

Post-exercise changes in blood pressure, heart rate and rate pressure product at different exercise intensities in normotensive humans

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Abstract

To evaluate the effect of exercise intensity on post-exercise cardiovascular responses, 12 young normotensive subjects performed in a randomized order three cycle ergometer exercise bouts of 45 min at 30, 50 and 80% of VO_2peak , and 12 subjects rested for 45 min in a non-exercise control trial. Blood pressure (BP) and heart rate (HR) were measured for 20 min prior to exercise (baseline) and at intervals of 5 to 30 (R5-30), 35 to 60 (R35-60) and 65 to 90 (R65-90) min after exercise. Systolic, mean, and diastolic BP after exercise were significantly lower than baseline, and there was no difference between the three exercise intensities. After exercise at 30% of VO_2peak , HR was significantly decreased at R35-60 and R65-90. In contrast, after exercise at 50 and 80% of VO_2peak , HR was significantly increased at R5-30 and R35-60, respectively. Exercise at 30% of VO_2peak significantly decreased rate pressure (RP) product ($\text{RP} = \text{HR} \times \text{systolic BP}$) during the entire recovery period (baseline = 7930 ± 314 vs R5-30 = 7150 ± 326 , R35-60 = 6794 ± 349 , and R65-90 = 6628 ± 311 , $P < 0.05$), while exercise at 50% of VO_2peak caused no change, and exercise at 80% of VO_2peak produced a significant increase at R5-30 (7468 ± 267 vs 9818 ± 366 , $P < 0.05$) and no change at R35-60 or R65-90. Cardiovascular responses were not altered during the control trial. In conclusion, varying exercise intensity from 30 to 80% of VO_2peak in young normotensive humans did not influence the magnitude of post-exercise hypotension. However, in contrast to exercise at 50 and 80% of VO_2peak , exercise at 30% of VO_2peak decreased post-exercise HR and RP.

Key words

- Exercise intensity
- Blood pressure
- Heart rate
- Rate pressure product

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Introduction

Although an acute bout of submaximal exercise can cause important cardiovascular changes, there are few and controversial published data on the effect of different exercise intensities on blood pressure, heart rate and rate pressure product (an index of myocardial oxygen consumption). An increased understanding of this issue has clinical implications, in addition to being important in prescribing exercise intensities for humans.

Most studies dealing with post-exercise blood pressure responses have demonstrated that exercise reduces blood pressure during the recovery period (1-19). Nevertheless, the magnitude and the time course of blood pressure changes after exercise are inconsistent. In normotensives, the magnitude ranges from no change (4,8,14,15,20) to falls as great as 30 mmHg (19). In hypertensive humans, Somers et al. (18) observed that blood pressure levels are significantly lower than pre-exercise levels only during the first hour of recovery, while Pescatello et al. (14) found a significant blood pressure fall up to 12 h after exercise. In relation to post-exercise heart rate responses, the results are also controversial and less conclusive. Some investigators (2,4,5,7,10-12,15,19) have observed an increase in heart rate levels during the recovery period. In contrast, others have reported no change (1,3,6,8,12,14,20) or even decreases (9,13) in post-exercise heart rate responses. The experimental protocol, the initial levels of blood pressure, and the type, duration and intensity of exercise may explain, in part, these discrepancies in post-exercise blood pressure and heart rate responses (21).

It has been well established that the magnitude of neural and hemodynamic responses during exercise is directly related to exercise intensity (22,23). Thus, it is possible that different exercise intensities have also distinct effects on cardiovascular changes after

exercise. Hagberg et al. (9), in a study of older hypertensive men, observed that hypotension after exercise performed at 70% of maximal oxygen consumption ($VO_2\text{max}$) was greater and lasted longer than after exercise performed at 50% of $VO_2\text{max}$. Piepoli et al. (15) reported that maximal exercise provoked an increase, while low to moderate exercise caused no change in post-exercise heart rate levels in normotensive subjects.

Because blood pressure and heart rate responses during the recovery period can be influenced by exercise intensity, it is possible that different intensities of exercise may also have distinct effects on post-exercise rate pressure product. Therefore, the goal of the present investigation was to study the effect of different exercise intensities on post-exercise blood pressure, heart rate and rate pressure product in humans.

Material and Methods

Subjects

Twelve healthy young normotensive subjects (4 women and 8 men; exercised subjects) were studied in the exercise trials, and twelve subjects (7 women and 5 men; rested subjects) with similar physical and cardiovascular characteristics were studied in the non-exercise control trial (Table 1). Their healthy condition was confirmed by physical examination and a maximal exercise test. None of the subjects engaged in any regular physical activity program. All procedures were approved by the General Hospital Ethics Committee, University of São Paulo, and all subjects gave informed written consent to participate in the study.

Blood pressure measurements

After a 5-min rest in the sitting position, blood pressure was measured three times during two different visits to the laboratory.

On the occasion of each visit, blood pressure was measured by the same experienced observer using a standard mercury sphygmomanometer, taking the first and the fifth phases of Korotkoff sounds as systolic and diastolic values, respectively. Subjects were excluded if the average of the last two values obtained during each visit for systolic and diastolic blood pressures was greater than 139 and 89 mmHg, respectively.

Maximal exercise test

At the beginning of the study, exercised subjects underwent a maximal cycle ergometer exercise test, with 30 W increments every 3 min until exhaustion. Oxygen consumption and carbon dioxide were measured on a breath by breath basis, using a metabolic cart (Medical Graphics Corporation, CAD/NET 2001, St. Paul, MN), and peak oxygen consumption ($\text{VO}_{2\text{peak}}$) was recorded as the highest value of oxygen consumption achieved during the test, calculated during an average 30-s interval.

Study protocol

Exercise protocols. Twelve subjects were submitted in a randomized order to three exercise trials performed at 30, 50 or 70% of $\text{VO}_{2\text{peak}}$ with an interval of at least 5 days. Each of the six possible combinations of exercise intensities was performed by two subjects. Exercise trials were conducted at the same time of day. Subjects were instructed not to exercise 48 h prior to the exercise trials, and to maintain similar activities and meal patterns.

For each exercise trial, subjects arrived at the laboratory between 1:00 and 3:00 p.m. Baseline blood pressure was measured every two and a half minutes in the sitting position for 20 min. After this resting period, volunteers sat on a cycle ergometer (Mijnhardt, St. Paul, MN) and began the exercise, which consisted of 3 min of warm-

up, 45 min of exercise at a workload to result in an intensity equivalent to 30, 50 or 70% of $\text{VO}_{2\text{peak}}$, and 2 min of active recovery. After exercise, subjects rested in the sitting position for 90 min. During exercise and throughout the recovery period, blood pressure was measured every 5 min. Blood pressure was recorded by the same observer in all three exercise trials, using a standard mercury sphygmomanometer. Heart rate was monitored by electrocardiography (ECG-TEB, SM300, São Paulo, SP) throughout the experimental protocol (resting to recovery), and was recorded simultaneously to blood pressure measurements. Baseline blood pressure and heart rate levels (average of 6 measurements after eliminating the first and last measurements of this period) were compared to the post-exercise levels, which were calculated for three recovery periods as the average of all measurements performed between 5 and 30 (R5-30), 35 and 60 (R35-60), and 65 and 90 (R65-90) min of recovery. The rate pressure product was determined as the product of heart rate and systolic blood pressure.

Exercise workloads were calculated based on oxygen consumption and workload during the maximal exercise test, using a linear regression equation. This procedure permitted us to calculate the workload corresponding to 30, 50, and 70% of $\text{VO}_{2\text{peak}}$. To verify the accuracy of exercise intensity, oxygen

Table 1 - Physical and cardiovascular characteristics of the subjects studied.

BMI = Body mass index; SBP = systolic blood pressure; DBP = diastolic blood pressure; $\text{VO}_{2\text{peak}}$ = peak oxygen consumption.

Variable	Exercised subjects	Rested subjects
Age (years)	22 ± 0.7	22 ± 1.1
Weight (kg)	65.1 ± 2.1	66.6 ± 2.7
Height (cm)	171 ± 2.0	171 ± 2.0
BMI (kg/m^2)	22.3 ± 0.5	22.9 ± 1.0
Resting SBP (mmHg)	104 ± 2.4	109 ± 2.3
Resting DBP (mmHg)	71 ± 2.9	68 ± 1.9
$\text{VO}_{2\text{peak}}$ ($\text{ml kg}^{-1} \text{min}^{-1}$)	38.1 ± 2.2	-

consumption was measured in all subjects at 15, 30 and 45 min of exercise (Medical Graphics Corporation, CAD/NET 2001) during the exercise trials.

Resting protocol. To determine any potential diurnal variations in blood pressure, 12 subjects performed a non-exercise control trial. During this trial, the subjects were submitted to the same experimental protocol as applied to the exercise trials, but instead of pedaling they rested in the sitting position for 45 min. Blood pressure and heart rate were measured by the same procedures and at the same times as during the exercise trials.

Statistical analysis

Baseline levels in different exercise trials were analyzed by one-way analysis of variance for repeated measures. Blood pressure, heart rate and rate pressure product responses after exercise were evaluated by two-way analysis of variance (ANOVA) for repeated measures (BMDP, 1985, University of California, Los Angeles, CA), establishing exercise intensity (30, 50 and 70% of VO_2peak) and recovery stages (baseline, R5-30, R35-60 and R65-90) as main factors. During the resting protocol, heart rate, blood pressure and rate pressure product were analyzed by one-way analysis of variance for repeated measures. When significance was found, Scheffé's test was employed. $P < 0.05$ was accepted as being statistically significant. Data are reported as mean \pm SEM.

Results

Effect of exercise on cardiovascular responses

Exercise intensity. Oxygen consumption measured during exercise trials at 30 and 50% of VO_2peak corresponded to 32.8 ± 1.5 and $53.9 \pm 1.6\%$ of VO_2peak , respectively,

while oxygen consumption during the exercise trial at 70% of VO_2peak corresponded to $80.1 \pm 2.1\%$ of VO_2peak . Actually, these observations show that the exercise intensities studied were 30, 50 and 80% of VO_2peak .

As expected, heart rate, rate pressure product, and systolic and mean blood pressures increased significantly during exercise and the increases were greater with higher exercise intensities. Diastolic blood pressure did not change during exercise of any intensity (Table 2).

Blood pressure. Systolic, mean and diastolic blood pressure values are shown in Figure 1. Baseline systolic, mean and diastolic blood pressures were similar in all three exercise trials, and systolic, mean, and diastolic blood pressure responses were similar during the recovery period of exercise performed at 30, 50 and 80% of VO_2peak . Thus, systolic (marginal values - baseline = 107.4 ± 3.1 vs R5-30 = 103.6 ± 3.4 , R35-60 = 101.0 ± 3.5 and R65-90 = 102.5 ± 3.4 mmHg, $P < 0.05$), mean (marginal values - baseline = 85.6 ± 3.0 vs R5-30 = 82.2 ± 3.0 , R35-60 = 80.8 ± 3.2 and R65-90 = 82.2 ± 3.1 mmHg, $P < 0.05$) and diastolic (marginal values - baseline = 74.7 ± 3.1 vs R5-30 = 71.6 ± 3.0 , R35-60 = 70.8 ± 3.1 and R65-90 = 72.2 ± 3.0 mmHg, $P < 0.05$) blood pressure levels were significantly lower than baseline throughout the recovery period.

Heart rate. Heart rate values are shown in Figure 2. Baseline heart rate was similar in all three exercise trials. Exercise at 30% of VO_2peak provoked a significant decrease in heart rate at R35-60 (65.1 ± 2.3 bpm) and R65-90 (64.0 ± 2.0 bpm) when compared with baseline (72.5 ± 3.2 bpm). On the other hand, exercise at 50% of VO_2peak elicited a significant increase in heart rate at R5-30 (75.9 ± 2.4 bpm), but not at R35-60 (68.1 ± 2.3 bpm) or R65-90 (66.8 ± 2.6 bpm) when compared with baseline (69.4 ± 2.0 bpm). After exercise at 80% of VO_2peak , heart rate responses at R5-30 (96.5 ± 2.1 bpm) and R35-60 (81.2 ± 2.5 bpm) were significantly

higher than baseline (71.6 ± 2.5 bpm), but not at R65-90 (75.8 ± 7.2 bpm) when they were similar to baseline.

Rate pressure product. Rate pressure product values are shown in Figure 3. Baseline rate pressure product was similar in all three exercise trials. Exercise at 30% of VO_{2peak} significantly decreased rate pressure product throughout the recovery period (baseline = 7930 ± 314 vs R5-30 = 7150 ± 326 , R35-60 = 6794 ± 349 , R65-90 = 6628 ± 311 , $P < 0.05$). Exercise at 50% of VO_{2peak} , however, caused no significant change in post-exercise rate pressure product (baseline = 7459 ± 318 vs R5-30 = 7865 ± 299 , R35-60 = 6873 ± 323 , R65-90 = 6878 ± 377). Exercise at 80% of VO_{2peak} provoked a significant increase in rate pressure product at R5-30 (baseline = 7468 ± 267 vs R5-30 = 9818 ± 366 , $P < 0.05$), and no change at R35-60 (7931 ± 364) and R65-90 (7664 ± 322).

Effect of resting on cardiovascular responses

Blood pressure. During resting trial, systolic (baseline = 107.7 ± 2.4 vs R5-30 = 106.2 ± 3.1 , R35-60 = 105.9 ± 2.5 and R65-90 = 106.0 ± 2.2 mmHg) and mean (baseline = 80.8 ± 1.9 vs R5-30 = 81.2 ± 1.9 , R35-60 = 81.3 ± 1.9 and R65-90 = 82.3 ± 1.6 mmHg) blood pressures did not change significantly, while diastolic blood pressure was signifi-

cantly higher than baseline (67.2 ± 2.0 mmHg) at R65-90 (70.3 ± 2.1 mmHg, $P < 0.05$).

Heart rate. During the resting trial, heart rate did not change (baseline = 65.2 ± 2.3 vs R5-30 = 64.5 ± 2.4 , R35-60 = 63.7 ± 2.5 and R65-90 = 64.3 ± 2.8 bpm).

Rate pressure product. Rate pressure product was also unchanged throughout the non-exercise control trial (baseline = 7027 ± 303 vs R5-30 = 6832 ± 269 , R35-60 = 6748 ± 310 and R65-90 = 6823 ± 324).

Discussion

The main findings of the present study are: 1) exercise bouts performed at 30, 50 and 80% of VO_{2peak} provoked similar post-exercise hypotension in young normotensive humans; 2) exercise at 30% of VO_{2peak} , but not at 50 and 80% of VO_{2peak} , significantly decreased heart rate levels during the recovery period; 3) after exercise at 30% of VO_{2peak} , rate pressure product was lower than baseline levels, and 4) exercise at 50 and 80% of VO_{2peak} did not reduce post-exercise rate pressure product.

The present data confirm the results obtained in previous studies (1,5,7,8,10-12,15,16,18,19) showing that a single bout of steady-state exercise significantly decreases post-exercise blood pressure in nor-

Table 2 - Systolic (SBP), mean (MBP) and diastolic (DBP) blood pressures, heart rate (HR), and rate pressure product (RP) measured at baseline and during exercise at 30, 50 and 80% of VO_{2peak} .

* $P < 0.05$ significantly different from baseline. + $P < 0.05$ significantly different from other intensities (two-way analysis of variance for repeated measures and Scheffé's post-hoc test).

	30% VO_{2peak}		50% VO_{2peak}		80% VO_{2peak}	
	Baseline	Exercise	Baseline	Exercise	Baseline	Exercise
SBP	110 ± 3	124 ± 3**	107 ± 3	148 ± 5**	105 ± 3	167 ± 5**
MBP	87 ± 3	91 ± 3**	86 ± 3	97 ± 3**	84 ± 3	105 ± 3**
DBP	76 ± 3	75 ± 3	75 ± 3	71 ± 3	73 ± 3	74 ± 3
HR	73 ± 3	101 ± 4**	69 ± 2	130 ± 3**	72 ± 3	170 ± 3**
RP	7930 ± 314 ⁺	12409 ± 365**	7459 ± 318	19211 ± 550**	7468 ± 267	28354 ± 877**

motensive humans. The absence of a blood pressure fall during the non-exercise control trial shows that, in fact, the decreased blood pressure levels after exercise are due to the exercise effect and not to the normal diurnal blood pressure variations.

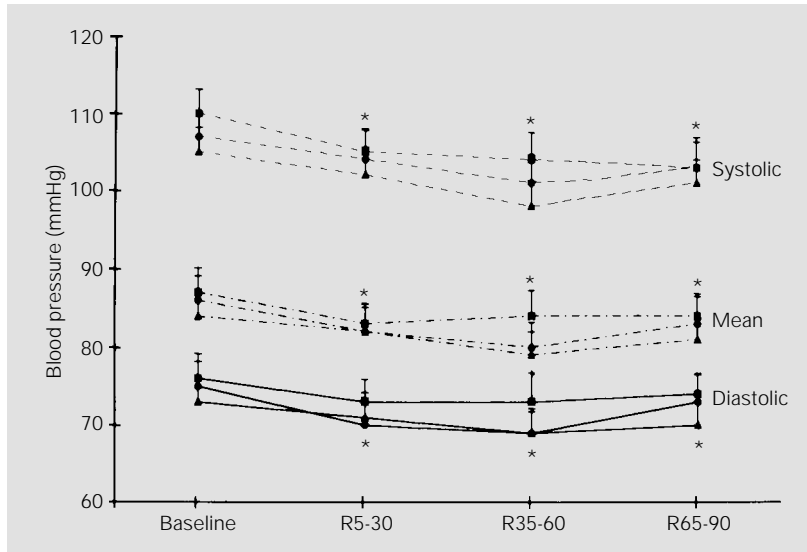


Figure 1 - Systolic, mean, and diastolic blood pressures at baseline and after exercise (R5-30, mean value between 5 and 30 min; R35-60, mean value between 35 and 60 min; R65-90, mean value between 65 and 90 min) performed at 30 (filled squares), 50 (filled circles) and 80% (filled triangles) of VO_2 peak. * $P < 0.05$ compared to baseline (two-way analysis of variance for repeated measures and Scheffé's post-hoc test).

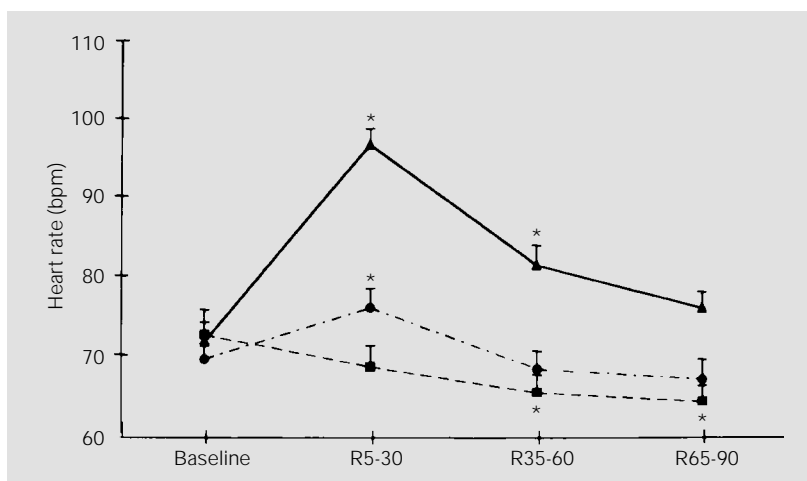


Figure 2 - Heart rate at baseline and after exercise (R5-30, mean value between 5 and 30 min; R35-60, mean value between 35 and 60 min; R65-90, mean value between 65 and 90 min) performed at 30 (filled squares), 50 (filled circles) and 80% (filled triangles) of VO_2 peak. * $P < 0.05$ compared to baseline (two-way analysis of variance for repeated measures and Scheffé's post-hoc test).

Since exercise intensity plays a role in hemodynamic, thermoregulatory and neural responses during exercise (22,23), we would expect that different exercise intensities would determine distinct post-exercise blood pressure responses. However, this was not the case in the present study, in which exercise performed at 30, 50 and 80% of VO_2 peak provoked similar hypotension during the recovery period. Pescatello et al. (14) did not observe any blood pressure fall after 30 min of exercise performed at 40% or 70% of VO_2 peak, and Piepoli et al. (15) observed that maximal exercise, but not submaximal steady-state exercise, provoked a post-exercise blood pressure fall in young normotensive humans. In older hypertensives, Hagberg et al. (9) reported that hypotension was greater after exercise at 70% of VO_2 peak than after exercise at 50% of VO_2 peak. The contrast between the present study and others (9,14,15) may be related to exercise duration or to the population investigated. In our study, normotensive humans were exercised for 45 min at all three intensities (30, 50 and 80% of VO_2 peak), while in the study of Pescatello et al. (14) normotensive humans were exercised for 30 min, and in the study of Piepoli et al. (15) exercise duration was shorter during maximal than submaximal bouts. In fact, Bennett et al. (1) reported that a 10-min exercise period did not induce a post-exercise blood pressure fall in normotensives, but additional exercise periods (4 bouts of 10 min) produced a significant decrease in blood pressure. Moreover, Overton et al. (13) observed that 40 min of exercise performed by spontaneously hypertensive rats provoked a greater and longer post-exercise hypotension than 20 min of exercise. Hagberg et al. (9) studied older hypertensive humans, while we investigated young normotensive humans. It is well understood that older subjects have vascular musculature alterations and decreased baroreceptor sensitivity (24) which may modify post-exercise hemodynamic responses.

Previous studies on young normotensive humans (1,4,5,7,8,10-12,14,15,18,19) have shown controversial results in regard to heart rate behavior during post-exercise period. The present results suggest that this controversy may be related to exercise intensity since we observed that low (30% VO_2peak), but not moderate (50% VO_2peak) intensity exercise provoked bradycardia during the recovery period. Moreover, high intensity exercise (80% VO_2peak) provoked a transient tachycardia after exercise. In fact, previous studies showed a tendency toward a positive relationship between exercise intensity and post-exercise heart rate levels.

The concept that the product of systolic blood pressure and heart rate (i.e., rate pressure product) is well correlated to myocardium oxygen consumption in young healthy subjects (25) as well as in cardiac patients (26) has been well established. Some investigators have observed that angina pectoris occurs at a constant value of rate pressure product (27), and that the degree of ST-segment depression is correlated with rate pressure product (28). Thus, the risk of cardiovascular problems increases with greater levels of rate pressure product. The present study demonstrated that an acute bout of low intensity exercise, besides producing lower increases in rate pressure product during exercise, also decreases post-exercise rate-pressure product below resting levels. Hence it reduces myocardial oxygen consumption and, consequently, the cardiovascular risks after exercise. In contrast, moderate and high intensity exercise bouts produce greater increases in rate pressure product during exercise and fail to reduce rate pressure product below baseline during the recovery period. These findings, if reproducible in cardiac patients, may have clinical implications, especially in relation to exercise prescription in cardiac rehabilitation programs.

The present study does not provide explanations about the mechanism by which low intensity exercise simultaneously de-

creases post-exercise blood pressure, heart rate and, consequently, rate pressure product in young normotensive subjects. Nevertheless, it is possible that low intensity exercise induces a reduction in sympathetic nerve activity to the heart and blood vessels during the recovery period. Chen et al. (3) observed a decrease in sympathetic tone to the heart after mild exercise in spontaneously hypertensive rats and Floras et al. (7) and Halliwill et al. (10) found a significant decrease in muscle sympathetic nerve activity after acute exercise in hypertensive and normotensive humans.

Despite the fact that both moderate and high intensity exercise decreased blood pressure, no reduction in rate pressure product was observed. Halliwill et al. (10) observed that after an acute bout of moderate exercise, baroreflex control of heart rate and blood vessels is distinctly regulated. In other words, sympathetic drive to the heart increases, while sympathetic drive to blood vessels decreases. Thus, it is possible that different exercise

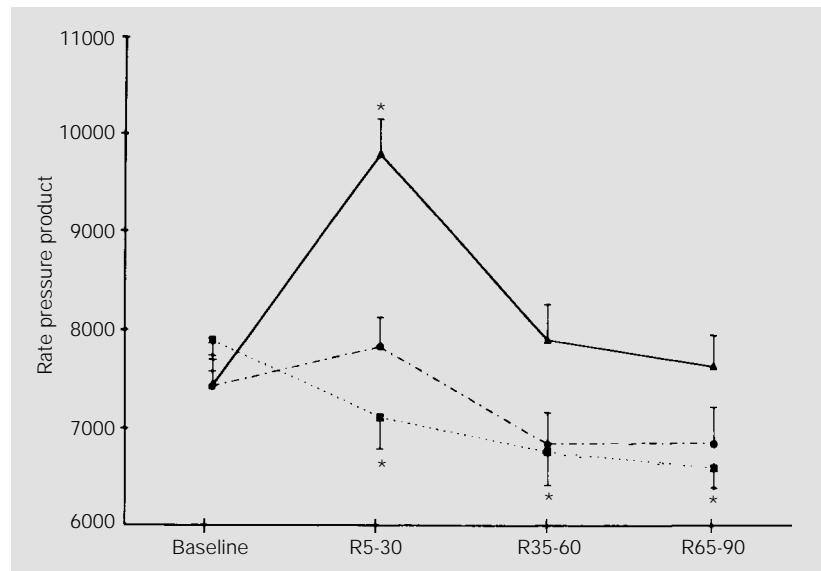


Figure 3 - Rate pressure product (heart rate x systolic blood pressure) at baseline and after exercise (R5-30, mean value between 5 and 30 min; R35-60, mean value between 35 and 60 min; R65-90, mean value between 65 and 90 min) performed at 30 (filled squares), 50 (filled circles) and 80% (filled triangles) of VO_2peak . * $P < 0.05$ compared to baseline (two-way analysis of variance for repeated measures and Scheffé's post-hoc test).

intensities may distinctly affect the baroreflex control of heart rate, but not the baroreflex control of blood vessels. Moreover, the vasodilatory response (15) and/or the decrease in blood volume (13) may play a role in the post-exercise blood pressure fall. It is well understood that muscle metabolites and heat accumulation are directly related to exercise intensity, and sweating rate is greater during more intense exercise (29). In addition, the increased local muscle metabolites (30,31) and/or heat production (8) are also potential stimuli for the increased heart rate responses after moderate and high intensity exercise.

Limitations. We studied the impact of acute exercise performed at 30, 50 and 80% of VO_2peak on cardiovascular responses up to 90 min of recovery in young healthy subjects. Thus, we do not know whether the decreased rate pressure product after low intensity exercise persists longer than 90 min or occurs in the presence of certain cardiovascular disturbances such as hypertension, myocardial ischemia or even heart

failure. Future studies, however, should address the effect of varying intensity of exercise on post-exercise cardiovascular responses in patients with cardiovascular disturbances.

We studied exercise at three different intensities (30, 50 and 80% of VO_2peak). It is possible that other intensities could have distinct effects on post-exercise cardiovascular responses.

In conclusion, in young normotensive humans, varying the intensity of exercise from 30 to 80% of VO_2peak did not influence the magnitude of post-exercise hypotension. However, in contrast to exercise at 50 and 80% of VO_2peak , exercise at 30% of VO_2peak significantly decreased rate pressure product during the recovery period.

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