

Post-concurrent exercise hemodynamics and cardiac autonomic modulation

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Abstract Concurrent training is recommended for health improvement, but its acute effects on cardiovascular function are not well established. This study analyzed hemodynamics and autonomic modulation after a single session of aerobic (A), resistance (R), and concurrent (A + R) exercises. Twenty healthy subjects randomly underwent four sessions: control (C:30 min of rest), aerobic (A:30 min, cycle ergometer, 75% of VO_2 peak), resistance (R:6 exercises, 3 sets, 20 repetitions, 50% of 1 RM), and concurrent (AR: A + R). Before and after the interventions, blood pressure (BP), heart rate (HR), cardiac output (CO), and HR variability were measured. Systolic BP decreased after all the exercises, and the greatest decreases were observed after the A and AR sessions (-13 ± 1 and -11 ± 1 mmHg, respectively, $P < 0.05$). Diastolic BP decreased similarly after all the exercises, and this decrease lasted longer after the A session. CO also decreased similarly after the exercises, while systemic vascular resistance increased after the R and AR sessions in the recovery period ($+4.0 \pm 1.7$ and

$+6.3 \pm 1.9$ U, respectively, $P < 0.05$). Stroke volume decreased, while HR increased after the exercises, and the greatest responses were observed after the AR session (SV, $A = -14.6 \pm 3.6$, $R = -22.4 \pm 3.5$ and $AR = -23.4 \pm 2.4$ ml; HR, $A = +13 \pm 2$, $R = +15 \pm 2$ vs. $AR = +20 \pm 2$ bpm, $P < 0.05$). Cardiac sympathovagal balance increased after the exercises, and the greatest increase was observed after the AR session ($A = +0.7 \pm 0.8$, $R = +1.0 \pm 0.8$ vs. $AR = +1.2 \pm 0.8$, $P < 0.05$). In conclusion, the association of aerobic and resistance exercises in the same training session did not potentiate post-exercise hypotension, and increased cardiac sympathetic activation during the recovery period.

Keywords Blood pressure · Heart rate variability · Strength training · Cardiovascular risk

Introduction

A single bout of exercise produces controversial effects on cardiovascular load during the recovery period. After exercise, blood pressure (BP) falls below pre-exercise levels (Brandao Rondon et al. 2002; Forjaz et al. 1998, 2004; Halliwill et al. 1996; Hayes et al. 2000; Jones et al. 2007; Pescatello et al. 2004a; Pricher et al. 2004; Queiroz et al. 2009; Raine et al. 2001; Rezk et al. 2006; Senitko et al. 2002; Takahashi et al. 2000). On the other hand, heart rate (HR) persists elevated for many minutes or hours after exercise (Dujic et al. 2006; Forjaz et al. 1998, 2004; Heffernan et al. 2006; Kannankeril et al. 2004; Mourou et al. 2004; Parekh and Lee 2005; Queiroz et al. 2009; Rezk et al. 2006; Savin et al. 1982; Seiler et al. 2007).

The mechanisms underlying post-exercise hypotension remain controversial. However, when recovery is

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conducted in the sitting position, decreases in BP observed after aerobic (Brandao Rondon et al. 2002; Dujic et al. 2006; Forjaz et al. 2004; Senitko et al. 2002; Takahashi et al. 2000) as well as resistance (Rezk et al. 2006) exercises have been attributed to a decrease in stroke volume (SV), leading to a reduction in cardiac output (CO) that was not compensated by an increase in systemic vascular resistance (SVR). Moreover, after both aerobic (Heffernan et al. 2006; Kannankeril et al. 2004; Parekh and Lee 2005; Savin et al. 1982; Seiler et al. 2007) and resistance (Heffernan et al. 2006; Rezk et al. 2006) exercises, the increase in HR during the recovery period has been attributed to a persistent increase in sympathetic modulation, and a decrease in parasympathetic modulation to the heart.

Because aerobic training leads to cardiovascular benefits, while resistance training promotes musculoskeletal benefits, their combination (called concurrent training) has been recommended for health and fitness improvement (American College of Sports Medicine 2006). Nevertheless, the effects of concurrent exercise on post-exercise hypotension, as well as the physiological mechanisms underlying this response have not yet been studied. Since physiological responses after both modes of exercise were similar, we hypothesized that their association might have an additive effect, leading to a greater decrease in BP, and also promoting a greater increase in HR during the recovery period.

Thus, the aim of the present study was to evaluate the effects of a single session of aerobic, resistance, and concurrent exercises on post-exercise hypotension and to analyze the physiological mechanisms involved in this response. This study was conducted with healthy subjects in order to understand post-exercise physiology without pathological influences.

Methods

Subjects and preliminary testing

Twenty young normotensive subjects (10 women and 10 men) gave written consent to take part in the present study, which was approved by the Ethics Committee of the General Hospital, Medical School, University of São Paulo, Brazil.

Prior to the enrollment in the study, all subjects were asked about their health status and physical activity levels. They were excluded if they reported any current disease, medication use or regular physical activity practice. Weight and height were measured, and subjects were also excluded if body mass index was ≥ 25 kg/m². Auscultatory seated BP was measured three times in two visits to the laboratory, using a mercury sphygmomanometer and

employing, respectively, phases I and V of the Korotkoff's sounds for systolic and diastolic BP determinations. In addition, BP was also measured at sitting rest in the pre-intervention period of all the experimental session. The mean value of all these measures was calculated and applied to determine subjects' BP levels. All subjects had systolic and diastolic BP levels ≤ 120 and ≤ 80 mmHg, respectively (Chobanian et al. 2003).

Prior to the experiments, maximum aerobic and resistance performances were assessed. Peak oxygen uptake (VO₂ peak) was measured directly by a metabolic cart (MGC, CAD/NET 2001) during a maximum exercise test performed on a cycle ergometer using a protocol with 30 W increments every 3 min until exhaustion. Maximal strength was assessed using one repetition maximum (1RM) test in bench press, leg press, lat pull down, knee flexion, arm curl, and squat in smith machine (Kraemer and Fry 1995). Before the 1RM test, subjects underwent four familiarization sessions (3 sets, 20 repetitions of each exercise with the minimum weight allowed by the machines) on non-consecutive days. Subjects' characteristics are shown in Table 1.

Experimental protocol

All subjects underwent, in a random order, four experimental sessions: control (C), aerobic (A), resistance (R), and concurrent (AR) exercises. Each session was initiated between 1 and 3 p.m., and an interval of at least 5 days was maintained between them. Subjects were instructed to take

Table 1 Physical, cardiovascular and fitness characteristics of the subjects ($n = 20$)

Characteristics	Value
Men/women	10/10
Age (year)	26 \pm 1
Weight (kg)	62.7 \pm 1.9
Height (m)	1.68 \pm 0.02
Body mass index (kg/m ²)	22.1 \pm 0.4
Resting systolic blood pressure (mmHg)	111 \pm 2
Resting diastolic blood pressure (mmHg)	74 \pm 1
Resting mean blood pressure (mmHg)	86 \pm 1
Resting heart rate (bpm)	71 \pm 2
Peak workload (watts)	161 \pm 9
Peak oxygen uptake (ml kg ⁻¹ min ⁻¹)	33.4 \pm 1.5
1-RM bench press (kg)	64 \pm 8
1-RM leg press (kg)	104 \pm 5
1-RM lat pull down (kg)	47 \pm 4
1-RM knee flexion (kg)	26 \pm 3
1-RM arm curl (kg)	20 \pm 2
1-RM squat in smith machine (kg)	43 \pm 6

a light meal 2 h before the experiments, to avoid physical exercise and alcohol ingestion for at least the prior 48 h, and to avoid smoking and caffeine in the previous 12 h.

In each session, subjects rested in the seated position for 20 min (pre-intervention). Then, they moved to the exercise room, where they remained resting in the C session, and exercising in the A, R, and AR sessions. Subjects were blinded to the sessions that they were going to perform until the exercise or the rest began.

In the A session, subjects performed 30 min of exercise on a cycle ergometer at 75% of VO_2 peak. In the R session, subjects performed 3 sets of 20 repetitions in 6 resistance exercises (bench press, leg press, lat pull down, knee flexion, arm curl, and squat in smith machine) with a workload of 50% of 1 RM. Intervals of 45 and 90 s were guaranteed between the sets and exercises, respectively, completing a 30-min exercise session. The AR session was composed of the execution of the exercise protocol performed in the A session followed by the exercise protocol performed in the R session, completing a 60-min exercise session. In the C session, subjects rested in the cycle ergometer for 30 min, and then they moved to the resistance exercise machines, where they stayed resting for 30 min. This session simulates the AR session without the exercise execution.

After the interventions, subjects returned to the laboratory, where they rested in the sitting position for 120 min (post-intervention period).

Hemodynamic data were measured in triplicate in the pre-intervention, and at 30, 60, 90, and 120 min of the post-intervention period. The median value measured in each moment was used for analysis. Moreover, ECG and respiratory data were collected for autonomic assessment for 10 min in the pre-intervention period, and between 20–30 and 50–60 min of the post-intervention period.

Measurements

BP was measured by the auscultatory method, using a mercury sphygmomanometer immediately before CO determination. Mean BP was calculated by the sum of diastolic BP and one third of pulse pressure. ECG was continuously monitored (TEB, M-10), and HR was registered immediately after BP measurement. The rate pressure product was calculated by the product of systolic BP and HR.

CO was estimated by the indirect Fick method of CO_2 rebreathing (Jones et al. 1967) using a metabolic cart (MGC, CAD/NET 2001). Briefly, subjects breathed spontaneously until a steady CO_2 production was achieved. This procedure was followed by the rebreathing of a mixed gas with high CO_2 concentration (8–10%) and 35% of O_2 until an equilibrium was achieved (maximal of 15 s). At this

moment, CO was calculated by the Fick formula. SVR was calculated by the quotient between mean BP and CO, and SV by the quotient between CO and HR.

For autonomic evaluation, ECG (TEB, D10) and respiratory movements (thoracic belt-UFI, Pneumotrace II) were recorded for 10 min with a sample frequency of 500 Hz per channel. R–R intervals and respiratory time series were determined by PRE software (Calcolo Segnali di Variabilità Cardiovascolari—20/06/95, Dipartimento di Bioingegneria Del Politécnico di Milano). An autoregressive spectral analysis of R–R variability was performed using LA software (Programma di Analisi Lineare—14/12/1999, Dipartimento di Scienze Precliniche, Università Degli Studi di Milano); its theoretical and analytical procedures were described previously (Task 1996). Briefly, on stationary segments of the time series, autoregressive parameters were estimated by the Levinson–Durbin recursion, and the order of the model was chosen according to the Akaike's criterion. An autoregressive spectral decomposition was performed, and the components were assigned based on their central frequency as low- ($\text{LF}_{\text{R-R}}$, 0.04–0.15 Hz) and high-frequency ($\text{HF}_{\text{R-R}}$, 0.15–0.4 Hz). HF power was considered in dependence to a significant coherence with the respiratory spectrum. $\text{LF}_{\text{R-R}}$ and $\text{HF}_{\text{R-R}}$ components were reported in normalized units (nu), which represent the relative value of each power component in proportion to the total power minus the very low-frequency component ($\text{VLF}_{\text{R-R}}$, 0–0.04 Hz). Normalized $\text{LH}_{\text{R-R}}$ and $\text{HF}_{\text{R-R}}$ components were accepted, respectively, as markers of the predominant cardiac sympathetic and parasympathetic modulations (Task 1996).

Statistical analysis

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

The Gaussian distribution of the data was verified by the Shapiro–Wilk test, and a natural logarithmic (ln) transformation was employed when necessary (for variables: absolute LF, absolute HF, HFnu, and LF/HF). Changes in hemodynamic and autonomic parameters in each experimental session were calculated by the difference between post- and pre-intervention values. These changes were compared by a two-way analysis of variance (ANOVA) for repeated measures (Statistica for Windows 5.1, Statsoft Inc.), establishing sessions and stages as the main factors. For all the analyses gender was included as a covariate. Post-hoc comparisons were done by Newman–Keuls test. $P < 0.05$ was accepted as statistically significant. Data are presented as mean \pm standard error.

Results

Five subjects initiated the protocol with the C session, 4 with the A session, 6 with the R session, and 5 with the AR session. In the A session, subjects exercised at $75 \pm 1\%$ of VO_2 peak. To achieve this intensity, the workload was set at 1.4 ± 0.1 kpm in a mechanic cycle ergometer. In the R session, the loads used in each exercise were: 31.4 ± 4.2 kg for bench press, 51.3 ± 2.7 kg for leg press, 22.7 ± 1.7 kg for lat pull down, 12.2 ± 1.4 kg for knee flexion, 10.0 ± 0.8 kg for arm curl, and 22.5 ± 2.9 kg for squat in smith machine. These loads corresponded, respectively, to 48.9 ± 0.7 , 49.3 ± 0.4 , 48.6 ± 0.7 , 48.0 ± 1.4 , 51.5 ± 1.5 , and $57.3 \pm 4.1\%$ of 1RM.

Almost all of the hemodynamic and autonomic variables were similar among the experimental sessions before the interventions (Table 2), except for mean BP that was slight lower in the C and AR sessions than in the A session ($P < 0.05$).

In comparison with the pre-intervention values, systolic BP (Fig. 1a) did not change after the C session, and decreased after the A, R and AR sessions (greatest decreases = -13 ± 1 , -8 ± 1 , and -11 ± 1 mmHg, respectively, $P < 0.05$). The decreases were greater after the A, R, and AR than after the C session ($P < 0.05$). Moreover, they were also greater after the A and AR than after the R session ($P < 0.05$), and they were lower after the AR than after the A session at 120 min ($P < 0.05$).

In comparison with pre-intervention values, diastolic BP (Fig. 1b) increased after the C (greatest increase = $+4 \pm 1$ mmHg, $P < 0.05$), and decreased after the R, AR, and A sessions until 30, 60, and 90 min, respectively

(greatest decreases = -2 ± 1 , -3 ± 1 , and -3 ± 1 mmHg, respectively, $P < 0.05$). The decreases after the A, R, and AR were greater than after the C session ($P < 0.05$).

In comparison with pre-intervention values, mean BP (Fig. 1c) increased after the C session at 90 and 120 min of the post-intervention period (greatest increase = $+3 \pm 1$ mmHg, $P < 0.05$), and decreased after the A, R, and AR sessions at all the recovery stages (greatest decreases = -6 ± 1 , -4 ± 1 , and -6 ± 1 mmHg, respectively, $P < 0.05$). The decreases were greater after the A, R, and AR than after the C session ($P < 0.05$), and they were lower after the R than after the AR and A sessions ($P < 0.05$).

In comparison with pre-intervention values, rate pressure product (Fig. 1d) decreased after the C session (greatest decrease = -861 ± 156 mmHg.bpm, $P < 0.05$), and increased after the A, and the R sessions at 30 min, and after the AR session until 60 min of recovery (greatest increase = $+664 \pm 176$, $+1,101 \pm 199$, and $+1,336 \pm 228$ mmHg.bpm, respectively, $P < 0.05$). Afterwards, in all the exercise sessions, it returned to baseline, and then decreased below pre-exercise values after the A session at 90 and 120 min, and after the R and the AR sessions at 120 min (greatest decrease = $-1,008 \pm 176$, -633 ± 179 and -495 ± 162 mmHg.bpm, respectively, $P < 0.05$). The changes after the A, R, and AR were greater than after the C session ($P < 0.05$). In addition, they were also greater after the R than after the A session ($P < 0.05$), and after the AR than after the R and A sessions ($P < 0.05$).

In comparison with the pre-intervention values, CO (Fig. 2a) decreased similarly after the C, A, R, and AR sessions at all the recovery periods ($P < 0.05$).

Table 2 Hemodynamic and autonomic parameters measured pre-interventions in the control (C), aerobic (A), resistance (R) and concurrent (AR) exercise sessions in normotensive subjects ($n = 20$)

	Sessions			
	C	A	R	AR
Systolic BP (mmHg)	109 ± 2	111 ± 2	110 ± 2	110 ± 1
Diastolic BP (mmHg)	74 ± 1	75 ± 1	75 ± 1	73 ± 1
Mean BP (mmHg)	$85 \pm 1^{\S}$	87 ± 1	87 ± 1	$85 \pm 1^{\S}$
RPP (mmHg.bpm)	$7,634 \pm 263$	$7,863 \pm 288$	$7,666 \pm 269$	$7,586 \pm 238$
CO (l/min)	4.5 ± 0.3	4.4 ± 0.3	4.3 ± 0.3	4.5 ± 0.2
SVR (U)	20 ± 1	21 ± 1	21 ± 1	20 ± 1
SV (ml)	67 ± 6	64 ± 5	65 ± 5	67 ± 4
HR (bpm)	70 ± 2	71 ± 2	70 ± 2	69 ± 2
$\text{LF}_{\text{R-Rnu}}$	60.8 ± 4.1	62.2 ± 3.8	65.5 ± 5.0	57.3 ± 4.6
$\ln \text{HF}_{\text{R-Rnu}}$	32.6 ± 4.3	31.6 ± 4.0	31.0 ± 4.8	35.9 ± 5.2
$\ln \text{LF}/\text{HF}$	5.3 ± 0.2	5.4 ± 0.2	5.4 ± 0.3	5.2 ± 0.2

BP blood pressure, RPP rate pressure product, CO cardiac output, SVR systemic vascular resistance, SV stroke volume, HR heart rate, LF low-frequency component, HF high-frequency component

[§] Significantly different from A ($P < 0.05$)

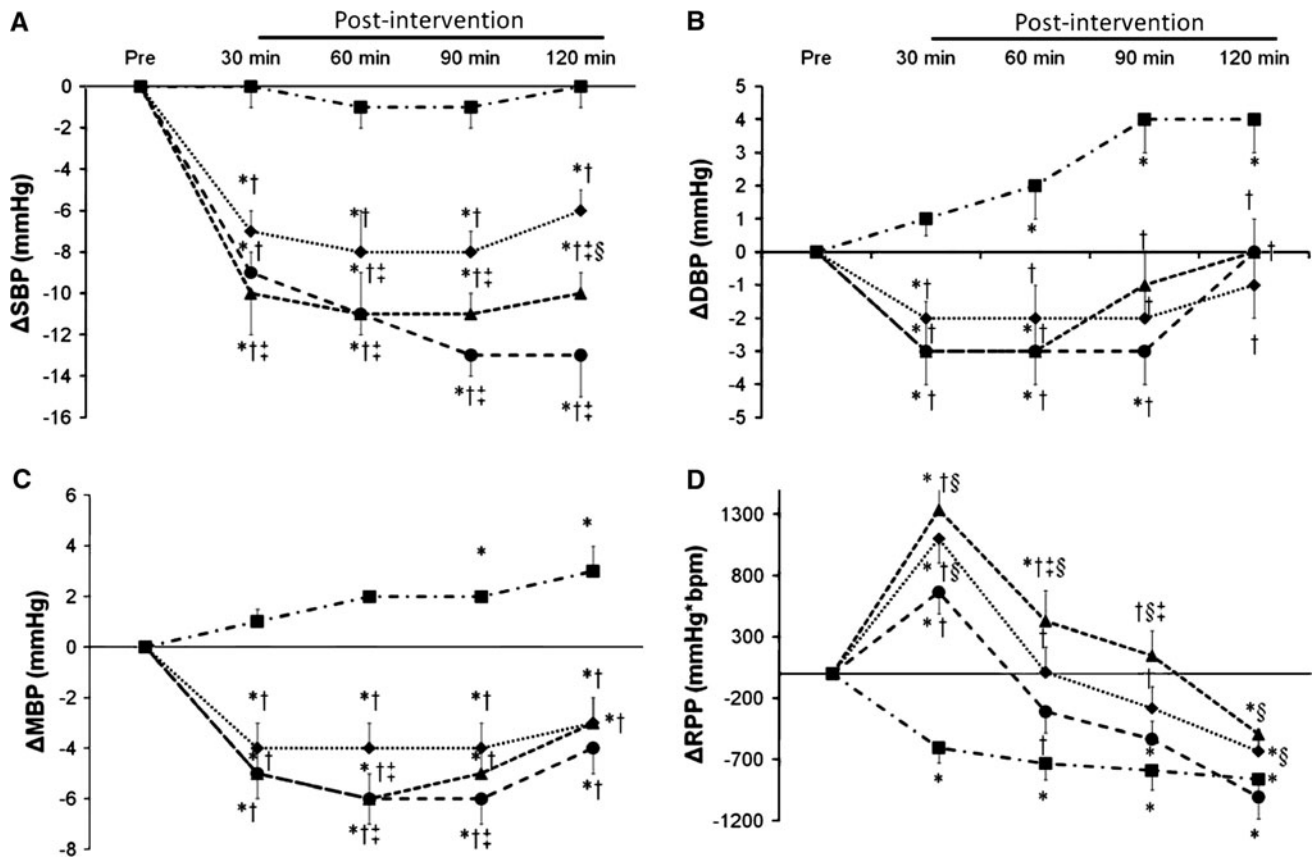


Fig. 1 Changes in systolic (Δ SBP, **a**), diastolic (Δ DBP, **b**), mean blood pressure (Δ MBP, **c**), as well as rate pressure product (Δ RPP, **d**) observed after interventions in the control (C, squares), aerobic (A, circles), resistance (R, diamonds), and concurrent (AR, triangles)

exercise sessions in normotensive subjects ($n = 20$). *Significantly different from pre, †significantly different from C, ‡significantly different from R, §significantly different from A ($P < 0.05$)

In comparison with pre-intervention values, SVR (Fig. 2b) did not change after the A session, and increased after the C, R, and AR sessions (greatest increases = $+6.9 \pm 1.5$, $+4.0 \pm 1.7$, and $+6.3 \pm 1.9$ U, respectively, $P < 0.05$). The increases were greater after the C than after the R and A sessions ($P < 0.05$), and they were also greater after the AR than after the A session ($P < 0.05$).

In comparison with pre-intervention values, SV (Fig. 2c) decreased after the C session at 90 and 120 min (greatest decrease = -9.2 ± 2.3 ml, $P < 0.05$), and after the A, R, and AR sessions at all the recovery stages (greatest decreases = -14.6 ± 3.6 , -22.4 ± 3.5 , and -23.4 ± 2.4 ml, respectively, $P < 0.05$). The decreases were greater after the A, R, and AR than after the C session ($P < 0.05$), and they were also greater after the R than the A session, and after the AR than after the A and R sessions ($P < 0.05$).

In comparison with pre-intervention values, HR (Fig. 2d) decreased after the C session (greatest decrease = -8 ± 1 bpm, $P < 0.05$), increased after the R session until 60 min (greatest increase = $+15 \pm 2$ bpm, $P < 0.05$), and after the A and AR sessions until 90 min (greatest increases = $+13 \pm 2$ and $+20 \pm 2$ bpm, respectively,

$P < 0.05$). The increases were greater after the A, R, and AR than after the C session ($P < 0.05$), and they were also greater after the R than the A session, and after the AR than the A and R sessions ($P < 0.05$).

Because of technical problems, autonomic data were not assessed in two subjects. These data are presented in Table 3.

In comparison with the pre-intervention values, the R-R interval increased after the C session at 50 min of recovery, and decreased after the A, R, and AR sessions at all the recovery stages ($P < 0.05$). The decreases after the A, R, and AR were greater than after the C session ($P < 0.05$), and they were also greater after the AR than the A and R, and after the A than the R session ($P < 0.05$).

In comparison with pre-intervention values, absolute lnLF did not change after the C session, and decreased after all the exercise sessions at 20 min of recovery. The decreases were greater after the A, R, and AR than after the C session ($P < 0.05$). Absolute lnHF did not change after the C session, and decreased after the A and R sessions at 20 min, and after AR session at both recovery stages ($P < 0.05$). The decreases after the A, R, and AR were

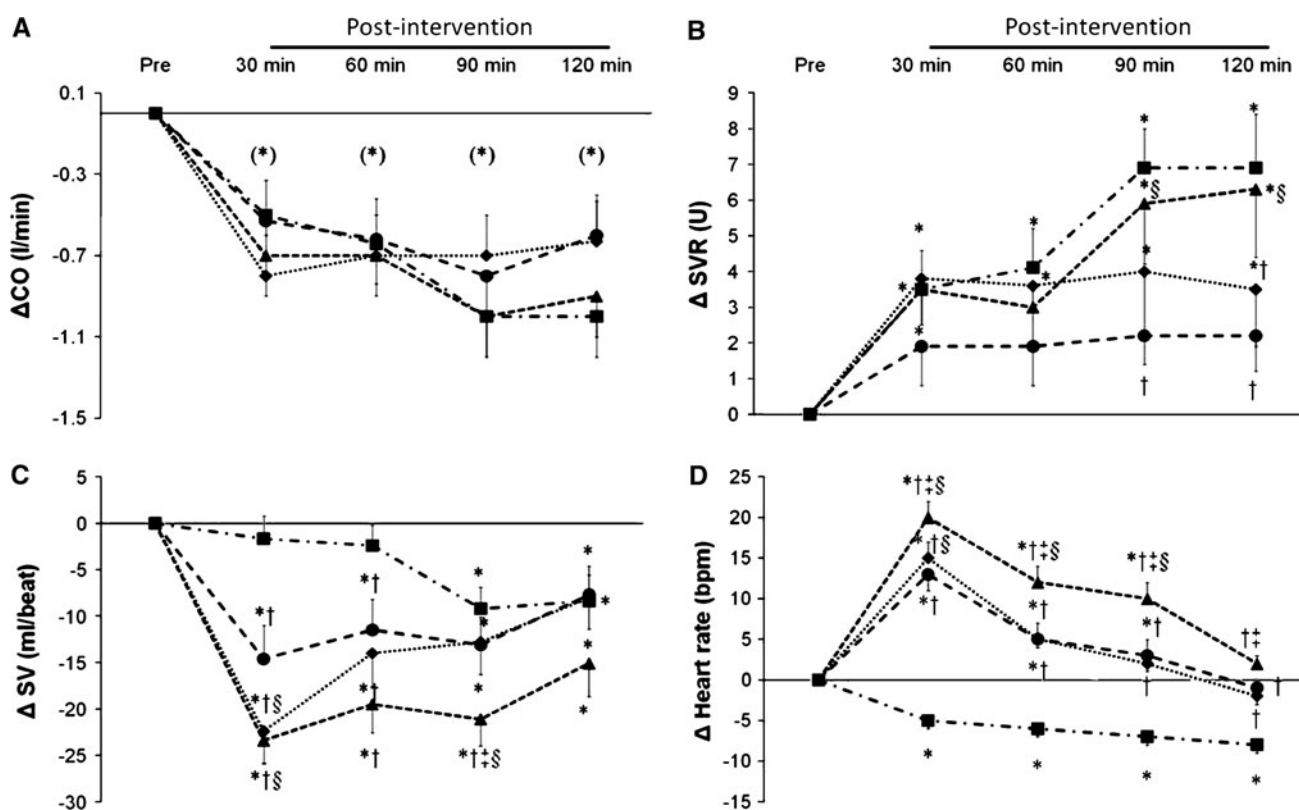


Fig. 2 Changes in cardiac output (CO, **a**), systemic vascular resistance (SVR, **b**), stroke volume (SV, **c**), and heart rate (HR, **d**) measured after interventions in the control (C, squares), aerobic (A, circles), resistance (R, diamonds), and concurrent (AR, triangles)

exercise sessions in normotensive subjects ($n = 20$). *Significantly different from pre, (*) main time effect, †significantly different from C, ‡significantly different from R, §significantly different from A ($P < 0.05$)

greater than after the C session ($P < 0.05$), and were also greater after the R than the A session, and after the AR than after the A and R sessions ($P < 0.05$).

In comparison with pre-intervention values, LF_{R-Rnu} and $\ln HF_{R-Rnu}$ did not change after the C session, but LF_{R-Rnu} increased and $\ln HF_{R-Rnu}$ decreased after the A and R sessions at 20 min, and after the AR session at all the recovery stages ($P < 0.05$). The changes were greater after the A, R, and AR than after the C session ($P < 0.05$), and they were also greater after the AR than after the A and R sessions ($P < 0.05$).

In comparison with pre-intervention values, $\ln LF/HF$ did not change after the C session, and increased after the A session at 20 min, and after the R and AR sessions at both recovery stages ($P < 0.05$). The increases were greater after the A, R, and AR than after the C session ($P < 0.05$), and they were greater after the AR than after the A and the R sessions ($P < 0.05$).

Discussion

The main findings of the present study were: (i) aerobic and resistance exercises, performed alone or in association,

produced post-exercise hypotension; this effect was greater after aerobic exercise, and was not potentiated by the association of the two exercise modalities; (ii) independently of the exercise modality, BP fall during the recovery period was attributed to a lack of SVR to increase enough in order to compensate for a decrease in CO, induced by a fall in VS; (iii) after aerobic and resistance exercises, HR and cardiac sympathetic modulation to the heart persisted elevated for many minutes, and the association of these two modes of exercise enhanced and prolonged these effects.

As expected, both exercise modes (aerobic and resistance), when performed alone, promoted post-exercise hypotension. The magnitude of BP falls was similar to the ones previously reported in literature (Pescatello et al. 2004a). Nevertheless, systolic BP falls were greater, and diastolic BP decreases lasted longer after the A and AR sessions than after the R session, showing that a greater post-exercise hypotensive effect was achieved when aerobic stimuli was present.

Nevertheless, conversely to our hypothesis, no additive effect was observed when aerobic and resistance exercises were combined. Systolic BP fall was not different between the A and AR sessions until 120 min of recovery, when it became greater in the A session. Although these results

Table 3 Changes in autonomic modulation indexes after the interventions in the control (C), aerobic (A), resistance (R) and concurrent (AR) exercise sessions in normotensive subjects ($n = 18$)

	Pre	Post-intervention	
		20 min	50 min
R-R interval (ms)			
C	0	+37 ± 18	+80 ± 19*
A	0	-151 ± 22*†	-86 ± 19*†
R	0	-149 ± 22*†	-53 ± 18*†§
AR	0	-204 ± 23*†‡§	-123 ± 14*†‡§
ln LF (ms²)			
C	0	-108 ± 478	+180 ± 333
A	0	-686 ± 387*†	-353 ± 224
R	0	-567 ± 157*†	-143 ± 174
AR	0	-428 ± 135*†	+199 ± 353
ln HF(ms²)			
C	0	+94 ± 86	+142 ± 197
A	0	-100 ± 201*†	-151 ± 74†
R	0	-494 ± 196*†§	-232 ± 150†
AR	0	-453 ± 114*†§	-382 ± 87*†‡§
LF_{R-Rnu}			
C	0	-0.2 ± 2.3	-5.2 ± 3.3
A	0	+10.0 ± 3.6*†	+1.1 ± 2.7
R	0	+11.4 ± 2.9*†	+4.1 ± 4.1†
AR	0	+23.3 ± 3.7*†‡§	+19.3 ± 3.0*†‡§
ln HF_{R-Rnu}			
C	0	-0.1 ± 2.5	+2.2 ± 3.2
A	0	-11.5 ± 2.9*†	-4.2 ± 2.4
R	0	-13.5 ± 2.8*†	-6.4 ± 4.7
AR	0	-21.0 ± 4.5*†‡§	-16.2 ± 3.6*†‡§
ln LF/HF			
C	0	0.0 ± 0.0	-0.1 ± 0.5
A	0	+0.7 ± 0.8*†	+0.2 ± 0.6
R	0	+1.0 ± 0.8*†	+0.4 ± 1.0*†
AR	0	+1.2 ± 0.8*†§	+1.0 ± 0.7*†‡§

LF low-frequency component, HF high-frequency component

* Significantly different from pre, † significantly different from C, ‡ significantly different from R, § significantly different from A ($P < 0.05$)

might suggest that resistance exercise blunted the hypotensive stimuli promoted by previous aerobic exercise, they should be analyzed with caution, taking into consideration the fact that time between the end of aerobic stimuli and recovery were different in the A and AR sessions. In our protocol, 120 min after the AR session represents 150 min after the end of the aerobic stimuli in the AR session. When comparing similar times after aerobic stimuli in the A and AR sessions (90 vs. 120 min of recovery, respectively) no difference was verified in BP decrease, showing that A and AR sessions had similar hypotensive effects. Moreover,

diastolic BP fall lasted until 60 min after the AR session, which corresponded to 90 min after the end of the aerobic stimuli, similarly to the behavior observed after the A session.

It is possible to speculate that the absence of an additive effect when aerobic and resistance exercises were combined might be related to the exercise intensity employed in the present investigation (75% of VO_2 peak). Some studies (Cornelissen and Fagard 2004; MacDonald et al. 1999; Pescatello et al. 2004b) have not observed any influence of exercise intensity on post-exercise hypotension. However, we (Forjaz et al. 2004) and others (Kenny et al. 2003; Piepoli et al. 1994; Quinn 2000) have previously shown that high-intensity aerobic exercise promoted greater post-exercise hypotension than lower intensity ones. Thus, it is possible that, a maximal hypotension had already occurred with the aerobic exercise, avoiding any additive effect of the association with the resistance exercise. This hypothesis is supported by the fact that the magnitude of BP fall observed after the AR session in the present study was similar to the greatest decreases reported for clinic BP values in normotensive subjects, and it was also similar to the ones reported for hypertensive subjects (Cornelissen and Fagard 2004; Pescatello et al. 2004a). However, this hypothesis should be specifically addressed in future research.

BP reductions after the three exercise sessions were accompanied by a decrease in CO, which has already been reported by us (Forjaz et al. 2004) and others (Brandao Rondon et al. 2002; Dujic et al. 2006; Senitko et al. 2002; Takahashi et al. 2000). However, CO also decreased in the C session, and in this session this reduction was due to a decrease in HR and a slight fall in SV. Nevertheless, in the C session, the decrease in CO was followed by an increase in SVR, which prevented the reduction in BP. This hemodynamic response has been observed previously in many studies (Forjaz et al. 2004; Queiroz et al. 2009; Rezk et al. 2006), and is caused by the orthostatic stress produced by the sitting position (Gotshall et al. 1994). This position might decrease venous return (reflected in the decreased in SV), which deactivates the cardiopulmonary baroreflex, increasing SVR, and DBP (Mark and Mancia 1996).

The decreases in CO observed after the three exercise sessions were due to a decrease in SV which might be related to an increase in post-load, a decrease in cardiac contractility, and/or a reduction in pre-load. The increase in post-load is not probable since SVR did not increase or increase similarly after the exercises and the C sessions. A decrease in cardiac contractility is not feasible because it has been observed in healthy subjects only after strenuous exercise that was not the case in the present study (Niemela et al. 1984). Thus, the most probable mechanism for the

reduction in the SV is a decrease in preload, since central volume depletion has already been observed after exercise (Hanel et al. 1997). Some mechanisms have been proposed for this response. A decrease in plasma volume has been reported after exercise (Charkoudian et al. 2003), and may explain the decrease in venous return at the beginning of the recovery, but plasma volume seems to be quickly restored to the pre-exercise values after the end of the effort (Pricher et al. 2004). A decrease in venous compliance has also been reported after exercise and might explain the decrease on venous return for a longer time (Dujic et al. 2006). Unfortunately, these mechanisms were not addressed in the present study and should be investigated in the future.

Despite the fact that SV decreased more after the R, A, and AR sessions than after the C session, suggesting that venous return also decreased more after the exercises, the increases in SVR were similar (R and AR sessions) or even lower (A session) after the exercises than after the C session. This response suggests that previous exercise blunted the cardiopulmonary reflex (Bennett et al. 1984), decreasing sympathetic drive to the muscle (Bisquolo et al. 2005) or acting direct on the peripheral vessels, decreasing their responsiveness to sympathetic activation (Halliwill et al. 1996) or increasing the endothelial function (Jungersten et al. 1997; Patil et al. 1993). These mechanisms were out of the scope of the present study, and might be investigated in the future. Nevertheless, the present results support the notion that post-exercise hypotension was mainly triggered by the incapacity of SVR to increase enough in order to compensate for the decrease in CO, and these mechanisms were more evident after aerobic stimuli. It is important to point out that the hemodynamic mechanisms observed in the present study were evaluated while the subjects were in the sitting position, which might have facilitated a decreased venous return, SV and CO. In fact, when recovery was conducted in the supine position after aerobic exercise, a decrease in SVR instead of a decrease in CO has been consistently reported (Halliwill et al. 1996, 2000; Pescatello et al. 2004a).

HR persisted elevated for many minutes after all the exercise sessions. The maintenance of post-exercise tachycardia was accompanied by a persistent decrease in absolute values of LF and HF, as well as a decrease in normalized HF_{R-R} and an increase in normalized LF_{R-R} and LF/HF , which supports an increase in sympathetic and a decrease in vagal modulation to the heart (Task 1996). Similar results have been reported previously with spectral analysis (Mourot et al. 2004; Parekh and Lee 2005; Rezk et al. 2006), and have been confirmed with drug blockade studies (Heffernan et al. 2006; Savin et al. 1982). The similar HR and autonomic responses after the A and R sessions have been previously observed

(Heffernan et al. 2006), and shows that the time course for sympathetic and parasympathetic return to pre-exercise levels did not differ between exercise modes. Nevertheless, when aerobic and resistance exercises were combined, HR, HF_{R-R} , and LF_{R-R} changes were greater and lasted longer. This response might be, at least in part, attributed to the longer time of exercise performed in the AR session, and it might be related to the greater fall in SV, leading to a greater deactivation of cardiopulmonary receptors, increasing the sympathetic activation to the heart (Furlan et al. 2001).

Post-exercise hypotension reflects a decrease in cardiovascular load (Pescatello et al. 2004a), while increases in cardiac sympathetic activity and heart rate reflects an increases in it (Cole et al. 2000). Thus, in the present study, all exercises promoted a paradoxical effect on cardiovascular risk after their execution. By considering the rate pressure product, which equalizes both aspects, it was observed that aerobic exercise, when performed alone, resulted in lower risk and even offered some protection, since cardiovascular load persisted at elevated levels for only a few minutes and then decreased below to pre-exercise levels, similarly to the C session. On the other hand, the association of aerobic and resistance exercises resulted in a higher and longer increase in cardiovascular load, probably due to the longer duration of this session. Although these results were obtained from healthy subjects, they might be similar in subjects with cardiovascular disease, which should be tested in the future. Assuming this similarity, the execution of aerobic and resistance exercises in different sessions might be considered in order to decrease cardiovascular load in subjects with high cardiovascular risk.

A possible concern about this study is the higher exercise volume in the AR session than in the R and A sessions, which might have influenced the results. Nevertheless, this is the kind of concurrent training that is recommended and performed for health maintenance (30 min of aerobic followed by 30 min of resistance exercise). Moreover, as post-exercise measurements were performed at many moments during the recovery, it was possible to compare similar times after the aerobic stimulus in the A and AR sessions, which permits some evaluation of the possible effect of greater exercise volume, as discussed previously for each variable. In this study, both genders were included in the sample. As men and women might present different hemodynamic and neural responses in certain situations, gender was included as a covariate in all the analyses. During hemodynamic assessment, BP was measured immediately before CO determination. This difference in the time of measurements might not have influenced the results because CO rebreathing technique did not influence BP (Turner et al. 1996). Finally, the mean BP was slight

different between the sessions in the pre-intervention period, however, this difference might not have influenced the results since it was within the measurement error.

Conclusion

A single session of aerobic, resistance and concurrent exercise promoted post-exercise hypotension due to a decrease in CO, caused by a reduction in SV, which was not completely compensated for an increase in SVR. Post-exercise hypotension was accompanied by HR and cardiac sympathetic modulation increases. A concurrent exercise session did not potentiate post-exercise hypotension, and it increased and prolonged post-exercise cardiac sympathetic activation and tachycardia, which reflects a greater and longer increase in cardiovascular load during the recovery period.

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