CLINICAL SCIENCE

Post-resistance exercise hypotension in patients with intermittent claudication

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OBJECTIVE: To verify the acute effects of resistance exercise on post-exercise blood pressure in patients with intermittent claudication.

METHODS: Eight patients randomly underwent two experimental sessions: a session of resistance exercise (R: 6 exercises, 3 sets of 12, 10 and 8 reps with a perceived exertion of 11 to 13 on the 15-grade Borg scale) and a control session (C: resting on exercise machines).

RESULTS: Before and for 60 min following an intervention, auscultatory blood pressure was measured while subjects rested in a sitting position. After the C session, systolic, diastolic and mean blood pressures did not change from the pre-intervention values, while these values decreased significantly after the R session throughout the entire recovery period (greatest decreases = -14 ± 5, -6 ± 5, and -9 ± 4 mmHg, respectively, $P < 0.05$).

CONCLUSION: After a single bout of resistance exercise patients with intermittent claudication exhibited reduced systolic, diastolic and mean blood pressures, suggesting that acute resistance exercise may decrease cardiovascular load in these patients.

KEYWORDS: Blood pressure; Strength exercise; Peripheral artery disease; Recovery; Cardiovascular load.

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INTRODUCTION

Peripheral arterial disease is mainly caused by an atherosclerotic pathophysiological process that alters the normal structure and function of the arteries of the lower extremities, decreasing peripheral blood flow, and leading to symptoms of intermittent claudication (IC).[1] Hypertension affects almost 90% of the patients with intermittent claudication (IC).[2] Since high blood pressure (BP) levels are directly related to acute cardiovascular events,[3] hypertension might be associated with the high cardiovascular mortality in these patients.

Exercise training is known to improve walking capacity of patients with IC,[4-9] therefore, it is recommended as the first line of treatment for these patients.[1] However, considering the high cardiovascular risk of IC patients,[10] improvements on cardiovascular function should also be targeted by means of exercise therapy. Exercise training is also widely recommended for decreasing BP[11] and reducing cardiovascular mortality.[12-14] According to the American College of Sports Medicine,[11] a single bout of aerobic exercise can lead to a significant and long lasting decrease in BP during the recovery period, thus decreasing cardiovascular load. This phenomenon is known as post-exercise hypotension,[15] and has clinical relevance for hypertensive patients.

In a recent study,[16] resistance training was shown to be as effective as walking training for improving walking capacity in patients with IC. Moreover, it has been observed that a single bout of resistance exercise decreases BP during the recovery period in healthy[17,18] and hypertensive[19] subjects without IC. However, the cardiovascular effects of resistance exercise in patients with IC are unknown. Since patients with IC have altered cardiovascular function and regulation, such as increased peripheral vascular resistance,[1] endothelial dysfunction,[20] and an increase in cardiovascular sympathetic modulation to the heart,[21] the hypotensive effect of resistance exercise is likely to be blunted in these patients. Thus, the purpose of this study was to analyze the acute effects of a single session of resistance exercise on post-exercise BP in patients with IC.

METHODS

Subjects

Eight patients with peripheral arterial disease, enrolled in a tertiary center specialized in vascular disease, were invited to participate in this study. Patients were included if they met the following criteria: Fontaine stage II of peripheral arterial disease, symptoms of IC for at least 6 months, ankle/brachial index (ABI) at rest $\leq$ 0.90 in 1 or 2 legs, reduction of ABI after treadmill test, and exercise tolerance limited by claudication. Patients were excluded under the following conditions: presence of chronic lung disease, poorly controlled BP, electrocardiogram response suggestive of myocardial ischemia during the treadmill test, and history of revascularization in the previous year.

This study was approved by the Joint Committee on Ethics of Human Research of the Clinics Hospital of the University of São Paulo (process 0813/08). Each patient was informed of the risks and benefits involved in the study, and signed a written informed consent before participation.

Peripheral arterial disease diagnose

For the ABI assessment, systolic brachial BP was obtained by auscultation, and systolic ankle BP was measured by Doppler ultrasound (Martec DV 6000, Ribeirão Preto, Brazil). A mercury column sphygmomanometer was used for both measurements. These procedures have been applied previously,[16] and similar results were obtained when brachial BP was assessed with auscultatory and Doppler techniques.[22] Ankle BP was measured in the dorsalis pedis artery and in the posterior tibial artery, and the lowest value obtained in each leg was recorded. ABI was calculated for the left and right legs as the quotient between systolic ankle and brachial BPs, and the lowest value was used for sample description.

All patients performed a progressive graded cardiopulmonary treadmill test until maximal claudication pain, as previously described for these patients.[23] All patients were already familiarized with the test protocol before the experiments. During the test, electrocardiogram was continuously monitored and registered at the end of each stage. Oxygen uptake (VO_2) was continuously measured by a metabolic cart (Medical Graphics Corp CPX/D, St. Paul, Minnesota, Minn), and peak VO_2 was defined as the highest VO_2 achieved during the test. Initial claudication distance and total walking distance were defined, respectively, as the distances walked until the patient first reported pain in the leg, and were unable to go on due to leg pain.

Familiarization sessions

Before the beginning of the experimental protocol, subjects underwent four familiarization sessions. In the first two sessions, subjects learned the correct execution of the exercises using the lowest load allowed in each exercise. In the third session, a trial and error procedure was employed for identifying the workload necessary for eliciting a perceived exertion corresponding to 11 to 13 on the 15-grade Borg's scale in each exercise.[24] The adequacy of these workloads to trigger the desired perceived exertion rate was checked in the fourth familiarization session, and necessary adjustments were performed when necessary. The workloads corresponding to 11 and 13 of the perceived exertion were recorded and used in the experimental session.

Experimental protocol

The experimental protocol was conducted with a crossover design, in which all the subjects were submitted to both experimental sessions: control (C) and resistance exercise (R). The order of the experimental session was randomly determined. Each session started between 7 and 9a.m., and an interval of at least 3 days was maintained between them.

Patients were instructed to have a light meal 2 h before the experiments, avoid physical exercise and alcohol ingestion for at least 48 hours prior to the experiments, and also avoid smoking and caffeine 12 hours before the sessions. Moreover, patients were instructed to keep their medication routine on experimental days.

In each experimental session, patients rested in the sitting position for 10 min (pre-intervention). Then, they went to the exercise room where they remained seated in the C session, and exercised in the R session. Patients were blinded to which session they were going to perform in each experimental day until the exercise or the rest began. After the interventions, subjects returned to the laboratory where they remained resting in the sitting position for 60 min (post-intervention period).

In the R session, patients performed 6 exercises: bench press, seated row, crunches, seated leg press, knee extension, and seated calf. In each exercise, they performed 3 consecutive sets of, respectively, 12, 10 and 8 repetitions, with a workload greater enough to elicit a perceived exertion corresponding to 11 to 13 on the 15-grade Borg scale.[24] An interval of 2 min was maintained between the sets and the exercises. To guarantee a good execution of the exercises, all subjects previously underwent 4 familiarization sessions, using the same exercise protocol. As the R session lasted approximately 30 min, subjects remained seated on the exercise machines for this period in the C session.

During the experimental sessions, auscultatory brachial BP was measured by the same observer. For these measurements, a sphygmomanometer cuff, with an appropriate size, was placed on the non-dominant arm of the subject. Cuff was inflated to a pressure exceeding the expected systolic BP, and it was deflated slowly in a rate of 2 mmHg per second. Phase I and V of Korotkoff's sounds were employed for determining systolic and diastolic blood pressures, respectively.[25] Mean BP was calculated as the diastolic BP plus 1/3 of pulse pressure. BP was measured 3 times in the pre-intervention period, and 3 times at 15, 30, 45 and 60 min of the post-intervention period. The intraclass coefficient correlations of these measurements were 0.99 and 0.92 for systolic and diastolic BP, respectively. For each triplicate measurement, the median value was used for the analysis.

Statistical analyses

Considering a power of 80%, and an alpha error of 0.05, and assuming a standard deviation of 3 mmHg, the sample size necessary to detect a mean reduction of 4 mmHg in BP was calculated to be 8 subjects.[17] The Gaussian distribution of the data was verified by the Shapiro–Wilks test, and the homogeneity of variance by Levene test. Changes in hemodynamic parameters were compared by a two-way analysis of variance for repeated measures, establishing sessions (C and R) and stages (pre and post-intervention 15, 30, 45 and 60 min) as the main factors. Post-hoc comparisons were performed with Newman–Keuls test. $P<0.05$ was

Table 1 - Clinical characteristics of the patients included in the study.

Characteristics	N = 8
Age, yrs	64.4 ± 6.6
Weight, kg	72.3 ± 12.6
Body Mass Index, kg/m^2	26.2 ± 2.9
Ankle Brachial Index	0.66 ± 0.13
Initial claudication distance, m	518 ± 68
Total walking distance, m	737 ± 73
Maximal heart rate, bpm	115 ± 10
Peak VO_2, ml.kg^{-1}.min^{-1}	15.4 ± 1.3
Risk factors	
Physical Inactivity, %	25.0
Current smoker, %	37.5
Hypertension, %	75.0
Diabetes, %	37.5
Coronary artery disease, %	25.0
Medication	
β-blockers, %	12.5
Calcium channel blocker, %	12.5
ACE inhibitor, %	67.5
Angiotensin II receptor antagonist, %	12.5
Diuretic, %	25.0
Anti-platelet agents, %	87.5

Continuous variables are presented as mean ± SE

accepted as statistically significant. Data are presented as mean ± standard error.

RESULTS

Patients' characteristics are shown in Table 1. Patients were mostly elderly, non-obese, and were currently taking antihypertensive drugs. The mean resting ABI of the sample was 0.66 ± 0.13.

Four patients initiated the protocol with the C session and 4 with the R session. In the R session, the loads used in each exercise were: 17 ± 4 kg for bench press, 37 ± 9 kg for seated row, 35 ± 6 kg for crunches, 61 ± 19 for seated leg press, 12 ± 1 kg for knee extension and 56 ± 17 for seated calf raise.

BP measured before the interventions in both experimental sessions are shown in Table 2. Pre-intervention values for systolic, diastolic, and mean BPs were similar between the experimental sessions ($P>0.05$).

Systolic, diastolic, and mean BP changes observed in both experimental sessions are shown, respectively, in Figures 1, 2 and 3. In comparison with the pre-intervention values, systolic BP did not change after the C session, and decreased after the R session during all the recovery stages (greatest fall = -14 ± 5 mmHg, $P<0.05$; $\eta^2 = 0.99$ [CI95%: 0.06 – 2.17]). The falls in systolic BP after the R session differed significantly from the response observed in the C session at all the recovery stages ($P<0.05$).

Table 2 - Pre-intervention blood pressure (BP) measured in the control and resistance exercise sessions.

	Control Session	Resistance Exercise	P
Resting systolic BP, mmHg	117.8 ± 18.5	114.8 ± 17.3	0.52
Resting diastolic BP, mmHg	69.3 ± 5.9	70.3 ± 8.0	0.67
Resting mean BP, mmHg	85.4 ± 7.8	84.9 ± 7.5	0.87

Values are mean ± SE

In comparison with the pre-intervention values, diastolic BP did not change after the C session, and decreased after the R session during all the recovery stages (greatest fall = -6 ± 5 mmHg, $P< 0.05$; $\eta^2 = 1.14$ [CI95%: 0.09 – 2.21]). The falls in diastolic BP after the R session were significantly different from the response observed in the C session at 30 and 45 min of recovery ($P<0.05$).

In comparison with the pre-intervention values, mean BP did not change after the C session, and decreased after the R session during all the recovery stages (greatest fall = -9 ± 4 mmHg, $P < 0.05$; $\eta^2 = 1.29$ [CI95%: 0.18 – 2.32]). The falls in mean BP after the R session were significantly different from the response observed after the C session at all the recovery stages ($P<0.05$).

DISCUSSION

The novel finding of the present study was that one resistance exercise session decreased BP levels in patients with IC, and this effect lasted for at least one hour after the end of the exercise. This result provides initial evidence that acute resistance exercise might be useful to promote a decrease in BP in IC patients.

In order to have clinical relevance, post-exercise hypotension may require significant magnitude and should last a long time after the exercise.[15] The magnitude of systolic/diastolic BP reductions observed in this study (-14/5 mmHg) was significant. It is similar to the ones previously reported in hypertensive women (-12/6 mmHg),[26] and also after aerobic exercise (-15/4 mmHg).[11] In fact, the magnitude of post-resistance exercise hypotension varies significantly between studies, ranging from 3 mmHg[27] to 23 mmHg.[28] Moreover, some authors have reported a maintenance[29] or even an increase[29-32] in BP after a single bout of resistance exercise. Differences in the results among studies may be partly explained by differences in the exercise protocols employed in each investigation. Moderate-intensity resistance exercise protocols[18] including more than 12 sets of exercise for the major muscles[17,18,26,33] seem to produce greater post-exercise hypotensive effects. The present exercise protocol included a total of 18 sets, and its design was similar to the one employed in a previous study that observed a significantly post-exercise hypotension.[18] Moreover, it was also similar to the resistance exercise protocol employed in a resistance training that resulted in a significant increase in walking capacity in IC patients.[16] The rate of 11 to 13 on the 15-grade Borg's perceived exertion scale corresponded to a moderate intensity,[34,35] and has also been employed for training IC patients.[16] Therefore, the occurrence of post-resistance exercise hypotension in the present study might have been caused by the exercise protocol applied, since it presented the characteristics required for this purpose.

In regard to the duration of post-exercise hypotensive effect, BP was measured for 60 min after the intervention. Although post-exercise hypotension is clinically relevant when it lasts for a long period,[15] it is interesting to note that at 60 min of recovery BP levels remained lower than in the pre-intervention period, which suggests that hypotensive effects might last longer than one hour. In a previous study, we observed that post-resistance exercise hypotension lasted 10 hours in medicated hypertensive women.[26] Nevertheless, the duration of post-resistance exercise hypotension remains unknown in IC patients, and should

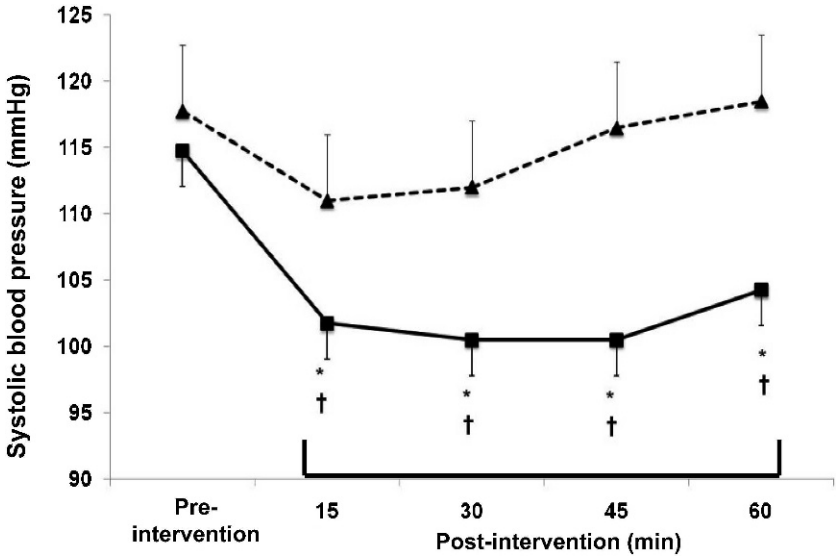

Figure 1 - Systolic blood pressure measured pre-intervention, and at 15, 30, 45 and 60 minutes after the interventions in the control (dashed line with triangles) and the resistance exercise (solid line with squares) sessions. * Significantly different from pre-intervention ($P<0.05$). † Significantly different from the control session ($P<0.05$).

be investigated in future studies employing the ambulatory blood pressure monitoring in order to assess the possible clinical relevance of this phenomenon.

Mechanisms responsible for the BP fall after exercise were out of the scope of the present investigation. However, in healthy subjects, post-resistance exercise hypotension has been attributed to a decrease in the cardiac output which was not compensated by an increase in peripheral vascular resistance. The decrease in cardiac output was determined by a decrease in stroke volume produced by a reduction in venous return.[18] Moreover, the BP fall was only possible because exercise had an effect on peripheral vessels, blunting the vasoconstrictive reflex triggered by the cardiopulmonary receptors deactivation produced by venous return reduction.[36] We did not expect post-exercise hypotension because of the presence of endothelial dysfunction in these patients that may blunt the effects of exercise on peripheral vessels. However, contrary to our hypothesis, post-exercise hypotension was evident and significant, which might be explained by the fact that many of the patients were taking medications, such as ACE inhibitors, calcium channel blockers, and angiotensin II receptors antagonist, that reduce vasoconstriction and may have potentiated the effects of exercise on peripheral vessels. This hypothesis should be addressed in future studies.

The experimental design employed in this investigation is a strength point of the study. The presence of a control session allowed determining the net effect of exercise, eliminating any possible influence of time on the variables. In addition, since all subjects underwent both experimental

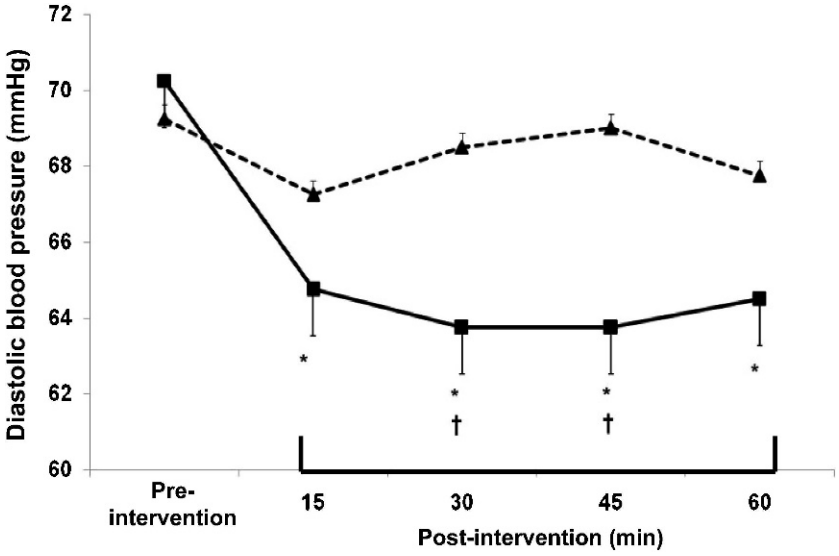

Figure 2 - Diastolic blood pressure measured pre-intervention, and at 15, 30, 45 and 60 minutes after the interventions in the control (dashed line with triangles) and the resistance exercise (solid line with squares) sessions. * Significantly different from pre-intervention ($P<0.05$). † Significantly different from the control session ($P<0.05$).

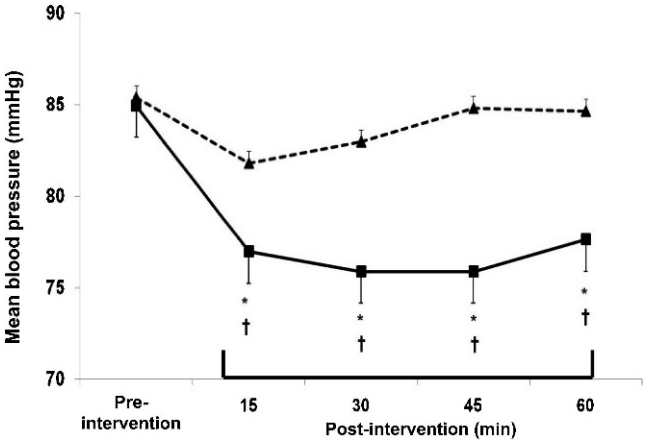

Figure 3 - Mean blood pressure measured pre-intervention, and at 15, 30, 45 and 60 minutes after the interventions in the control (dashed line with triangles) and the resistance exercise (solid line with squares) sessions. * Significantly different from pre-intervention ($P<0.05$). † Significantly different from the control session ($P<0.05$).

sessions, the inter-subject differences that might influence the results were minimized. On the other hand, this study has some limitations that should be considered in the interpretation of the results. Patients' characteristics, such as body mass index and physical fitness, as well as medication use might influence BP responses after exercise. These factors were not standardized in the present study, which limits the interpretation of the results in regard to their possible influence. However, in clinical practice, IC patients usually have different characteristics and are receiving different medications,[1] and thus, not controlling for these factors increases the applicability of the present results. Exercise intensity was established based on Borg's scale since this method has been validated for establishing resistance exercise intensity in several populations,[34,35,37,38] and may also be useful in IC patients. However, a strength test that might directly assess the exercise intensity was not performed in the present study.

CONCLUSIONS

A single bout of resistance exercise reduces systolic, diastolic, and mean BPs for at least 60 min after exercise in patients with peripheral arterial disease and IC. These results suggest that resistance exercise acutely reduces cardiovascular load in these patients, which might have clinical relevance.

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