Postexercise hypotension and hemodynamics: the role of exercise intensity

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Postexercise hypotension and hemodynamics: the role of exercise intensity

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Aim. Although postexercise hypotension (PEH) has already been extensively demonstrated, the influence of exercise intensity on its magnitude and mechanisms is still controversial.

Methods. Twenty-three normotensive subjects were submitted to a control (45 minutes of rest) and 3 exercise sessions (cycle ergometer, 45 minutes at 30%, 50% and 75% of VO_{peak}) to investigate the role of exercise intensity on PEH. Blood pressure (BP - auscultatory), heart rate (HR - ECG), and cardiac output (CO - CO_{2} rebreathing) were measured before and after the control and exercise sessions.

Results. Systolic BP decreased significantly after exercise at 50% and 75% of VO_{peak}. Diastolic BP increased significantly during the control session, did not change after exercise at 30% of VO_{peak}, and decreased significantly after exercise at 50% and 75% of VO_{peak}. This fall was greater and longer after more intense exercise. CO and systemic vascular resistance (SVR) responses were similar between sessions, CO increased whereas SVR decreased significantly. Stroke volume (SV) increased and heart rate (HR) decreased following control and exercise at 30% of VO_{peak}, whereas SV decreased and HR increased after exercise at 50% and 75% of VO_{peak}.

Conclusion. PEH is greater and longer after more intense exercise. BP profile is followed by a decrease in SVR and an increase in CO, what was not influenced by previous exercise. The increase in CO is caused by an increase in SV after rest and low intensity exercise and by an increase in HR after moderate and more intense aerobic exercise.

Key words: Exercise - Blood pressure - Cardiac output - Vascular resistance - Stroke volume - Heart rate.

Many studies have observed that a single bout of aerobic exercise produces a significant fall in blood pressure levels in normotensive subjects as well as in hypertensive subjects during the recovery period. In fact, BP levels remain below pre-exercise or control levels for many minutes or even hours after exercise. Although postexercise BP does not achieve really hypotensive levels, this phenomenon has been called postexercise hypotension.

Hemodynamic mechanisms involved in PEH are still controversial. Some authors observed a decrease in cardiac output (CO) after exercise whereas others showed a decrease in systemic vascular resistance (SVR). Moreover, a biphasic mechanism was identified in a study, showing a decrease in SVR during the first minutes of recovery, and a decrease in CO afterwards. Some factors, such as exercise intensity or population studied, may account for this controversy.

The effect of exercise intensity on postexercise BP
TABLE I.—Physical and functional characteristics of the volunteers.

<table>
<thead>
<tr>
<th>Characteristics</th>
<th>Values</th>
</tr>
</thead>
<tbody>
<tr>
<td>Men/women</td>
<td>8/15</td>
</tr>
<tr>
<td>Age (y)</td>
<td>24±1</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>61.3±2.8</td>
</tr>
<tr>
<td>Height (m)</td>
<td>1.66±0.02</td>
</tr>
<tr>
<td>Body mass index (kg/m²)</td>
<td>22.0±0.7</td>
</tr>
<tr>
<td>Systolic blood pressure (mmHg)</td>
<td>109±2</td>
</tr>
<tr>
<td>Mean blood pressure</td>
<td>84±2</td>
</tr>
<tr>
<td>Diastolic blood pressure (mmHg)</td>
<td>71±2</td>
</tr>
<tr>
<td>Heart rate (beats/min)</td>
<td>69±2</td>
</tr>
<tr>
<td>Peak oxygen uptake (ml·kg⁻¹·min⁻¹)</td>
<td>39.4±1.7</td>
</tr>
<tr>
<td>Peak workload (Watts)</td>
<td>157±8</td>
</tr>
</tbody>
</table>

is not clear. In normotensives, different exercise intensities produced either similar postexercise BP falls 5,8 or a greater decrease after more intense exercise.9,11 In addition, exercise intensity influenced postexercise heart rate (HR). We have previously verified 5 that low-intensity exercise decreased HR whereas moderate and high-intensity exercises increased it during the recovery period. As HR is a determinant of CO, exercise intensity can affect CO response and, consequently, BP profile after exercise. As far as we know, this issue has not been investigated yet.

Thus, this study was designed to evaluate the effect of different exercise intensities on PEH and its systemic hemodynamic mechanisms in young normotensive healthy subjects.

Materials and methods

Subjects

Twenty-three young normotensive subjects gave a written consent to take part in the current study, approved by the Ethics Committee of the Heart Institute (InCor), Medical School, University of São Paulo, Brazil. None of the subjects were participating in any regular physical activity program. Subjects were excluded in case they had any cardiovascular or metabolic diseases. Their physical and cardiovascular characteristics are shown in Table I.

Auscultatory BP was measured 3 times in 2 visits, using a mercury sphygmomanometer. Mean value was applied to determine BP and subjects were excluded if systolic or diastolic BP were ≥140 and 90 mmHg, respectively. Peak oxygen uptake (VO₂peak) was measured directly by a metabolic cart (MGC, CAD/NET 2001) during a graded maximal exercise test performed on a cycle ergometer with 30 W increments every 3 minutes until exhaustion.

Experimental protocol

All subjects were submitted, in a random order, to 4 experimental sessions (1 control and 3 exercise sessions), which were performed in the morning with an interval of at least 5 days between the sessions. Subjects were instructed to avoid physical activity during the preceding 48 hours.

In each session, an ambulatory BP device was placed on the subjects non-dominant arm and BP monitoring was started. Subjects rested in a comfortable armchair for 20 minutes (baseline). They moved to the cycle ergometer, where they remained for 45 minutes, resting in the control session or pedaling at 30%, 50% and 75% of VO₂peak in the exercise sessions. Afterwards, subjects returned to the armchair and stayed in a sitting position for 90 minutes (postintervention period). Subjects were instructed to return the ambulatory monitor the next morning.

During the experimental sessions, auscultatory BP, CO and HR were measured at 10 minutes of baseline and after 15, 30, 60 and 90 minutes of interventions. At each moment, measurements were repeated twice and a mean value was calculated.

Measurements

Auscultatory BP was measured immediately before CO determination. The same observer performed BP measurements for each subject in all experimental sessions. Mean blood pressure was calculated by the formula: Mean BP = Diastolic BP+1/3 (Pulse BP).

ECG was continuously monitored (TEB, SM300) during all the experiments and HR was registered immediately after BP measurement.

CO was estimated by the indirect Fick method of CO₂ rebreathing,25,26 using a metabolic cart (MGC, CAD/NET 2001). Briefly, subjects breathed spontaneously until a steady CO₂ production was achieved. This procedure was followed by the rebreathing of a mixed gas with a high CO₂ concentration (8% to 12%) until 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 stroke volume (SV) by the quotient between CO and HR.

Twenty-four-hour BP was measured by an oscillo-
metric BP device (SpaceLabs, 90207) programmed to take measurements every 10 minutes for 24 hours. It was periodically calibrated against a mercury column. Data were only accepted if at least 75% of the measurements were taken successfully. Subjects were instructed to keep similar patterns of sleep, activities and nutrition on all experimental days, what was checked by a daily report made by each subject.

**Data analysis**

To analyze the hemodynamic response after the control and exercise sessions, differences between post-intervention values and baseline were calculated. Two-way analysis of variance ANOVA for repeated measurements (BMDP Statistical Software, 1985, University of California, Los Angeles, CA) was employed. Session (control, 30% of VO\textsubscript{2peak}, 50% of VO\textsubscript{2peak}, 75% of VO\textsubscript{2peak}) and period (baseline and 15, 30, 60 and 90 minutes after interventions) were set as main factors. For ambulatory data, only measures taken after interventions were analyzed. The averages of 24-hour (all measures taken), daytime (all measures taken during the period subjects reported to be awake), and nighttime (all measures taken during the period subjects reported to be asleep) BP and HR, as well as daytime and nighttime BP loads (percentage of BP measurements ≥140/90 and 120/80 mmHg for daytime and nighttime periods, respectively) were calculated for each experimental session and were compared by one-way analysis of variance for repeated measurements. Newman-Keuls test was applied as posthoc. A 2-tailed analysis was employed. P<0.05 was accepted as statistically significant. Data are presented as mean±SE.

**Results**

**Exercise intensity**

During the exercise sessions, subjects exercised at 33.6±1.3%, 51.4±1.6%, and 75.1±2.6% of VO\textsubscript{2peak}. To achieve these levels, workloads were set at 33±3, 68±4, and 104±6 watts, which corresponded to an oxygen uptake of 12.9±0.3, 19.9±0.7, and 29.0±1.0 ml·kg\textsuperscript{-1}·min\textsuperscript{-1}.

**Postexercise hemodynamic responses**

Systolic, diastolic, and mean BP responses during the experimental sessions are shown in Figure 1. Systolic BP did not change during the control and exercise at 30% of VO\textsubscript{2peak} sessions. After exercise at 50% of VO\textsubscript{2peak}, systolic BP decreased in relation to baseline at 30, 60 and 90 minutes (-4.7±1.4, -7.3±1.4 and -4.8±1.4 mmHg, respectively, p<0.05). BP fall at 60 minutes of recovery was greater than the one found in the control session. After exercise at 75% of VO\textsubscript{2peak}, systolic BP also decreased according to baseline at 30, 60 and 90 min of recovery (-7.6±1.5, -9.2±1.1 and -8.4±1.6 mmHg, p<0.05). BP decreases at these moments were significantly greater than in the control and 30% VO\textsubscript{2peak} sessions. Moreover, BP fall at 90 minutes of recovery was also significantly greater than after exercise at 50% VO\textsubscript{2peak}.

Diastolic BP increased in relation to baseline during all the postintervention period in the control session (15 min=+3.3±1.0, 30 min=+3.4±1.0, 60 min=+3.2±1.0, and 90 min=+3.0±1.0 mmHg, p<0.05). After exercise at 30% of VO\textsubscript{2peak}, diastolic BP did not change, and this response was significantly different from that observed in the control session at 15, 30 and 60 min of recovery. After exercise at 50% of VO\textsubscript{2peak}, diastolic BP decreased in relation to baseline at 30 minutes (-3.6±1.3 mmHg, p<0.05), whereas after exercise at 75% of VO\textsubscript{2peak} it decreased at 30 and 60 minutes of recovery (-3.5±1.3 and -4.4±1.2 mmHg, respectively, p<0.05). After these 2 exercise intensities, diastolic BP responses were significantly different from the control session during all the recovery periods, and were also lower than in the 30% of VO\textsubscript{2peak} session at 30 and 90 minutes of recovery. Moreover, diastolic BP response at 60 minutes of recovery was also different after exercise at 30% and 75% of VO\textsubscript{2peak}.

Mean BP did not change in relation to baseline after control and exercise at 30% of VO\textsubscript{2peak}, but this response was significantly lower than in the control session at 15 and 60 minutes after exercise at 30% of VO\textsubscript{2peak}. After exercise at 50% of VO\textsubscript{2peak}, mean BP decreased in relation to baseline at 30, 60 and 90 minutes (-4.0±1.2, -4.0±1.1 and -3.2±1.3 mmHg, respectively, p<0.05), and after exercise at 75% of VO\textsubscript{2peak}, it decreased according to baseline during all the recovery periods (15 min=-3.2±1.1, 30 min=-4.9±1.2, 60 min=-6.0±1.0, and 90 min=-4.3±1.2 mmHg, p<0.05). These falls were significantly greater than in the control session during all recovery periods, and were also greater than in the 30% of VO\textsubscript{2peak} session at 30 and 90 minutes after exercise at 50% of VO\textsubscript{2peak} and at 30, 60 and 90 minutes after exercise at 75% of VO\textsubscript{2peak}.

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SVR and CO responses during the experimental sessions are shown in Figure 2. SVR and CO behavior did not differ between sessions. At 60 and 90 min after interventions, SVR decreased (marginal value = -2.0±0.7, and -1.7±0.7 mmHg·l⁻¹·min⁻¹, p<0.05) and CO increased in relation to baseline (marginal value=+0.4±0.1 and +0.3±0.1 min, p<0.05).

SVR responses during the experimental sessions are shown in Figure 3. SV did not change in the control session, but it increased according to baseline at 60 minutes after exercise at 30% of VO₂peak (+13.0±3.4 ml/beat, p<0.05). SV did not change after exercise at 50% of VO₂peak, but this response was significantly different from the control session at 15 minutes of recovery, and from the exercise session at 30% of VO₂peak at 15, 60, and 90 minutes. SV decreased in relation to baseline 15 minutes after exercise at 75% of VO₂peak (-14.8±3.5 ml/beat, p<0.05). During all postintervention periods, SV response was different from the control and exercise at 30% of VO₂peak sessions, and at 15 minutes of recovery, it was also significantly different from the exercise at 50% of VO₂peak session.

HR responses during the experimental sessions are shown in Figure 3. In the control session, HR decreased according to baseline at 30, 60 and 90 minutes after intervention (-5.0±1.3, -5.1±1.2, and -6.1±1.6 beats/min, respectively, p<0.05). Sixty minutes after exercise at 30% of VO₂peak, HR also decreased in relation to baseline (-4.1±1.3 beats/min, p<0.05). Moreover, at 30 minutes of recovery, HR response was significantly different from the one observed in the control session. After exercise at 50% of VO₂peak, HR increased in relation to baseline at 15 and 30 minutes of recovery (+7.1±1.7 and +4.5±1.7 beats/min, respectively, p<0.05). Besides, during all the recovery periods after exercise at 50% of VO₂peak, HR response differed significantly from the control.
session, and it also differed from the exercise at 30% of $\text{VO}_2\text{peak}$ session at 15, 30 and 60 minutes of recovery. After exercise at 75% of $\text{VO}_2\text{peak}$, HR increased according to baseline during all recovery periods (15 min=+23.1±2.7, 30 min=+15.6±2.5, 60 min=+10.7±2.3, and 90 min=+6.2±2.5 beats/min, p<0.05). Moreover, at all moments, this response was significantly different from all the other experimental sessions.

Hourly BP averages measured after exercise or rest in all sessions are shown in Figure 4, while average values for 24-hour, daytime and nighttime periods are presented in Table II. Twenty-four-hour, daytime and nighttime systolic, mean and diastolic BPs and BP loads did not differ between the experimental sessions. However, 24-hour HR was significantly greater after exercise at 75% of $\text{VO}_2\text{peak}$ than in the other sessions. Daytime HR was significantly greater after exercise at 30%, 50% and 75% of $\text{VO}_2\text{peak}$ than in the control session. And daytime HR was significantly higher after exercise at 75% of $\text{VO}_2\text{peak}$ than after exercise at 30% and 50% of $\text{VO}_2\text{peak}$.
Discussion and conclusions

The main findings of the present investigation are that in healthy normotensive subjects: 1) PEH is affected by exercise intensity, occurring mainly after exercise sessions from 50% to 75% of VO_{2peak} and being greater and longer after more intense aerobic exercise; 2) postintervention BP profile is followed by a decrease in SVR and by an increase in CO, what is not influenced by previous exercise; 3) CO increase observed in all sessions is due to different hemodynamic mechanisms: after rest and low intensity exercise, it is caused by an increase in SV while after more intense exercise, it is due to an increase in HR.

In normotensive subjects, exercise from 30% to 75% of VO_{2peak} had hypotensive effects during the recovery period. After exercise at 30% of VO_{2peak}, hypotensive effect was characterized by the absence...
of diastolic BP increase observed during the control session. In the other sessions (50% and 75% of \( V_{O2\text{peak}} \)), hypotensive effect was evident by significant systolic and diastolic BP reductions in comparison to pre-exercise and control values. This post-exercise hypotension was greater after more intense exercise, which was evident by the greater systolic BP fall observed 90 minutes after exercise at 75% when compared to exercise at 50% of \( V_{O2\text{peak}} \). Besides, PEH duration was also greater after more intense exercise, since diastolic BP reduction according to baseline lasted for up to 30 minutes of recovery after exercise at 50% of \( V_{O2\text{peak}} \), and for up to 60 minutes of recovery after exercise at 75% of \( V_{O2\text{peak}} \).

Although not unanimous, postexercise BP fall has already been reported in normotensive subjects. However, the effect of exercise intensity is still controversial, since some authors, like the present results, observed a greater hypotension after more intense exercise and others did not. This controversy may be due to sample size, which was greater in the present study, or to the measurement of other hemodynamic variables, such as CO determination that can elicit some stress and BP increase. As previous exercise reduces BP response to stress, this blunted response can be increased by more intense exercise, resulting in a greater PEH.

After all sessions, BP profile was followed by a SVR decrease and a CO increase. As these responses after the exercise sessions did not differ from those of the control session, they cannot be attributed to previous exercise. Thus, the question about the systemic hemodynamic mechanism responsible for PEH is still unanswered. In fact, analyzing the individual data corrected by the changes in the control session, it can be seen that, in all the sessions, approximately 50% of the subjects showed a decrease in CO while the other 50% showed a reduction in SVR (Figure 5). These results suggest that other factors, not only exercise intensity, in opposition to our previous hypothesis, may be responsible for systemic hemodynamic mechanism that determines BP fall after exercise. These factors should be addressed by future studies.

Although postintervention CO increased similarly in all sessions, SV and HR profiles were different. After the control and mild exercise sessions, there was an increase in SV and a decrease in HR, whereas after moderate and intense exercises, there was an increase in HR and a decrease in SV. The decrease in SV after exercise has already been shown, and could be attributed to an increase in cardiac after-load and/or to a decrease in cardiac contractility and/or preload. An increase in after-load seems not to explain the present results, since SVR was lower after all sessions. A decrease
in ventricular function, due to a decrease in cardiac contractility, was observed after strenuous, but not after mild exercise. A reduction in preload owing to a decrease in plasma volume may be involved. In fact, Rondon et al. observed, in older subjects, a reduction in end-diastolic volume after exercise at 50% of VO\textsubscript{2peak}. Moreover, Hayes et al. demonstrated, in young subjects, that plasma volume was decreased (-14%) at the end of an exercise bout at 65% of VO\textsubscript{2peak}, and Gillen et al. and Yang et al. verified a greater reduction (-15% and -18%, respectively) after exercise at 85% of VO\textsubscript{2peak}. As the present study did not evaluate plasma volume neither cardiac function, these mechanisms need to be further investigated. HR responses after exercise were also related to exercise intensity in previous studies. It is known that reflexes triggered by a drop in BP increase HR. Thus, the lower BP observed after more intense exercise could result in greater increase in HR due to higher arterial baroreflex deactivation.

In the current study, although exercise reduced BP levels for 90 min under laboratorial conditions, this PEH was not observed for the next 24 hours. In fact, studies investigating ambulatory BP responses after exercise have shown different results. In hypertensives, many authors have observed a significant increase in ambulatory BP after exercise, while others have not. In normotensives, most of the studies found no change in ambulatory BP levels after exercise, similarly to the present data. However, in a previous study, we observed a significant 24-hour BP fall after exercise at 50% of VO\textsubscript{2peak}, which was greater in normotensive subjects with higher BP levels. In the present study, just one of the subjects showed high normal BP, which could explain the difference between studies.

In conclusion, a single bout of exercise from 30% to 75% of VO\textsubscript{2peak} has hypertensive effects in young normotensive subjects. However, postexercise hypotension is more evident after exercise bouts from 50% to 75% of VO\textsubscript{2peak}, being greater and longer after more intense aerobic exercise. In this population, the systemic hemodynamic mechanism responsible for BP fall after exercise may vary from one subject to another, and it is not determined by exercise intensity. Although CO behavior does not change after different exercise intensities; there is a decrease in HR and an increase in SV after low intensity exercise, and a decrease in SV and an increase in HR after moderate and more intense aerobic exercise.

**Limitations**

Results in the present study are limited to young healthy normotensive subjects. Moreover, they are limited to the 3 exercise intensities studied, which corresponded from low to moderate aerobic exercise, and to recovery in the seated position.

CO\textsubscript{2} rebreathing technique presents some variability. To reduce this effect, each measurement was repeated twice and a mean value was calculated. Therefore, CO recorded at baseline in all sessions did not differ significantly and presented a coefficient of variation of 14±2%. This method was chosen because it is non-invasive and has already been used by others.

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**References**

12. Boone JB, Probst MM, Rogers MW, Berger R. Postexercise hypo-