Tittle: Effects of dynamic resistance training on post-exercise hypotension and its mechanisms in hypertensive men.

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Conflicts of Interest

We declare that the authors do not have any conflict of interest.

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ABSTRACT

Background: A possible chronic effect of exercise training is the attenuation of the acute decrease in blood pressure observed after the execution of a session of exercise (i.e. called post-exercise hypotension - PEH). However, there are few empirical data regarding this issue, and the possible mechanisms involved in this blunted response have not been studied. **Aims:** To evaluate the effects of dynamic resistance training (DRT) on PEH and its systemic, vascular, and autonomic mechanisms. **Methods**: Data from 16 middled-aged treated hypertensive men who underwent DRT (8 exercises, 50% of 1RM, 3 sets until moderate fatigue) 3 times/week for 10 weeks were analyzed. Before and after the training period, the participants underwent an experimental session in which blood pressure (auscultation), systemic hemodynamics (CO_2 rebreathing), vascular function (duplex ultrasound) and cardiovascular autonomic modulation (spectral analysis of heart rate and BP variability) were assessed before and after a session of DRT. **Results:** DRT reduced pre-exercise systolic BP and mitigated the systolic PEH, which occurred before but not after the training period ($P = 0.017$). DRT did not change the diastolic PEH that occurred with similar magnitude before and after the training period ($P = 0.024$). DRT did not change the PEH mechanisms, except for cardiac sympathovagal balance that increased significantly more after the session of DRT conducted in the post-training evaluation ($p = 0.017$). **Conclusion:** In medicated hypertensive men, 10 weeks of DRT decreased pre-exercise systolic BP, abolished systolic PEH, and induced a greater increase in post-dynamic resistance exercise sympathovagal balance.

INTRODUCTION

Dynamic resistance training (DRT) is characterized by the execution of skeletal muscle contractions against an external load, producing joint movement with alternations between concentric and excentric muscle actions[1]. DRT is recommended as a complementary therapy for the treatment of hypertension by the current guidelines[2–4]. Previous meta-analyses showed that a period of DRT can reduce systolic/diastolic blood pressures (SBP/DBP) by -6/-3 mmHg in hypertensives[5]. Additionally, it has been shown that a single session of dynamic resistance exercise can acutely decrease blood pressure (BP) during the recovery period[6]; which has been called post-exercise hypotension (PEH)[7].

Pre-exercise BP is an important predictor of PEH as patients who present higher pre-exercise BP show greater reductions in BP after a session of exercise[8]. Therefore, as a period of exercise training reduces BP, researchers have raised the hypothesis that PEH may be blunted or mitigated after training. However, there are few empirical data testing this hypothesis, and the investigations involving DRT have shown controversial results with studies showing that PEH was blunted[9] or unaltered[10,11] after a period of training. Additionally, these previous studies have not elucidated the possible mechanisms involved in the changes of PEH after DRT.

Recently, we conducted a randomized clinical trial that investigated the acute and chronic effects of DRT on BP and its mechanisms in hypertensives. Acutely, we observed that a session of DRT decreased BP, increased brachial vascular conductance, and decreased baroreflex sensitivity in the post-exercise period[12]. Chronically, we observed that 10 weeks of DRT decreased BP and improved vascular function[13]. In these previous studies, however, we did not investigate the effects of a period of DRT on the acute cardiovascular responses observed after the execution of a session of dynamic resistance exercise, which can contribute to the understanding of the relationship between the acute and the chronic responses of BP to DRT. Thus, the objective of the present study was to evaluate the effects of 10 weeks of DRT on the acute post-exercise responses of BP and its mechanisms to a session of dynamic resistance exercise.

METHODS

Sample

This study used data from a bigger trial registered at the Brazilian Clinical Trials (https://ensaiosclinicos.gov.br/rg/RBR-4fgknb), which employed 4 parallel groups: DRT, isometric handgrip training, combined resistance training and control. All procedures were approved by the Ethics Committee of the School of Physical Education and Sport, University of São Paulo (process number 2.870.688). As mentioned in the introduction, the results of this trial concerning the acute and chronic effects of these training protocols on office BP and its mechanisms have already been published elsewhere[12,13]. The novelty of the present study was to evaluate the effect of 10 weeks of DRT on the acute post-exercise responses to this exercise modality that is recommended for hypertensives. All participants were informed of the benefits and risks of the study prior to providing written consent. The experimental procedures were performed at the School of Physical Education and Sport of the University of São Paulo, while preliminary medical evaluation was performed at the Hospital das Clínicas of the Faculty of Medicine of the University of São Paulo.

Male adults with hypertension and aging between 30 and 65 years old were invited to participate. Recruitment was performed through the University of São Paulo's social media. To take part in the study, the participant had to be under the same anti-hypertensive pharmacological treatment for at least the previous 4 months and should not be physically active (defined as <150 min/week of leisure physical activity, ≤ 2 exercise training sessions per week, and not having performed regular resistance training in the previous 6 months). The exclusion criteria were: (i) presence of secondary hypertension; (ii) presence of hypertension-induced target-organ damage; (iii) use of medications that act directly on sinus node affecting cardiac chronotropism (i.e., betablockers and non-dihydropyridine calcium channel blockers); (iv) presence of any cardiovascular disease besides hypertension; (v) presence of contraindication or limitation to do resistance training; (vi) presence of symptoms or electrocardiographic alterations during a graded maximal exercise test; (vii) body mass index ≥ 35 kg/m^2; (viii) presence of diabetic complications or insulin use; and (ix) SBP/DBP > 160/105 mmHg.

Inclusion and exclusion criteria were checked during preliminary procedures. For that, in a visit to the laboratory, the participants answered a survey and underwent anthropometric and BP measurements. The survey included questions about their health conditions, regular medication use, and physical activity habits. The participants also fulfilled the International Physical Activity Questionnaire[14]. Their weight and height were measured (Welmy® W300A, São Paulo, Brazil), and their body mass index calculated. BP was assessed through the auscultatory method using a calibrated aneroid sphygmomanometer (Mikatos, Missouri, São

Paulo, Brazil) after the participants were seated for a minimum of 5 minutes and readings were taken three times on each arm. This BP evaluation was repeated in a subsequent visit carried out at least 2 days apart. The six readings obtained for each arm were averaged and the highest mean value between the arms was recorded as the participant's BP. In another visit, a medical evaluation was performed, and urine and blood samples were collected to rule out secondary hypertension and target-organ damage as recommended by the Brazilian Hypertension Guidelines[2]. Afterwards, in another visit, the participants underwent a progressive maximal exercise test with electrocardiographic responses monitored and analysed by a physician.

The participants who met all the study criteria underwent two familiarization sessions to the exercises employed in the study as already described elsewhere[15]. Briefly, in these sessions, they performed two sets of 20 repetitions using the lowest workload allowed by each piece of exercise equipment (Edge Line, Movement Fitness, Sao Paulo, Brazil) for the following eight dynamic resistance exercises: bench press, leg press, lat pull down, left leg extension, right leg extension, arms curl, left leg curl, and right leg curl. On a separate day, the participants completed one-repetition maximum tests (1RM) for all the aforementioned dynamic resistance exercises following a standardized protocol[16].

Procedures

The primary outcome for this study was PEH induced by dynamic resistance exercise, and the secondary outcomes were PEH mechanisms, including systemic hemodynamics, cardiovascular autonomic modulation, and vascular function.

Data from the participants who were randomly assigned to DRT in the bigger trial and had participated in experimental sessions to assess PEH before and after the training intervention were used for this study.

The intervention period lasted 10 weeks, with three training sessions conducted per week. Each session was supervised by an exercise specialist and conducted at the institution's gym facility. 1RM tests were conducted before and after the training period. PEH and its mechanisms were evaluated in the experimental sessions conducted before and after the training period. The experimental sessions conducted after the training period were performed with a minimal interval of 48 h after the last training session.

Experimental sessions

Prior to the experimental sessions, the participants received the following instructions: (i) refrain consuming vitamin supplements within the previous 72 hours, (ii) avoid exercise within the last 48 hours, (iii) abstain from alcoholic beverages within the last 24 hours, (iv) do not smoke for the last 8 hours, (v) maintain regular daily activities and sleep patterns in the previous day, (vi) take usual medication as prescribed, and (vii) arrive for the experimental session after fasting for at least 8 hours. The experimental sessions began between 7:00 and 7:30 a.m., and the laboratory temperature was maintained between 20 and 22 °C.

The experimental sessions were composed of 3 moments: pre-exercise, exercise, and post-exercise. Pre-exercise assessments started after 10 minutes of seated rest. Continuous signals of ECG, photoplethysmographic BP and respiration were recorded for 10 minutes to evaluate cardiovascular autonomic modulation. Photoplethysmographic BP was measured on the non-dominant limb. Subsequently, auscultatory BP (assessed on the dominant limb), cardiac output (CO), and heart rate (HR) were measured in triplicate to evaluate systemic hemodynamics. Finally, to evaluate vascular function, participants changed to the supine position and after 10 minutes, images and Doppler flow signals of the brachial artery of the dominant limb were recorded for 1 minute. Then, the participants moved to a gym where they executed a session of DRT. The exercises were executed with the same relative intensity before (i.e. with 50% of 1RM measured before training) and after the training period (with 50% of 1RM assessed after 10 week of the training). Afterwards, for the post-exercise assessments, the participants returned to the laboratory and remained resting in the seated position for 60 minutes. During this period, cardiovascular autonomic modulation was assessed from 30 to 40 minutes, and systemic hemodynamics was evaluated between 40 and 55 minutes. Then, the participants lied down, and vascular function was assessed from 65 to 70 minutes.

Measurements during the experimental sessions

BP and systemic hemodynamic assessment - Auscultatory BP was measured by a trained evaluator using a calibrated aneroid sphygmomanometer (Mikatos, Missouri, Sao Paulo, Brazil). Measurements were done on the dominant arm employing an adequate cuff size. SBP and DBP were respectively defined as phases I and V of Korotkoff sounds. Mean BP (MBP) was calculated as: $MBP=DBP+ [1/3 \times (SBP - DBP)]^2$. HR was assessed by radial pulse palpation for 15s and checked with the ECG signal (Cardioperfect, ST 2001 model,

Netherlands). CO was estimated through the indirect method of Fick[17], employing the CO_2 rebreathing technique and a metabolic cart (CPX Ultima, Medical Graphics Corporation) as reported elsewhere[18]. Systemic vascular resistance (SVR) was calculated as SVR = MBP/CO and stroke volume (SV) as SV = CO/HR.

Cardiovascular autonomic modulation - Cardiovascular autonomic modulation was assessed by the spectral analyses of HR and BP variabilities according to "Task Force" consensus guidelines[19]. For that, R-R intervals were obtained by ECG (Cardioperfect, ST 2001 model, Netherlands), beat-by-beat BP by finger photoplethysmography (Finometer, Finapress Measurement System, Arnhem, Netherland) and breath-by-breath respiration by a piezoelectric belt (UFI, Pneumotrace2, Morro Bay, USA) positioned at the subjects' thorax. These signals were continuously recorded for 10 min through a data acquisition system (Windaq, Dataq Instruments, Akron, Ohio, USA) with a sample rate of 500Hz per channel. Then, they were exported to Heart Scope II software (A.M.P.S. LLC, Version 1.3.0.3, New York, USA) that created temporal series of R-R intervals, respiration and SBP. Afterwards, the variabilities of these series were analysed in the frequency domain in stationary segments of 200 to 300 heartbeats, employing the autoregressive modelling and applying the Akaike criteria for choosing the order of the model. As indicated in the Task Force guidelines[19], cardiac sympathovagal balance was represented by the ratio between the low and high frequency components of the HR variability (LF/HF), vasomotor sympathetic modulation by the low frequency component of SBP variability (LF_{SBP}), and spontaneous baroreflex sensitivity (BRS) by the maximum magnitude of the transfer function between R-R interval and SBP variabilities at the low-frequency component.

Local hemodynamics - Brachial vascular parameters were evaluated through vascular ultrasound according to Expert Consensus guidelines[20]. Duplex doppler ultrasound was employed using a linear transducer [L9-3 probe, GE, Logiq E (R7), California, USA] positioned on the anteromedial arm region, ~5 cm distal to antecubital fossa, to assess the brachial artery of the dominant arm. Brachial artery images and blood flow velocities were obtained for 1 min. From these records, arterial diameter was determined by an automatic software (QUIPU – Cardiovascular Suite, Pisa, Italy), and brachial blood flow (BF) was calculated by the product between arterial cross-sectional area and blood flow velocity, being expressed in ml/min. Vascular conductance (VC) was calculated by dividing BF by MBP and was expressed in $ml.min^{-1}.\ mmHg^{-1}$.

Interventions

DRT was composed of the 8 dynamic resistance exercises mentioned earlier and executed on a specialized equipment (Edge Line, Movement Fitness, Sao Paulo, Brazil). In each exercise, participants executed 3 sets of repetitions until moderate fatigue (defined by a visual reduction of the movement velocity) with an intensity of 50% of 1RM, and keeping intervals of 90 seconds between the sets and the exercises. During the training period, exercise intensity was increased by 2-5% for the upper limb exercises and by 5-10% for the lower limb exercises whenever the participants were able to perform more than 15 repetitions without experiencing moderate fatigue in two consecutive sets. This DRT protocol followed the recommendations for hypertension management[2–4].

Statistical analysis

Sample size was defined *a priori* for the bigger trial and accepted by convenience for this study. Data normality was checked by Shapiro–Wilks' test and outliers were identified through box plots. Non-normal data were transformed by natural logarithm and normal distribution was achieved.

Paired t-test were used to compare maximal strength and exercise loads used in the acute exercise sessions before and after 10 weeks of training. The chronic effect of DRT on PEH and its mechanisms was analysed by two-way repeated measures ANOVAs considering study phase (before vs. after 10 weeks of training) and session moment (pre-exercise vs. post-exercise) as within-group factors. When significant main effects or interactions were observed, pairwise comparisons were done by Newman-Keuls post-hoc tests. Data are presented as mean ± standard deviation, and significance level was set at P value < 0.05 for all analyses.

RESULTS

For the bigger trial, 17 participants were randomly allocated to DRT. From them, 16 completed the entire protocol and underwent the experimental sessions before and after the training period, composing the sample of the present study. The sample characteristics are presented in Table 1. Ten weeks of DRT significantly increased 1RM in all exercises (all $p < 0.05$). All exercises were performed with significantly higher loads after the training (bench press: 27±7 vs. 33±10 kg; leg press: 55±12 vs. 71±13 kg; lat pull down: 23±5 vs. 29±6 kg; left

leg extension: 19±6 vs. 24±6 kg; right leg extension: 19±4 vs. 24±4kg; arms curl: 18±5 vs. 24±7kg; left leg curl: 12±2 vs. 15±5 kg; and right leg curl: 11±2 vs. 15±3kg, all $p < 0.05$) to result in the same relative intensity of 50% of 1RM.

Considering the effect of DRT on PEH and its mechanisms (Table 2 and Figure 1), ANOVAs revealed a significant study phase*session moment interaction for SBP and LF/HF (both $P<0.05$), and significant main effects of study phase for DBP, VS, HR, BRS and VC (all $P<0.05$).

Therefore, in the experimental session conducted before DRT, SBP decreased significantly from pre- to post-exercise, while in the experimental session conducted after 10 weeks of DRT, SBP did not change from pre- to post-exercise. Additionally, pre-exercise SBP was significantly lower in the experimental session conducted after than before the training period. DBP decreased significantly and similarly after the session of dynamic resistance exercise before and after DRT.

Regarding the PEH mechanisms, SV and BRS decreased significantly (both $P<0.05$), while HR and VC increased significantly (both $P<0.05$) after the session of dynamic resistance exercise, and these changes were similar before and after DRT (all $P>0.05$). On the other hand, LF/HF increased significantly after the acute session of dynamic resistance exercise before and after DRT ($P<0.05$), but the increase was significantly greater after DRT ($P<0.05$).

DISCUSSION

The main finding of the current study was that PEH was abolished after 10 weeks of DRT in medicated hypertensive men, and this response was accompanied by an enhanced increase in post-exercise sympathovagal balance.

Regardless of the study phase, the current study observed acute post-exercise responses to dynamic resistance exercise in line with previous literature. Before training, a single session of dynamic resistance exercise produced a mean decrease of -6±5/-2±6 mmHg in SBP/DBP during the recovery, which is within the range observed in previous meta-analyses[6]. Additionally, as observed in other studies[21–23], PEH occurred concomitantly with decreases in SV and BRS, and increases in HR, LF/HF_{R-R} and VC. Interesting, PEH did not present a main systemic hemodynamic determinant, since both CO and SVR were unchanged after the dynamic resistance exercise both before and after DRT. This absence of a specific change in the

whole sample may result from the high inter-individual variability of PEH hemodynamic determinant, with some subjects presenting a decrease in CO and others a decrease in SVR as already reported in a previous study from our group[15].

The current study also revealed, as hypothesized by the researchers, that PEH was influenced by a period of training given that a significant interaction was found between the study phase and session moment (pre- vs. post-exercise) for SBP. Such result indicates that systolic PEH was abolished after 10 weeks of DRT (-6±5 vs. -2±3 mmHg, P=0.017). Although one previous study did not find any change in PEH after DRT in normotensive individuals[10], the two studies[9,11] that have evaluated hypertensive individuals found blunted PEH after DRT. Thus, the current finding reinforces that DRT can abolish PEH in hypertensives. It is important to highlight that this abolishment was observed despite the fact that the absolute load of the acute exercise was increased after the training to maintain the relative exercise intensity, which supports that the abolishment of systolic PEH reflects a true effect of training and not a response to a lower exercise stimulus after the training.

The abolishment of PEH observed after 10 weeks of DRT may be explained by the chronic BP-lowering effect of DRT. Indeed, pre-exercise BP is considered an important determinant of PEH and, in a previous study[8], we demonstrated pre-exercise BP as its main predictor. Looking for this association in the present data, a complementary analysis also revealed a significant correlation ($r = 0.403$; $p = 0.022$) between the SBP decrease after the dynamic resistance exercise and the pre-exercise SBP. Therefore, the lower PEH observed after 10 weeks of training may be related to the fact that training has decreased pre-exercise SBP (125±12 vs. 119±12 mmHg). Strengthening this argument, pre-exercise diastolic BP was not reduced by training in the present study and thus diastolic PEH was not blunted after the training period (-2±6 vs. -2±4 mmHg, P=0.967). The abolishment of PEH after training may have clinical implications. PEH helps to reduce cardiovascular overload when BP levels are elevated, but a decrease in BP when BP levels are normal may impar tissues perfusion. Therefore, as systolic PEH occurred before training when the patients presented elevated SBP and was abolished after the training when the patients present lower SBP, this abolishment may represent a beneficial adaptation induced by training.

Considering the possible mechanisms involved, to the best of our knowledge, this is the first study to evaluate systemic, vascular, and autonomic mechanisms of PEH before and after a period of DRT. Only the increase in LF/HF_{R-R} after the acute session of dynamic resistance exercise was amplified after DRT (+0.77±1.01 vs. +1.55±1.02, P=0.017), while the post-

exercise responses of all other mechanisms (i.e., SVR, CO, SV, HR, LF_{SBP}, BRS, BF and VC) were not altered by training. Thus, it can be proposed that the greater increase in post-exercise cardiac sympathovagal balance after 10 weeks of DRT might have contributed to mitigate PEH, and this mechanism should be specific explored by future studies. It can also be speculated that the greater increase in post-exercise cardiac sympathovagal balance after the training was due to the higher total absolute volume load of the dynamic resistance exercise session employed after the training (9,305±2,084 vs. 7,241±1,447 kg.repetitions, $p<0.001$), once the absolute load was increased after the training. However, such influence may be unlikely considering that the relative intensity was maintained (50% of 1RM). Nevertheless, future studies should investigate this aspect comparing the effects of a period of DRT on post-exercise cardiac autonomic modulation after exercises conducted with similar and different total absolute volume loads. Interesting, although the increase in LF/HF_{R-R} after the acute dynamic resistance exercise session was amplified by DRT, the post-exercise responses of LF_{SBP} remained unchanged, indicating a selective impact of the training on cardiac and not peripheral autonomic response to acute exercise. Indeed, a similar behaviour was reported in a study that showed a decrease in BP without any change in muscle sympathetic nerve activity after DRT[24].

It is important to mention the limitations of the current study. It involved middle-aged hypertensive men who had no cardiovascular disease. Considering that men and women present different magnitude of PEH[25] and different PEH´s mechanisms[26], both sexes should not be jointed in a single sample. Thus, as a first investigation, a male sample was chosen for convenience, and future studies should investigate women. Thus, caution is required when extrapolating these findings to women, as well to elderly and individuals with other cardiovascular conditions. In addition, it is crucial to note that the results are specifically limited to the DRT protocol employed in this study. Alternative training protocols might yield different outcomes. However, it is worth mentioning that the DRT protocol employed in the present study was based on the recommendations from hypertension guidelines[2–4], and probably results would not differ with similar training protocols. Yet, it is important to highlight that the current study found acute cardiovascular responses after dynamic resistance exercise which are in line with previous literature.

CONCLUSION

Ten weeks of DRT abolishes PEH and enhances the increase in sympathovagal balance observed after a session of DRE in medicated hypertensive men.

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428 **FIGURE LEGEND**

429

430 Figure 1 – Post-exercise responses of systolic blood pressure (ΔSBP), diastolic blood pressure
431 (ΔDBP), cardiac output (ΔCO), systemic vascular resistance (ΔSVR), stroke volume
432 (ΔSV), heart rate (ΔHR), ratio between low- and high-frequency bands of R-R interval
433 variability ($\Delta LF/HF_{R-R}$), low-frequency band of systolic blood pressure variability
434 (ΔLF_{SBP}), baroreflex sensitivity (ΔBRS), blood flow (ΔBF) and vascular conductance
435 (ΔVC) measured before and after 10 weeks of dynamic resistance training. Data: mean
436 ± standard error. ln = natural logarithm. Analysis: Two-way ANOVA. # = different from
437 pre-training. Note: Decreased sample size (n=15) for the autonomic and local vascular
438 variables due to technical issues.