Introduction

Aerobic training complemented with resistance training is recommended as part of the treatment for hypertension [2, 25]. Single sessions of aerobic as well as resistance exercise promote a significant decrease in blood pressure (BP) during the recovery period, which has been called post-exercise hypotension [16]. However, to have clinical relevance, post-exercise hypotension should have significant magnitude and last for many hours, persisting during daily activities [16].

Recently, we demonstrated that a single session of resistance exercise promoted post-resistance exercise hypotension (PREH) in non-medicated hypertensive (HT) men [28]. However, although the magnitude of BP decrease was significant, PREH lasted for only 1 h and was not sustained under ambulatory conditions. Other authors [12] have also observed short-lasting PREH in non-medicated HT, whereas in HT subjects receiving anti-hypertensive drugs, long-lasting PREH has been reported, suggesting antihypertensive drugs might potentiate PREH.

Mechanisms responsible for PREH have been poorly studied. In our previous study of non-medicated HT men [28], PREH was followed by a decrease in cardiac output (CO) in some subjects and a decrease in systemic vascular resistance (SVR) in others. Importantly, a decrease in stroke volume (SV) always accompanied PREH, which might have resulted from a decrease in pre-load that may have led to deactivation of cardiopulmonary reflex, increasing angiotensin release [10, 21]. This might increase sympathetic activity and decrease baroreflex sensitivity (BRS) [36], favoring the increase in HR and precluding the possible vasodilatory effects of exercise in peripheral vessels. The magnitude of these effects may vary among the HT subjects, explaining the differences in CO and SVR among them. Thus, physiological responses after resistance exercise are complex [19], and some interventions, such as anti-hypertensive drugs, may modify these mechanisms, favoring PREH.

Among the different classes of anti-hypertensive drugs, the angiotensin-converting enzyme (ACE) inhibitors, like captopril, are among those most widely used [20]. Captopril reduces angioten-
sin II formation [8], decreases sympathetic activity [7] and increases endothelial function [14]. By acting on these mechanisms, it is possible to hypothesize that captopril may facilitate vasodilation after exercise, exacerbating SVR reduction and promoting a greater and longer PREH. Supporting this rationale, a previous study with HT individuals receiving captopril reported ambulatory PREH for 10 h [22]. However, the mechanisms involved in this response were not assessed.

Thus, the purpose of the present study was to evaluate whether captopril potentiates PREH in HT men and the possible hemodynamic and autonomic mechanisms involved in this response.

Methods

Participants

Men with a previous diagnosis of essential hypertension at stages 1 or 2 were invited to participate in this study. All subjects who volunteered to participate were receiving regular drug treatment with one or 2 antihypertensive medications that included different classes of drugs, such as beta blockers, diuretics, ACE inhibitors, angiotensin II receptor antagonists and calcium channel inhibitors. Before study enrolment, all participants signed an informed written consent form approved by the Ethics Committee of the School of Physical Education and Sport, University of São Paulo (n° 2010/05). The study followed the ethical standards of this journal [13] and was registered at the Brazilian Registry of Clinical Trials (RBR-8MX5G6). Data from the HT subjects while receiving placebo have already been published [28] in a study comparing PREH in normotensive and non-medicated HT subjects. The novelty of the present study is the investigation and comparison while they were receiving captopril.

Inclusion criteria were: age between 30 to 60 years, non-smoker, body mass index lower than 35 kg/m², no diabetes, no previous diagnosis of cardiovascular or musculoskeletal diseases and not physically active (no practice or practicing at most twice a week). The exclusion criteria were: presence of secondary HT, target organ damage, cardiovascular disease, and systolic/diastolic BP while receiving placebo lower than 140/90 or greater than 160/105 mmHg.

For safety reasons, preliminary evaluations to assess fulfillment of the study criteria (except BP level) were conducted with the subjects receiving their regular drug treatment. These evaluations included: i) medical history and examination; ii) routine exams from the hypertension unit of the general hospital that followed the Brazilian Guidelines for Hypertension [3]; and iii) a maximal exercise test in which resting and exercise electrocardiograms were analyzed.

After preliminary exams, all subjects who fulfilled the study criteria underwent a 2-week washout period with placebo (administered 3 times a day) before starting the experimental protocol. During this period, auscultatory BP was measured 3 times in 2 visits, after 5 min of rest in the seated position. The first and the fifth Korotkoff sounds were employed, respectively, to determine systolic and diastolic BP. The mean of all measurements was calculated and subjects continued in the study only if their systolic/diastolic BP remained between 140 and 160/90 and 105 mmHg (last exclusion criterion). The lower limits identify hypertension presence [3, 20] and the upper limits represent values considered safe for beginning exercise [3].

In addition, to assure the safety of the patients, BP was measured every week throughout the study period, and subjects were excluded from the study and regular medication was resumed if BP was above 160/105 mmHg in 2 visits. Moreover, for ethical reasons, the subjects were informed that they would receive a placebo during the study. However, they were not informed at which weeks of the study the placebo would be administered. No participant reported adverse effects during the captopril regimen.

Experimental protocol

The experimental protocol is shown in Fig. 1. After the washout period, the subjects underwent, in a random order and with a double-blinded randomized crossover design, 2 experimental periods in which they received for 4 weeks placebo or captopril (50 mg) administered 3 times a day. These therapy periods were interspersed with a 2-week period of washout with placebo. To guarantee double-blindness, drugs were masked by a hospital service and were delivered to the subject by the research physicians who did not participate in the data collection. Drugs were unmasked only at the end of the study.

In the first 2 weeks with both therapies, the subjects underwent 2 resistance exercise familiarization sessions (7 resistance exercises, 2 sets of 20 repetitions, with the minimum workload allowed by the equipment). In the third week, they underwent a one-repetition maximum (1 RM) test following Kraemer and Fry’s protocol [17]. The resistance exercises employed in all the sessions were: chest press, leg press, lat pull-down, squat, arm curl, right leg curl and left leg curl.

In the third week of each therapy period, 4 ml of venous blood plasma were collected for confirming the inhibitory effects of captopril on the ACE activity.

By the fourth week of each therapy period, the subjects underwent 2 experimental sessions: Control (C) and Resistance Exercise (RE). These sessions were performed in a random order in the first therapy period and this order was repeated in the second therapy period. An interval of at least 48 h was maintained between them, and they began at 8 a.m. in a climate-controlled room (21–23 °C). Subjects were asked not to ingest coffee, tea, alcohol or other central nervous system stimulants on the day of the experiments and to avoid exercise in the previous 48 h.

In each session, subjects remained seated for 60 min before the intervention (pre-intervention period – Pre). Then, they moved to the exercise room, where they stayed for 40 min (intervention period). Subjects did not know which intervention they were going to until the beginning of the intervention. In the C session, subjects were positioned on the exercise machines but did not perform any exercise, whereas in the RE session, they performed 3 sets of repetitions until moderate fatigue (slowing of movement, determined by the same researcher) in the 7 resistance exercises mentioned above. Exercises were performed at an intensity of 50% of 1 RM, and resting intervals between sets and exercises lasted 90 s. After the intervention period, subjects returned to the laboratory and remained seated for 60 min (first post-intervention period – Post1). Afterwards, they had 20 min to take a shower and an ambulatory BP device was attached to their non-dominant arm. Subjects were released from the laboratory to their daily activities and returned after 5 h. At that time, they rested again in the seated position for

Queiroz ACC et al. Captopril does not Potentiate... Int J Sports Med

Downloaded by: Serials Unit - Periodicals. Copyrighted material.
Fig. 1 Experimental protocol.

60 min (second post-intervention period – Post2). Afterwards, they left the laboratory again with the ambulatory monitor and returned the next day, 24 h after the end of the intervention period. During the ambulatory periods, subjects were instructed to maintain their usual activities and to avoid physical exercise, alcohol ingestion, showering and daytime sleep. They were also asked to report and maintain similar activities after all the experimental sessions.

At Pre, Post1 and Post2 periods, BP, CO and HR were measured in triplicate and the mean value was calculated. In addition, ECG, respiratory activity and beat-by-beat BP were assessed for 10 min before the hemodynamic measurements for autonomic evaluation.

Measurements

BP was measured by auscultatory method on the dominant arm using a mercury column. Measurements were done by the same trained observer in all the experimental sessions of each subject. 2 researchers performed the measurements and the intraclass correlation coefficients between them were 0.883 and 0.889 for systolic and diastolic BP, respectively. HR was measured by ECG and radial pulse palpation immediately after the BP measurement. CO was estimated by the indirect Fick method, employing the CO2 rebreathing technique [15] and a metabolic cart (Medical Graphics Corporation, CPX/D, Minnesota, EUA), as previously reported [34]. SV and systemic vascular resistance (SVR) were calculated.

For autonomic evaluation, ECG, respiratory activity (UFI, Pneumotrace 2, California, USA) and beat-by-beat BP (Finapres Medical System, Finometer, Amsterdam, Netherlands) were acquired by a data acquisition system (WinDaq DI-720, Akron, Ohio, USA) with a sampling rate of 500 Hz/channel. Autoregressive spectral analysis of R-R interval variability was performed (Heart Scope II, AMPS LLC, New York, USA) as previously described [33]. Low- (LF_RR: 0.04–0.15 Hz) and high-frequency (HF_RR: 0.15–0.4 Hz) components were expressed in normalized units (nu) that were calculated as each power component relative to the total power minus the very low-frequency component (VLF: 0–0.04 Hz). The LF_RR and HF_RR components of R-R variability were accepted as markers of predominant cardiac sympathetic and parasympathetic modulations, respectively, and the ratio between these components was considered a marker of cardiac sympathovagal balance [33]. BRS was evaluated by the highest value of the transfer function between R-R interval and systolic BP variabilities at the LF component [31].

Ambulatory data were measured every 15 min for 24 h by an oscillometric device (Spacelabs 90207, Spacelabs Inc, Redmond, Washington, USA) whose calibration was regularly checked [4]. Data were analyzed by the following means: 24 h (all measurements for 24 h), interval between Post1–2 (all measures between Post1 and Post2 periods); awake (all measurements while the subject reported to be awake), and asleep (all measurements while the subject reported to be sleeping) periods.

ACE activity was evaluated following the protocol of Alves et al. [1]. Briefly, 5 µl serum was added to an assay solution containing 10 µM Abz-FRK(Dnp)P-OH and a buffer (0.1 M Tris-HCl with 50 mM NaCl and 10 µM ZnCl2), totaling 200 µl. Enzymatic activity was monitored by continuous recording of the fluorescence (λex = 320 nm and λem = 420 nm), and captopril was used as a negative control.

Statistical analysis

Considering a power of 90 %, an alpha error of 5 %, and a standard deviation of 3 mmHg for systolic BP and 0.32 L/min for CO, the minimal sample sizes necessary to detect a difference of 4 mmHg and 0.32 L/min were calculated to be 10 and 11 subjects in each group, respectively.

Data distribution was checked by the Shapiro-Wilks test (SPSS for Windows, Illinois, USA), and logarithm transformation was applied for non-normally distributed variables.

In each therapy period (placebo and captopril), absolute values measured pre- and post-interventions in both sessions were compared by a 2-way ANOVA for repeated measures, establishing: sessions (Control and Exercise) and stages (Pre, Post1 and Post2) as main factors (Statsoft, Statistica for Windows, Oklahoma, USA). In addition, absolute ambulatory averages were compared by a paired t-test. Whenever necessary, post-hoc comparisons were done by the Newman-Keuls test. In addition, the effect sizes (Cohen’s d) and its confidence intervals (CI.95) of the main clinical variables were calculated for each treatment period.
For comparing the effect of previous exercise between the 2 therapy periods, the exercise net effect was calculated for each therapy by the formula: Exercise net effect = (post-exercise – pre-exercise) – (post-control – pre-control). The exercise net effect of ambulatory variables was calculated by: Exercise net effect = exercise session – control session. The net effects obtained with placebo and captopril were compared by paired t-test. P ≤ 0.05 was defined as significant. Data are presented as mean ± SE.

Results
36 HT men volunteered for the study. During preliminary evaluation, 23 were excluded for not fulfilling the study criteria or not having time for study participation. 13 subjects began the experimental sessions, and one dropped out because of incorrect use of medication. Thus, 12 subjects completed the study protocol and their characteristics are shown in ▶ Table 1.

7 subjects began the crossover therapy periods with captopril and 5 with placebo. 6 subjects began the experimental sessions with C and 6 with RE. Adherence to captopril use was 97.4 ± 2.0 % (pill counting). ACE activity, systolic and diastolic BP were significantly lower with captopril compared with placebo (83 ± 11 vs. 175 ± 5 × 10^8 uF.min⁻¹.ml⁻¹, 127 ± 3 vs. 135 ± 3 mmHg, 85 ± 2 vs. 91 ± 2 mmHg, respectively). The workloads employed for each exercise were similar with placebo and captopril (52 ± 3 vs. 51 ± 3 kg for chest press, 93 ± 5 vs. 94 ± 4 kg for leg press, 37 ± 2 vs. 37 ± 2 kg for lat pull down, 59 ± 4 vs. 58 ± 4 kg for squat, 16 ± 1 vs. 16 ± 1 kg for arm curl, 18 ± 1 vs. 18 ± 1 kg for right leg curl, and 18 ± 1 vs. 18 ± 1 kg for left leg curl, respectively, P > 0.05).

Physiological behaviors after C and RE sessions were similar with placebo (▶ Fig. 2a) and captopril (▶ Fig. 2b). At Post1, with both therapies, systolic and diastolic BPs decreased only after RE. In addition, SVR increased and CO decreased significantly and similarly in both C and RE; whereas SV decreased and HR increased only after RE. HF and nu decreased after RE, whereas LF and nu and LF/HF increased. BRS increased after C, and did not change after RE. The exercise effects sizes calculated for these variables at Post1 were: systolic BP = placebo d = 0.977 (CI: –0.22, 2.17) and captopril d = 0.936 (CI: –0.26, 2.13); diastolic BP = placebo d = 1.393 (CI: 0.08, 2.71) and captopril d = 1.278 (CI: –0.02, 2.58); SVR = placebo d = 0.406 (CI: –0.73, 1.55) and captopril d = 0.148 (CI: –0.99, 1.28);

![Table 1](image)

<table>
<thead>
<tr>
<th>N</th>
<th>12</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, yrs</td>
<td>50 ± 3</td>
</tr>
<tr>
<td>Weight, kg</td>
<td>82.6 ± 3.1</td>
</tr>
<tr>
<td>Height, cm</td>
<td>172 ± 2</td>
</tr>
<tr>
<td>Body mass index, kg/m²</td>
<td>27.8 ± 0.8</td>
</tr>
<tr>
<td>Resting systolic BP, mmHg (&amp;)</td>
<td>134 ± 3</td>
</tr>
<tr>
<td>Resting diastolic BP, mmHg (&amp;)</td>
<td>94 ± 1</td>
</tr>
<tr>
<td>Resting heart rate, bpm</td>
<td>65 ± 3</td>
</tr>
</tbody>
</table>

All values are expressed as means ± SE. BP = blood pressure, (&) = Values measured 3 times after 5 min of seated rest in 2 visits after 2-week washout period

Discussion
The most important findings in this study were that: a) captopril did not potentiate PREH in HT men; b) hypotension occurred inside the laboratory and lasted for 60 min, but it was not sustained under ambulatory conditions; c) the systemic hemodynamic determinant of hypotension (CO or SVR) varied among the subjects; d) BP reduction was accompanied by SV reduction, HR increase, cardiac sympathetic modulation enhancement, and BRS reduction; and e) these hemodynamic and autonomic alterations were also not sustained long after the exercise.

To the best of our knowledge, this is the first study to describe the effect of an ACE inhibitor on PREH. Based on the effects of captopril decreasing angiotensin II formation [8] and sympathetic activity [7], and improving endothelial function [14], the hypothesis was that captopril would potentiate PREH by potentiating SVR decrease after exercise. Contradicting this hypothesis, post-exercise physiological responses were similar with and without captopril.

The effectiveness of captopril was demonstrated by the reduction in ACE activity and by the decrease in systolic and diastolic BP of 5.9 and 6.6 %, respectively, after 4 weeks of therapy. This magnitude of reduction is within the range usually observed when captopril is administered as a monotherapy in HT [11]. Nevertheless, despite the effectiveness on decreasing baseline BP, captopril did not potentiate PREH magnitude and duration and did not change its mechanisms. 2 main reasons might explain this result. First, the stringent inclusion and exclusion criteria adopted in the present study (absence of obesity, diabetes and other alterations) might have resulted in a sample with few physiological alterations that might be changed by captopril. Second, therapies were administered for just 4 weeks because of ethical concerns (keeping patients on placebo for more than 8 weeks ‒ 2 washout weeks + 4 weeks of experiment + 2 weeks of new washout before the next therapy). It is possible that this period was not enough to induce more expressive changes in hemodynamic and autonomic mechanisms. Actu-
ally, the studies that reported ambulatory PREH in medicated HT involved subjects who were receiving anti-hypertensive drugs chronically for at least 2 months [22, 32].

Regardless of drug use, RE produced a significant PREH in HT men. The systolic/diastolic BP reductions observed after exercise (Placebo = –13 ± 2/–9 ± 1 and captopril = –12 ± 2/–10 ± 1 mmHg) were similar [22, 23] or greater [6, 32] than the changes previously reported after resistance exercise in medicated HT. In addition, they were also similar to the decreases usually reported after aerobic exercise [24]. Nevertheless, concerning PREH duration, BP reduction was observed at Post1, but not during the ambulatory period between Post1 and 2, and at Post2. Thus, it lasted only for approximately 60 min. To the best of our knowledge, just 3 studies reported ambulatory BP decrease after resistance exercise. One was conducted with women receiving captopril [22], one with HT men and women receiving different drugs [32] and one with non-motensive and non-medicated HT African men [26]. Together these studies suggest that population characteristics may not be the key point for differences. In one of these studies [32], ambulatory PREH was greater after a greater volume RE; in the other one [26], however, ambulatory PREH was observed after a very low-volume session (8 exercise, 1 set, 8–12 repetitions). Thus, based on available data, it is possible to suggest that, unless under specific conditions, PREH was not sustained for a long period after exercise in HT subjects. Therefore, the specific conditions that may prolong PREH should be studied in the future to enhance the clinical relevance of this phenomenon.

Regardless of captopril use, PREH could not be attributed to a decrease of either CO or SVR, because they varied in the same direction after both C and RE sessions with both therapies. Interestingly, however, part of the sample (placebo = 33 %, captopril = 50 %) presented a decrease in CO and another part (placebo = 50 %, cap-

Fig. 2 Systolic blood pressure (SBP), diastolic blood pressure (DBP), cardiac output (CO), systemic vascular resistance (SVR), stroke volume (SV), heart rate (HR), high-frequency component of RR interval variability (HFRR), low-frequency component of RR interval variability (LFRR), low to high frequency component ratio of RR interval variability (LF/HFRR) and baroreflex sensitivity (BRS) measured before and after 30-60 min (Post1) and 7 hours (Post2) the interventions in the control (dashed line) and resistance exercise (solid line) sessions while hypertensive subjects were receiving placebo (panel A) and captopril (panel B). All values are expressed as means±SE. * Significantly different from Pre (P ≤ 0.05). # Significantly different from the control session (P ≤ 0.05).
Captopril does not Potentiate... Int J Sports Med

Physiology & Biochemistry Thieme

Queiroz ACC et al. Captopril does not Potentiate... Int J Sports Med

Physiology & Biochemistry Thieme

Queiroz ACC et al. Captopril does not Potentiate... Int J Sports Med

Table 2 Ambulatory blood pressure measured in the control (C) and the resistance exercise (RE) sessions.

<table>
<thead>
<tr>
<th>Placebo Therapy</th>
<th>24 h</th>
<th>Awake Post1–2</th>
<th>Awake</th>
<th>Asleep</th>
</tr>
</thead>
<tbody>
<tr>
<td>Systolic BP, mmHg</td>
<td>C</td>
<td>138 ± 3</td>
<td>143 ± 3</td>
<td>144 ± 3</td>
</tr>
<tr>
<td>RE</td>
<td>139 ± 3</td>
<td>145 ± 4</td>
<td>144 ± 4</td>
<td>125 ± 3</td>
</tr>
<tr>
<td>Diastolic BP, mmHg</td>
<td>C</td>
<td>91 ± 3</td>
<td>96 ± 3</td>
<td>96 ± 2</td>
</tr>
<tr>
<td>RE</td>
<td>90 ± 2</td>
<td>96 ± 3</td>
<td>95 ± 3</td>
<td>78 ± 3</td>
</tr>
<tr>
<td>Mean BP, mmHg</td>
<td>C</td>
<td>107 ± 3</td>
<td>112 ± 3</td>
<td>112 ± 3</td>
</tr>
<tr>
<td>RE</td>
<td>106 ± 3</td>
<td>113 ± 3</td>
<td>111 ± 3</td>
<td>93 ± 3</td>
</tr>
<tr>
<td>Heart Rate, bpm</td>
<td>C</td>
<td>75 ± 3</td>
<td>80 ± 3</td>
<td>78 ± 3</td>
</tr>
<tr>
<td>RE</td>
<td>74 ± 2</td>
<td>82 ± 3</td>
<td>77 ± 2</td>
<td>67 ± 2</td>
</tr>
</tbody>
</table>

Captopril Therapy

| Systolic BP, mmHg | C | 134 ± 3 | 141 ± 4 | 140 ± 3 | 120 ± 4 |
| RE | 134 ± 3 | 140 ± 3 | 141 ± 3 | 119 ± 3 |
| Diastolic BP, mmHg | C | 86 ± 2 | 92 ± 3 | 92 ± 2 | 74 ± 3 |
| RE | 87 ± 2 | 92 ± 3 | 93 ± 2 | 74 ± 2 |
| Mean BP, mmHg | C | 102 ± 3 | 108 ± 3 | 108 ± 3 | 89 ± 3 |
| RE | 102 ± 2 | 107 ± 3 | 108 ± 2 | 90 ± 2 |
| Heart Rate, bpm | C | 73 ± 2 | 80 ± 4 | 76 ± 3 | 65 ± 2 |
| RE | 75 ± 3 | 82 ± 3 | 78 ± 3 | 68 ± 3 |

Post1 – 2 = 5-h interval between the post-intervention measurements 1 and 2. Values = means ± SE.

topril = 42 %) a reduction in SVR. This individualized hemodynamic behavior has already been reported after aerobic [9] as well as resistance [28] exercise in normotensives, but the reasons why some subjects presented a decrease in CO and others in SVR are unclear at the moment and should be investigated in the future. It is interesting to observe that, unlike as hypothesized, captopril use for 4 weeks was not able to enhance SVR decrease after resistance exercise, which suggests that mechanisms other than the renin-angiotensin system might prevail for this physiological adjustment. Studies with anti-hypertensive drugs that act on other physiological pathways may help to clarify this aspect.

Although CO and SVR responses varied among the subjects, in all of them BP reduction was accompanied by SV reduction, as observed in other studies [24, 27, 29, 34]. The mechanisms involved in SV decrease were not investigated in the present study; but reduction in pre-load seems to be the most probable mechanism because resistance exercise decreases plasma volume and venous return [5]. SV decrease was accompanied by an increase in HR that may reflect many mechanisms, including cardiopulmonary deactivation by preload decrease, baroreflex response to BP reduction, and thermoregulation, among others [30]. However, because exercise decreased BRS, HR increase was not able to blunt PREH. HR increase was accompanied by an increase in cardiac sympathetic and a decrease in cardiac parasympathetic modulations, which was observed by the spectral analysis of HR variability (decreased HFnu and increased LFnu and LF/HF).

This study has some limitations that should be addressed in future investigations. Physiological responses after RE might change with different exercise protocols. The present study employed an exercise protocol recommended for HT [2, 3, 37] conducted at low intensity to moderate fatigue. Higher exercise intensities have been shown to promote greater decreases in SV and increase in HR and cardiac sympathovagal during the recovery period [18, 29]. Thus,
captopril’s influence on PREH characteristics and mechanisms might be different after more intense exercise. In addition, the study included only sedentary men and subjects without comorbidities, and the drug was administered for only 4 weeks. Captopril’s influence might also be different in other populations and used for a longer period of time. Finally, this study showed that the hemodynamic mechanism of PREH varies among the subjects, but it did not employ a specific analysis of individual responses; which might be interesting to be done in the future because responses to drugs and exercise present individual variations.

Regarding the clinical and practical application of the findings, Luttrell & Halliwill [19] recently reported that acute physiological responses after exercise might represent a window of opportunity or a vulnerable state seems inadequate in this population. However, PREH has been shown to directly correlate with chronic hypotensive effect of resistance training, suggesting that subjects who present greater PREH also present greater BP decrease after a resistance training period [35]. Thus, by showing that an acute bout of RE can produce significant PREH of 12–13 mmHg in HT men, the results suggest that this population (HT receiving or not receiving captopril) may also respond to resistance training with a decrease in BP, and perhaps with a reduction in the drug treatment. These hypotheses, however, should be investigated in the future.

In conclusion, captopril did not potentiate the magnitude and duration of PREH in HT men and did not influence PREH hemodynamic and autonomic mechanisms. PREH had a significant magnitude but did not last under ambulatory conditions. The hemodynamic determinant varied from one subject to another, and PREH was accompanied by an SV reduction and HR increase promoted by a cardiac sympathetic cardiac modulation increase and BRS reduction. These alterations likewise did not last longer after exercise.

Acknowledgements

The authors want to acknowledge the subjects who contributed to this study and Dr. Giovânia Vieira da Silva for medical care provided to the subjects. In addition, we like to thank the General Hospital staff, the members of the Laboratory of Biochemistry and Molecular Biology of Exercise (Glória F. Mota and Tiago Fernandes), of the Laboratory of Molecular and Cellular Exercise Physiology (Alex W. Monteiro and Marcele A. Coelho) and of the Exercise Hemodynamic Laboratory (Andrezia A. P. Cavalli and Luiz A. R. Costa) who helped in data collection. This study was financially supported by FAPESP (2009/18219-3; 2011/06689-5), CNPQ (146168/2011-9; 237320/2012-6; 304003/2014-0), CAPES-PROEX, and Pró-Reitoria de Pós-Graduação USP.

Conflict of interest

Authors have no conflict of interest to declare.

References
