Post-Walking Exercise Hypotension in Patients with Intermittent Claudication

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1Hospital Israelita Albert Einstein, São Paulo, BRAZIL; 2Exercise Hemodynamic Laboratory, School of Physical Education and Sport, University of São Paulo, São Paulo, BRAZIL; 3School of Physical Education, Pernambuco University, Pernambuco, BRAZIL; 4Hospital das Clínicas, University of São Paulo, São Paulo, BRAZIL; and 5School of Health Sciences, University of East Anglia, Norwich, UNITED KINGDOM

ABSTRACT

CUCATO, G. G., M. DA ROCHA CHEHUEN, R. M. RITTI-DIAS, C. R. F. CARVALHO, N. WOLOSKER, J. M. SAXTON, and C. L. M. FORJAZ. Post-Walking Exercise Hypotension in Patients with Intermittent Claudication. Med. Sci. Sports Exerc., Vol. 47, No. 3, pp. 460–467, 2015. Purpose: This study aimed to investigate the acute effect of intermittent walking exercise (WE) on blood pressure (BP) responses in patients with intermittent claudication (IC). Secondly, this study aimed to gain improved insight into the physiological mechanisms controlling BP regulation after intermittent WE in this patient group. Methods: Twenty patients with IC participated in two experimental sessions in a random order, as follows: WE (15 × 2-min bouts of WE interpolated with 2-min rest intervals) and control (standing rest on a treadmill for 60 min). BP, cardiac output (CO: CO2 rebreathing), and cardiovascular autonomic modulation (spectral analysis of HR variability) were assessed before and after both experimental sessions during supine rest, and stroke volume (SV) and systemic vascular resistance (SVR) were calculated. Data were analyzed using two-way ANOVA. Results: WE decreased systolic, diastolic, and mean BP, with net effects of −13 ± 2, −5 ± 2, and −7 ± 2 mm Hg versus control, respectively (all P < 0.05). WE also decreased SV (−5.62 ± 1.97 mL, P < 0.05) and CO (−0.05 ± 0.13 L·min⁻¹, P < 0.05) versus preintervention and prevented the observed increase in SVR in the control condition (+4.2 ± 1.4 U, P < 0.05). HR showed a decrease (P < 0.05), consistent with evidence of increased vagal modulation, in the control condition. BP measurements over the subsequent 24 h were similar between experimental conditions. Conclusions: In patients with IC, WE induced a postexercise hypotension response that had a significant magnitude versus control but was not maintained over the next 24 h of daily activities. The acute postexercise hypotension response was mediated by a decrease in CO and SV, which was not compensated by an augmentation of SVR, as observed in the control arm of the study. Key Words: PERIPHERAL ARTERIAL DISEASE, INTERMITTENT WALKING, BLOOD PRESSURE, AEROBIC EXERCISE

Peripheral arterial disease (PAD) is commonly manifest as intermittent claudication (IC), a cramp-like pain in the lower-limbs during walking (18). Many patients have classic cardiovascular risk factors, including smoking, dyslipidemia, insulin resistance, and/or type 2 diabetes mellitus (19). Hypertension is also highly prevalent among the IC population, and patients are at significantly increased risk of cardiovascular mortality (4,35). Because of the diffuse nature of atherosclerotic lesions, patients are frequently managed conservatively for cardiovascular risk factors and are encouraged to stop smoking and increase their amount of daily walking exercise (WE) (26).

Evidence-based WE prescription typically involves repeated bouts of WE to the point of claudication pain, with short interpolated recovery intervals (28). Studies show that this type of WE prescription increases pain-free walking distance over time via improvements in skeletal muscle perfusion and/or oxygenation (3,36). However, its effect on systemic cardiovascular function and blood pressure (BP) responses has received little attention despite a body of literature reporting postexercise hypotension (PEH) effects in other populations with hypertension (6,10,14,22,23,29,30,32–34). Improved knowledge of the BP response to intermittent WE is needed to establish its use as a nonpharmacological treatment strategy for controlling hypertension in patients with IC.

Therefore, the main aim of this study was to investigate the acute BP response to intermittent WE, which is representative of evidence-based exercise prescription in this patient group. Secondly, this study aimed to gain improved insight into the physiological mechanisms controlling BP regulation after intermittent WE in these patients.
METHODS

Study population and sample size. Male patients with PAD and symptoms of IC were recruited from the Vascular Unit of the Hospital das Clínicas of the University of São Paulo, Brazil. Inclusion criteria were as follows: 1) ankle–brachial index (ABI), ≤0.90; 2) graded treadmill test limited by claudication; 3) nonobese (body mass index, <30 kg·m⁻²); 4) not currently engaged in an exercise program; 5) not using antihypertensive medications that affect the HR response to exercise (β-blockers and nondihydropyridine calcium channel blockers); 6) resting systolic BP <160 mm Hg and diastolic BP <105 mm Hg; and 7) no symptoms of myocardial ischemia or arrhythmias during a treadmill test. Considering a power of 90% and an alpha error of 0.05 and assuming an SD of 3 mm Hg, the sample size necessary to detect a mean reduction of 4 mm Hg in BP after exercise was calculated to be 10 participants. Allowing for approximately 50% patient dropout, 20 patients were recruited for the study.

This study was approved by the Joint Committee on Ethics of Human Research of the University (process 2008/55) and was registered in the Brazilian Registry of Clinical Trials (RBR–7M3D8W). Each patient was informed of the risks and benefits involved in the study and signed a written informed consent form before participation.

Participant screening. PAD was diagnosed by clinical history and ABI measurement. Arm systolic BP was measured by auscultation. Ankle systolic BP was measured by Doppler ultrasound (DV 6000; Martec, Ribeirão Preto, Brazil) in the dorsalis pedis artery and in the posterior tibial artery, and the highest value obtained in each leg was recorded. A mercury column sphygmomanometer was used for both measurements (Unitec, São Paulo, Brazil). ABI was calculated for the left and right legs as the quotient of systolic ankle and brachial BP, and the lowest value was recorded. Body mass and height were measured (Welmy, 110, São Paulo, Brazil), and body mass index was calculated. Resting BP was measured with the patient seated at rest for at least 5 min. Measurements were taken from both arms using the auscultatory method and a mercury sphygmomanometer (Unitec, São Paulo, Brazil). Phases I and V of the Korotkoff sounds were used to identify systolic and diastolic BP, respectively. Multiple measurements were taken for each arm until three consecutive measurements with differences lower than 5 mm Hg were obtained. This procedure was repeated in two visits to the laboratory on different days. The mean value of six valid systolic and diastolic BP measurements obtained from the arm with the highest BP was used as the resting value.

Determination of HR at claudication pain onset. An incremental treadmill test (Imbrasport ATL, Porto Alegre, Brazil) to maximum exercise tolerance was used to determine HR at claudication pain onset (12). Speed was maintained at 3.2 km·h⁻¹, and grade increased by 2% every 2 min. A 12-lead ECG was continuously monitored (Cardio Perfect MD; Welch Allyn, Inc., Skaneateles Falls, NY), and HR was recorded at the end of each stage. Oxygen uptake (VO₂) was continuously measured by a metabolic cart (CPX/D; Medical Graphics Corporation, St. Paul, MN), and averaged 30-s data were used for the analysis. Initial claudication distance (distance walked until claudication pain onset) and total walking distance (unable to continue walking because of leg pain) were recorded. HR coinciding with claudication pain onset was used to set the intensity of intermittent WE in the experimental condition of the study (see later section). This exercise intensity was shown to be above anaerobic threshold and to evoke tolerable levels of claudication pain during repeated 2-min bouts of WE (8).

Experimental protocol. All patients underwent two experimental sessions (WE and control) conducted in a random order and separated by at least 48 h. Sessions were commenced between 7:00 and 8:00 a.m., and patients were instructed to have a light meal at least 2 h before arriving at the laboratory and not to ingest coffee, tea, caffeinated soft drinks, or other stimulants thereafter. In addition, they were instructed to refrain from vigorous physical activity for the previous 48 h and from alcohol ingestion for the previous 24 h. Smokers were instructed not to smoke before testing sessions, and all patients continued to take their regular medication on experimental days.

During both experimental sessions, patients initially rested in the supine position for 90 min, which was called the preintervention period. ECG and breathing rate were collected for spectral analysis between 35 and 45 min; auscultatory BP, cardiac output (CO), and HR were measured in triplicate between 45 and 60 min, and the mean value was calculated for analysis. Lower and upper limb vascular resistance was determined during the last 30 min of supine rest.

In the WE condition, patients then performed 15 × 2-min bouts of WE on a treadmill, interpolated by 2-min rest intervals (total duration of 60 min). During each 2-min bout of WE, exercise intensity was adjusted to maintain HR at the HR of claudication pain onset ± 4 beats per minute. Treadmill speed was set at 3.2 km·h⁻¹, and grade was adjusted to achieve the target HR, which was continuously monitored (A3; Polar, Helsinki, Finland). Pain levels were assessed at the end of the fifth and 12th bouts. In the control condition, patients remained in a standing position on the treadmill for 60 min.

Thereafter, patients returned to the resting supine position for 90 min (postintervention period). ECG and breathing rate were collected for spectral analysis between 35 and 45 min; auscultatory BP, CO, and HR were measured in triplicate between 45 and 60 min, and the mean value was calculated for analysis. Lower and upper limb vascular resistances were determined during the last 30 min of supine rest.

Finally, an ambulatory BP monitor (90207; SpaceLabs Medical, Inc., Snoqualmie, WA) was placed on the nondominant arm and programmed to perform measurements every 15 min for 24 h. During this period, patients were asked to maintain normal daily activities and to refrain from ingesting alcoholic beverages and sleep during daytime hours.

Measurements. Auscultatory BP was measured in the dominant arm using a mercury sphygmomanometer (Unitec, São Paulo, Brazil) immediately before CO determination. A
single researcher was responsible for taking all BP measurements. Mean BP was calculated as the sum of diastolic BP and one-third of pulse pressure. ECG was continuously monitored (Cardio Perfect MD; Welch Allyn, Inc., Skaneateles Falls, NY), and HR was recorded immediately after BP measurement.

CO was estimated by the indirect Fick method (21) using the CO2 rebreathing technique and a metabolic cart (CPX/D; Medical Graphics Corporation, St. Paul, MN). Briefly, patients breathed spontaneously until a steady CO2 production was achieved. This procedure was followed by rebreathing of a mixed gas containing a high CO2 (8%–10%) and O2 (35%) concentration until equilibrium was achieved (maximal of 15 s). At this point, CO was calculated using the Fick formula. Systemic vascular resistance (SVR) was expressed as the quotient of mean BP and CO, and stroke volume (SV), as the quotient of CO and HR.

Autonomic cardiovascular modulation was assessed by the spectral analysis of HR variability, according to Task Force recommendations (37). ECG (M10; TEB, São Paulo, Brazil) and breathing signal (Pneumotrace; UFI, Morro Bay, CA) were recorded for 10 min with a sample frequency of 500 Hz per channel. RR interval and respiratory time series were generated by the PRE software (di Calcolo Segnali Variabilità Cardiovascolari-6.20.95, Milano, Italy). Autoregressive spectral analyses of these signals were performed using the LA software (Programma di Analisi Lineare—14/12/1999, Milano, Italy). Briefly, on stationary segments of at least 2 min, autoregressive parameters were estimated by the Levinson–Durbin recursion, and the order of the model was chosen according to the Akaike criterion. The autoregressive components were assigned on the basis of their central frequency as low frequency (LF, 0.04–0.15 Hz) and high frequency (HF, 0.15–0.4 Hz).

Upper and lower limb vascular resistances were determined in the dominant forearm and leg with the lowest ABI. Measurements were performed while blood flow to the hand and the foot were obstructed by a cuff inflated to 200 mm Hg and placed, respectively, around the wrist and the ankle. For the vascular resistance measurements, cuffs placed at the forearm and the leg were rapidly inflated for 10 s at 40–60 mm Hg, followed by 10 s of deflation. Increases in limb volumes were detected by mercury strain gauges (AI-6; Hokanson, Bellevue, WA) positioned at the largest circumference of the forearm and the leg. The signals were recorded using specific software (NIVP3; Hokanson, Bellevue, WA). Measurements were taken during 4 min (12 cycles), with the first two and the last measures being excluded from the analysis. Hence, the mean of nine cycles was used to determine blood flow, with vascular resistance calculated as the quotient of blood flow and mean BP and expressed in arbitrary units (U).

### Table 1. Physical characteristics, risk factors, medications, and cardiorespiratory and functional variables measurements in cardiopulmonary test.

<table>
<thead>
<tr>
<th>Variables</th>
<th>n = 20</th>
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<td>Age (yr)</td>
<td>61.2 ± 1.7</td>
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<td>Weight (kg)</td>
<td>71.5 ± 2.2</td>
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<td>Body mass index (kg m^2)</td>
<td>25.3 ± 0.6</td>
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<td>Ex-smoker (%)</td>
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<td>Current smokers (%)</td>
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<td>Hypertension (%)</td>
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<td>Diabetes (%)</td>
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<td>Dyslipidemia (%)</td>
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<td>Antihypertensive (%)</td>
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<td>Anticoagulants (%)</td>
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<td>Statins (%)</td>
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<td>Hypoglycemic (%)</td>
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<td>VO2max (mL kg^-1 min^-1)</td>
<td>18.9 ± 0.8</td>
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<td>HRmax (beats per minute)</td>
<td>126 ± 4</td>
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<td>COD (m)</td>
<td>297 ± 38</td>
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<tr>
<td>TWD (m)</td>
<td>743 ± 19</td>
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Values are mean ± SE.
COD, claudication onset distance; VO2, oxygen consumption; TWD, total walking distance.

**FIGURE 1**—Response (Δ) of systolic, diastolic, and mean BP measured before (PRE) and after (POST) the control (dashed line) and WE (solid line) sessions (n = 20). *Indicates difference from preintervention (P < 0.05). †Indicates difference between the WE and control condition (P < 0.05). Values are mean ± SE. DBP, diastolic BP; MBP, mean BP; SBP, systolic BP.
Statistical analysis. The normality of the data distributions and homogeneity of variances were evaluated using the Shapiro–Wilk and Levene tests, respectively, with skewed distributions being normalized using logarithmic transformations before analysis. Data were analyzed using two-way ANOVA for repeated measures, establishing the following as the main factors: condition (WE and control) and time (before and after intervention). The condition–time interaction was used to determine the net effect of WE versus control on study outcomes, presented as follows: (postexercise – preexercise) – (postcontrol – precontrol). Hourly ambulatory BP obtained up to 24 h after the WE and control conditions were also compared with a two-way ANOVA for repeated measures, establishing condition (WE and control) and time (1, 2, and 3 h, etc.) as the main factors. Statistical significance was set at \( P < 0.05 \), and data are presented as mean ± SE.

RESULTS

Patient characteristics and intensity of intermittent WE. Eighty patients initially volunteered for the study, with 26 of them fulfilling the inclusion criteria and signing an informed consent to participate. However, an additional six patients were excluded during the preliminary evaluation period (screening examinations and maximal test). Thus, 20 patients initiated the experimental protocol and all of them completed both experimental sessions. Their characteristics are shown in Table 1. Hypertension was present in 75% of the patients, and most of them were receiving anticoagulant medication, antihypertensive agents, and statins. Patients were able to perform all 2-min bouts of intermittent WE with tolerable levels of claudication pain, which subsided during the interpolated rest intervals. During exercise, HR measured at the fifth and 12th bouts of WE were 99 ± 16 and 101 ± 16 beats per minute, respectively, equating to 100% ± 3% and 102% ± 5% of the HR at claudication pain onset, respectively.

BP responses. In comparison with preintervention, systolic BP significantly decreased after WE (131 ± 3 vs 127 ± 4 mm Hg, \( P < 0.05 \)) whereas diastolic and mean BP were unchanged (77 ± 2 vs 78 ± 2 and 95 ± 2 vs 94 ± 2 mm Hg, respectively, \( P > 0.05 \)). Conversely, systolic, diastolic, and mean BP increased from preintervention values in the control condition (130 ± 3 vs 138 ± 4, 76 ± 1 vs 82 ± 2, and 94 ± 2 vs 100 ± 2 mm Hg, respectively; all \( P < 0.05 \)). Significant interactions demonstrated net effects of WE on BP responses versus control (\( j = 13 ± 2, −5 ± 2, \) and \( −7 ± 2 \) mm Hg, for systolic, diastolic, and mean BP, respectively; all \( P < 0.001 \)) (Fig. 1). In contrast, hourly systolic, diastolic, and mean BP measured in the 24 h after the laboratory sessions were similar between the WE and control conditions of the study (\( P > 0.05 \)) (Fig. 2).

Hemodynamic responses. CO (2.93 ± 0.13 vs 2.78 ± 0.12 L·min\(^{-1}\); \( P < 0.05 \)) and SV (44.6 ± 2.3 vs 39.2 ± 2.1 mL; \( P < 0.05 \)) decreased from preintervention values after WE, whereas SVR, leg and forearm vascular resistance, and HR were unchanged (\( P > 0.05 \)). CO was also reduced versus
preintervention in the control condition (3.03 ± 0.10 vs 2.93 ± 0.13 L-min⁻¹; P < 0.05), and this was accompanied by a decrease in HR (66 ± 2 vs 63 ± 2 beats per minute; P < 0.05), whereas SV was unchanged. Increases in SVR (32.4 ± 1.1 vs 36.8 ± 1.8 U; P < 0.05) and leg and forearm vascular resistance (50.9 ± 3.9 vs 63.5 ± 5.5 and 45.7 ± 4.2 vs 66.3 ± 5.5 U, respectively, P < 0.05) from preintervention values were also observed in the control condition. Significant interactions demonstrated net effects of WE versus control for SV (Δ5.62 ± 1.97 mL), HR (+5 ± 1 beats per minute), SVR (−3.1 ± 1.5 U), leg vascular resistance (−7.0 ± 2.5 U), and forearm vascular resistance (−6.1 ± 3.7 U) (all P < 0.05) (Fig. 3).

**Autonomic responses.** Autonomic responses are shown in Table 2. Because of the presence of arrhythmias and RR artifacts, autonomic data were not available for six patients. RR

![Graphs showing response (Δ) of CO, HR, SV, SVR, forearm vascular resistance, and leg vascular resistance measured before (PRE) and after (POST) the control (dashed line) and WE (solid line) sessions (n = 20). *Indicates difference from preintervention (P < 0.05). †Indicates difference between the WE and control condition (P < 0.05). Values are mean ± SE. VRf, forearm vascular resistance; VRl, leg vascular resistance.

![Graphs showing autonomic responses.](http://www.acsm-msse.org)

**TABLE 2. Response (Δ) of autonomic variables assessed by HR variability.**

<table>
<thead>
<tr>
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<th>Control Session</th>
<th>Exercise Session</th>
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<tr>
<td>RR (ms²) (n = 14)</td>
<td>+96 ± 25*</td>
<td>−16 ± 22**</td>
</tr>
<tr>
<td>TVLF (ms²) (n = 14)</td>
<td>−918 ± 590*</td>
<td>+103 ± 380**</td>
</tr>
<tr>
<td>LF (ms²) (n = 14)</td>
<td>+388 ± 177*</td>
<td>+61 ± 70**</td>
</tr>
<tr>
<td>HF (ms²) (n = 14)</td>
<td>+110 ± 42*</td>
<td>−13 ± 25**</td>
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</table>

Values are mean ± SE.

*Indicates difference from preintervention (P < 0.05).

** Indicates difference between the WE and control condition (P < 0.05).

LF, LF band; HF, HF band; RR, RR interval; TV, total variance.
interval, total variance, and absolute values of HF and LF increased from preintervention values in the control condition (all \( P < 0.001 \)) but were unchanged after WE (\( P > 0.05 \)). Significant interactions demonstrated net effects of WE versus control for RR interval (\(-15.6 \pm 22.0\) ms; \( P = 0.001 \)), total variance (\(-815 \pm 360\) ms\(^2\); \( P = 0.04 \)), HF (\(-123 \pm 45\) ms\(^2\); \( P = 0.04 \)), and LF (+61 \pm 76 ms\(^2\); \( P = 0.03 \)) (all \( P < 0.05 \)).

**DISCUSSION**

This is the first study to report the acute effect of intermittent WE (representative of evidence-based exercise prescription) on BP responses in patients with IC. The results show that intermittent WE resulted in a PEH response of significant magnitude versus the control condition, with net reductions in systolic, diastolic, and mean BP of \(-13 \pm 2, -5 \pm 2, \) and \(-7 \pm 2\) mm Hg, respectively. In relation to preintervention measures, the PEH response was less pronounced and was limited to changes in systolic BP (\(-4 \pm 2\) mm Hg). The modest reduction in systolic BP from the preintervention value after WE may be attributable to the time of the day when the experiments were conducted (7:00–10:00 a.m.). The circadian variation in BP is well defined, increasing progressively during the morning period, with peak values being observed at approximately 10:00 a.m. before showing a decrease (17). Hence, circadian variation probably explains the increase in BP from preintervention values in the control condition, as although standing produces an important orthostatic stress, hemodynamic measurements were taken at least 45 min after the patients had reverted back to the supine position (same for the WE condition). This is consistent with previous studies undertaken at a similar time of the day in this patient group (9,31). It is also interesting to note that a blunted or absent PEH response in comparison with preexercise values has been reported previously in healthy individuals after morning exercise (20).

Our results show that the increase in systolic and diastolic BP from preintervention in the control condition was attributable to an increase in SVR, and this was corroborated by simultaneous increases in lower and upper limb vascular resistances (13). Although diurnal increases in plasma catecholamines and angiotensin at this time of day (27) may explain the observed increase in SVR, this needs to be verified in future research. We also observed a decrease in CO from preintervention in the control condition, but this was insufficient to offset the effects of an augmented SVR on BP. Our data suggest that the latter resulted from a decrease in HR, which is consistent with evidence of an increase in cardiac vagal modulation (increases in total variance, LF, and HF). In contrast, the decrease in CO observed after intermittent WE was mainly attributable to a decrease in SV, which was not offset by increases in HR or SVR. The reduced SV after WE is consistent with previous studies that have reported PEH after aerobic exercise in different populations (6,11,38). The reduction in SV after bouts of aerobic exercise has been attributed to a decrease in venous return resulting from reduced plasma volume (sweating, fluid shifts to the interstitial spaces, etc.) and/or increased venous compliance (6,15). There is also evidence that aerobic exercise decreases peripheral sympathetic nerve activity (5,16) and depresses the vasoconstrictor response to sympathetic stimuli (16). Taking into account the increase in BP responses in the control condition of the study, our results therefore suggest that morning intermittent WE prevents the diurnal increase in BP in patients with IC.

This could have important clinical implications for cardiovascular risk, especially in light of the increased rate of cardiovascular events at this time of the day, which may be linked to diurnal BP changes (1,24). The hypotensive effect of intermittent WE was not maintained over the following 24 h, however, when patients returned to their normal daily activities. This is in contrast to previous studies that have reported PEH responses in normotensive and hypotensive individuals lasting 4–16 h (7). The lack of maintenance of PEH through out the following 24 h might be explained by the pain levels that patients experience during normal daily activities, including ambulation. It is known that pain activates pressor reflexes, which augment sympathetic activity (25), progressively increasing BP during WE in these patients (2). This “pain-pressor” reflex response could have mitigated the PEH effect of intermittent WE observed in the laboratory during (postintervention) pain-free supine rest, thus diminishing its clinical relevance.

The magnitude of the acute PEH response versus preintervention supine resting values could also have important clinical implications for patients with IC. A recent study (23) showed that the magnitude of PEH after a single session of aerobic exercise has direct relation with the chronic reduction of BP after a period of exercise training. If a causative relation for patients with IC exists, which is yet to be established, the magnitude of the PEH response could be used as a clinical strategy to identify patients and/or exercise times that are most likely to result in cardiovascular benefits. In the present study, a modest morning PEH response from supine resting values was observed in our patient group, perhaps attributable to the normal diurnal increase in BP (previously discussed), and the response was limited to a decrease in systolic BP. In this respect, undertaking intermittent WE at a time of day when BP is not increasing because of circadian variation might augment the cardiovascular health benefits to be gained, although further research is needed to confirm this.

This study had some important limitations. Firstly, all patients were male and had stage II of PAD. Thus, the results cannot be extrapolated to females or patients at other stages of the disease (asymptomatic, stages III and IV). Secondly, patients receiving \( \beta \)-blockers, nondihydropyridine calcium channel blocker, and peripheral vasodilators were not included, which precludes the applicability of the results to patients receiving these medications. Furthermore, patients were generally taking multiple medications, which does not allow evaluation of the interaction between exercise and any specific class of medication. Thirdly, although this study used an intermittent WE protocol, which is representative of evidence-based exercise
prescription in this patient group, the BP responses to alternative types of exercise may differ. Finally, because of methodological problems, the sample size for autonomic variables was slightly lower than that for hemodynamic variables, reducing the statistical power of these analyses. However, significant changes in autonomic variables were still apparent in the control condition, and these changes were concordant with the observed changes in hemodynamic variables.

In conclusion, intermittent WE induces a PEH response in patients with IC. This hypotensive effect was modest in relation to preexercise BP but greater in comparison with that in the control condition of the study. However, it is not maintained over the following 24 h. This response is mediated by a decrease in SV, which is not compensated by augmentation of SVR (as observed in the nonexercise control condition of the study). Further research is needed to understand the clinical relevance of BP changes after intermittent WE.

The authors want to thank the volunteers of the study for their collaboration and the Vascular Unit teams for their technical help with the study.

This research was supported by Fundação de Amparo à Pesquisa do Estado de São Paulo (2009-17371-6), Conselho Nacional de Desenvolvimento Científico e Tecnológico (141057/2011-4), and Coordenação de Aperfeiçoamento de Pessoal de Nível Superior (0851/12-4, PROEX).

The authors have no conflicts of interest to report.

The results of the current study do not constitute endorsement by the American College of Sports Medicine.

REFERENCES


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