (-2,16), HR (-0,56), RPP (-0,84), MVO₂ (-0,84,7), SBP (-1,05), MBP (2,18), HR (7,5), RPP (8,92) and MVO₂ (8,92) were trivial.

<table>
<thead>
<tr>
<th>Table 1. Sample characteristics.</th>
<th>Mean ± DP</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>34 ± 5</td>
</tr>
<tr>
<td>Bodyweight (kg)</td>
<td>92 ± 14</td>
</tr>
<tr>
<td>Stature (m)</td>
<td>1.75 ± 7</td>
</tr>
<tr>
<td>Fat body (%)</td>
<td>14 ± 3</td>
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<tr>
<td>Fat mass (kg)</td>
<td>13 ± 4</td>
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<tr>
<td>Fat mass (kg)</td>
<td>79 ± 10</td>
</tr>
<tr>
<td>Experience (years)</td>
<td>7 ± 3</td>
</tr>
<tr>
<td>PAS rest (mmHg)</td>
<td>135 ± 6</td>
</tr>
<tr>
<td>PAD rest (mmHg)</td>
<td>64 ± 3</td>
</tr>
<tr>
<td>Test 1RM squat (kg)</td>
<td>184 ± 22</td>
</tr>
<tr>
<td>Test 1RM bench press (kg)</td>
<td>153 ± 13</td>
</tr>
<tr>
<td>Test 1RM deadlift (kg)</td>
<td>202 ± 30</td>
</tr>
</tbody>
</table>

Figure 1. Values expressed as the mean ± standard error deviation of PHE to Panel A: systolic blood pressure (SBP), Panel B: diastolic blood pressure (DBP), Panel C: mean blood pressure (MBP), Panel D: heart rate (HR), Panel E: rate pressure product (RPP) and Panel E: myocardium oxygen volume (MVO₂) after powerlifting training session. Different letters indicate statistical differences (p<0.05) to ANOVA repeated measurements.
and hypertensive women after exercises at 50% of 1RM found significant reduction in SBP that persisted for at least 1 hour after a RT session and does not found effect on PHE magnitude after its modification.

Considering the influence of load at PHE, Fisher27 exercise session just in SBP. Duncan et al.3 and Hardy and Tucker26 found in this study the PHE were found after 60 min and 24 hour after PWL manipulation of volume and intensity on RE and the magnitude of PEH. In previous studies22-25 of external load (intensity) on PEH, demonstrating11,12 or absence22 PHE after exercise, are the variables that can promote increases on blood pressure during and after exercise. Another factor important consideration to PHE and its influence should be address to muscle mass involved in exercise and training session. Polito and Farinatti37 was not found changes PHE after leg extension exercise in different training models. Similar responses were found by D’Assunção et al.31 using exercises with small muscle mass compared large mass.

Therefore, muscle group or larger amount of muscle mass involved during exercise, cannot be the main reasons for changing the hypotensive effect. In this case the powerlifting modality the PHE may be due to the involvement of a larger number of joints in the same movement increase need for blood in the active region, increasing vasodilation, reduce peripheral resistance and favoring PHE6. Studies31,37,38 had showed differences on PHE during and post exercise. Another factor important consideration to PHE after RE28 can be involved on this effect. Simultaneously, other peripheral vasodilators such as nitric oxide, prostaglandins, adenosine, and potassium may also appear to influence the PHE31,34. It is speculated also that the blood lactate concentration interferes with the blood pressure response after exercise.15 The lactate accumulation could cause reduction in vascular resistance with consequent PHE. In this case, exercises performed with higher intensities would cause increased release of lactate30.

According to Negrão et al.30 the aspects of training load should be associated with hemodynamic response we should note the concern about the workload, and the percentage of 1 repetition maximum (% of 1RM), the number of series and number of repetitions, rest interval between series, factors that are associated with pressure changes during and after exercise session. Figueira Júnior et al.6 and Fagard46 considered the volume of strength training as number of series, repetition number and amount of exercise, are the variables that can promote increases on blood pressure during and post exercise. Another factor important consideration to PHE and its influence should be address to muscle mass involved in exercise and training session. Polito and Farinatti37 was not found changes PHE after leg extension exercise in different training models. Similar responses were found by D’Assunção et al.31 using exercises with small muscle mass compared large mass.

In 2015 Cavalcante et al.5 demonstrated that PHE occur independently of exercise intensity without expressed cardiovascular overload during the session, however, there are few study6,10-12 on literature evaluating different manipulation of volume and intensity on RE and the magnitude of PEH. In this study the PHE were found after 60 min and 24 hour after PWL exercise session just in SBP. Duncan et al.1 and Hardy and Tucker26 found a reduction in SBP that persisted for at least 1 hour after a RT session and Queiroz et al.1 found reduction of SBP after 24 hours after aerobic exercise. Considering the influence of load at PHE, Fisher27 studied normotensive and hypertensive women after exercises at 50% of 1RM found significant SBP reduction for 60 minutes. Moraes et al.28 also analyzed the hypotensive effect of resistance exercise in hypertensive middle-aged men with loads of 60% of 1RM significantly reduced SBP, DBP, and MBP, respectively, by an average of 16, 12, and 13 mmHg to pre-hypertensive values. To our knowledge, there is only one study Brandão Rondon et al.29 that showed a reduction in SBP, DBP, and MBP for 60 minutes simultaneously.

Figure 2 Values expressed as the mean ± standard error deviation of PHE to Panel A: systolic blood pressure (SBP), diastolic blood pressure (DBP) and mean blood pressure (MBP), Panel B: heart rate (HR), rate pressure product (RPP) and myocardium oxygen volume (MVO₂) after powerlifting training session. *p< 0.01 indicate statistically significant differences to rest.

**DISCUSSION**

The major new findings of the present study were 1) the powerlifting training sessions does not promote hemodynamic overload towards to range risk, 2) the exercise session promote decreased on SBP after 60 min of exercise session, and 3) the PHE persisted after 24 hours after powerlifting training sessions.

Important information should be address in this study, although had been used very high training intensities (>90% of 1RM), the exercise session does not promote hemodynamic overload during the practice. Some important consideration should be pointed about this fact, although the powerlift practice is usually done with high intensity, the values of systolic blood pressure was not high then (160 mmHg).

Traditionally, powerlifting training sessions often use small volumes and high intensity could reach 100% of 1RM13,14, which could lead to a sharp adjustment that provide unique and differentiated hemodynamic responses. There are few studies3,5,6,10-12 on literature that evaluated the impact of alterations of external load (intensity) on PEH, demonstrating11,12 or absence22 PHE after load modification. In relationship to training volume, previous studies22-25 does not found effect on PHE magnitude after its modification.

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Though the PHE had been much studied, the mechanisms responsible for decreases in post resistance exercise BP are outside the scope for this study, however, these have not been completely elucidated in the scientific literature30. Previous studies5,6,10, suggest that several mechanisms can influence the cardiovascular and PHE behavior. So, overall the hypotensive effect may be associated with factors such as occlusion of the vessels and arteries, cardiac output, stroke volume, autonomic modulation of heart rate through the sympathetic and parasympathetic nerves, in addition to peripheral vascular resistance that can be changed during training30-32.

In relation to autonomic modulation evidences indicate that at higher intensities (above 80%) appears to be a reduction in cardiac output mediated decrease in stroke volume (SV)31. The drop in SV would be offset by an increase in HR caused by increased sympathetic activity and parasympathetic reduction in heart. Additionally, the vasodilation mediated by the action of kallikrein system / kinin which also have higher concentrations after RE28 can be involved on this effect. Simultaneously, other peripheral vasodilators such as nitric oxide, prostaglandins, adenosine, and potassium may also appear to influence the PHE31,34. It is speculated also that the blood lactate concentration interferes with the blood pressure response after exercise15. The lactate accumulation could cause reduction in vascular resistance with consequent PHE. In this case, exercises performed with higher intensities would cause increased release of lactate30.

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Therefore, muscle group or larger amount of muscle mass involved during exercise, cannot be the main reasons for changing the hypotensive effect. In this case the powerlifting modality the PHE may be due to the involvement of a larger number of joints in the same movement increase need for blood in the active region, increasing vasodilation, reduce peripheral resistance and favoring PHE. Studies31,37,38 had showed differences on mono and multi-joint exercises. In this case, the exercise performed in this study by powerlifting athlete has multi-joints characteristics associated with large muscle groups, which may have enhanced the hypotensive effect15.

Although few studies have evaluated the effects of strength training with high intensity in the cardiovascular response, we noted that the elevation of systolic and diastolic blood pressure increase progressively, reaching higher values in the last repetition, and early fatigue. In general, it can be seen that low intensity and high reps are more elevated blood pressure13,15. Muscle mass exerts influence on cardiovascular response, on the other hand, there is still no consensus in the literature on the hypotensive effect. We can observe that when used the same intensity and the same amount in exercises involving larger mass muscle produces significant increase in blood pressure. Furthermore, the Valsalva maneuver may exert influence on the increase hypotensive, the Valsalva maneuver is inevitable with intensity equal or to greater than 80% of 1RM. However, in this study, the athletes perform several sets, interspersed with periods of
rest between 3-5 minutes which allowed full recovery of systolic pressure, hence its increase in subsequent sets will be higher.

The hypothesis for PHE on strength training with high intensity for powerlifting practitioner may be related to decreased cardiac output caused by a reduction in stroke volume. The increase in stroke volume is related to an increase in cardiac contractility, which led to gradual decrease in end systolic volume, in other words, greater volume of ventricular filling during diastole. The results obtained in this study may provide relevant information about PHE, the variables possibly related to hypotension mechanisms as sympathetic activity, blood flow, cardiac output and production of nitric oxide have not been evaluated. However, this experience becomes relevant, since it is the first to directly investigate the influence of the intensity of strength training on multi-joints exercises, involving the greatest amount of muscle mass and low number of repetitions, and the response of the PHE effect.

Our study does have some limitations that should be mentioned. First, the mechanisms of hypotension were not investigated in the present study. We did not assess the exercise effects on peripheral vascular resistance as well as sympathetic activity, systolic volume, beta-adrenergic receptors, or endothelial factors. Second, the auscultation method used for assessing blood pressure. This method, while universally used, has limitations compared to invasive methods, such as intra-arterial catheterization; however, all safeguards were taken to ensure that these measures were obtained in a consistent, reliable and accurate manner. Third, the number of subjects on sample and a lack of control group (not athletes) may be useful to comparative effects.

CONCLUSION

In summary, the data presented in this study show that PHE effect in powerlifting practitioners after 60 min and 24 hours after exercise session without promote hemodynamic overload during an exercise session in PWL athletes.

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All authors declare that there is no potential conflict of interest this article.