

Effect of Exercise Intensity on Postexercise Hypotension

Christy L. Smelker, MS; Carl Foster, PhD; Margaret A. Maher, PhD;
Raymond Martinez, PhD; John P. Porcari, PhD

Both acute and habitual exercise may contribute to reduced blood pressure in patients with hypertension through a variety of mechanisms including effects on the sympathetic nervous system, insulin sensitivity, electrolyte balance, neural and baroreceptor reflex mechanisms, and vascular structure.¹ Evidence from both observational and experimental studies supports the value of physical activity of low to moderate intensity as an intervention for hypertension.²

Acute reductions in blood pressure during the hours after an exercise bout have been well described and referred to as postexercise hypotension (PEH).³⁻⁹ Although mechanistic studies are less than definitive, PEH is most likely attributable to a sustained reduction in vascular resistance. Findings have shown PEH to be greater after oral L-arginine administration, suggesting linkage to the nitric oxide-mediated vasodilation that occurs with exercise.¹⁰ However, findings also have demonstrated PEH to be independent of nitric oxide-mediated pathways.¹¹ Although PEH has been observed after dynamic exercise performed at intensities ranging from 40% and 70% of VO_2 peak,⁷ systematic studies reporting the effect of exercise intensity on PEH are lacking. To the degree that PEH is dependent on a nitric oxide-mediated mechanism, the magnitude of PEH would be expected to increase with exercise intensity. To the degree that PEH is dependent on non-nitric oxide-mediated pathways, one would expect PEH to be

independent of exercise intensity. Accordingly, the purpose of the current study was to evaluate the acute effects of exercise intensity relative to PEH in mildly hypertensive individuals.

METHODS

The subjects for this study were 10 volunteers (2 women and 8 men) from the University of Wisconsin—La Crosse community. Each participant had a history of hypertension, but was otherwise apparently healthy and clinically stable. All the subjects regularly performed moderate-intensity aerobic exercise, although none were highly trained. Six had stage 1 hypertension, treated with lifestyle management, and four who had stage 1 hypertension but had not responded to lifestyle changes were receiving medical therapy. None of the subjects had documented coronary artery disease, prior myocardial infarction, symptoms suggestive of angina pectoris, or a history of revascularization procedures.

From University of Wisconsin-La Crosse, La Crosse Wisconsin.

Address correspondence to: Carl Foster, PhD, Department of Exercise and Sport Science, 133 Mitchell Hall, University of Wisconsin-La Crosse, La Crosse, WI 54601 (e-mail foster.carl@uwlax.edu).

Table 1 • MEAN (\pm SD) CHARACTERISTICS OF THE SUBJECTS

Characteristic	Men (n = 8)	Women (n = 2)
Age, y	58 \pm 8	49 \pm 13
Height, cm	175 \pm 10	169 \pm 8
Mass, kg	90 \pm 20	71 \pm 8
Resting BP, mmHg	136 \pm 10/84 \pm 5	133 \pm 9/81 \pm 6
VO ₂ peak, mL \cdot min ⁻¹ \cdot kg ⁻¹	27.9 \pm 4.5	29.9 \pm 2.6
VO ₂ peak, L \cdot min ⁻¹	2.49 \pm 0.16	2.12 \pm 0.11
VO ₂ @ VT, L \cdot min ⁻¹	1.77 \pm 0.14	1.69 \pm 0.09
Power _{max} , W	170 \pm 14	141 \pm 10
Power _{100%} VT, W	120 \pm 12	114 \pm 6
Power _{90%} VT, W	106 \pm 11	99 \pm 4
Power _{80%} VT, W	91 \pm 12	86 \pm 5
Power _{70%} VT, W	77 \pm 9	72 \pm 4

BP, blood pressure; VO₂, oxygen consumption; VT, ventilatory threshold.

The subjects receiving medical therapy took all their medications as usual. The characteristics of the subjects are presented in Table 1. Before participation, all the subjects provided informed consent. The research protocol had been approved by the University of Wisconsin—La Crosse Institutional Review Board for the Protection of Human Subjects.

Initially, each subject performed an incremental exercise test to identify the peak oxygen uptake (VO₂ peak) and the ventilatory threshold,¹² which then were used to prescribe exercise training session intensity. Ventilatory threshold (VT) was determined using the V-slope method, with confirmation based on the response of the ventilatory equivalents for oxygen and carbon dioxide. During this incremental exercise test, the electrocardiogram, hemodynamic responses, and symptoms were monitored. After this, each subject participated in five experimental sessions. All exercise bouts were 120 minutes in duration and scheduled at the same time of day for each subject. The subjects refrained from consuming food or drink for 2 hours and alcohol for 24 hours before each bout. None of the subjects used tobacco.

Blood pressure was measured by auscultation. Before the first measurement, the subject was allowed to sit quietly for 5 minutes. Then at least three measurements were performed until a consistent value was observed. During the remainder of the protocol, only a single blood pressure measurement was performed at each time point. Each subject was studied during four randomly ordered exercise bouts at least 48 hours apart and during a control trial involving 120 minutes of continuous seated rest. The four exercise bouts consisted of exercising on a cycle ergometer at a power output calculated to require 70%, 80%, 90%, or 100% of the VO₂ at the VT (in a range from quite easy to rather hard).

At arrival to the laboratory, the subject had 5 minutes of seated rest, after which blood pressure and heart rate

(via radiotelemetry) measurements were obtained. Immediately after the initial blood pressure had been obtained (minute zero), the subject exercised for 25 minutes. Heart rate and rate of perceived exertion (RPE) were monitored during exercise to document exercise intensity along with exercising blood pressures measured at 10, 20, and 25 minutes. The category ratio RPE scale (eg, 0-10) was measured at the same time points.

At the conclusion of the 25-minute exercise bout, a 5-minute low-intensity cool-down was allowed, after which a resting seated blood pressure was taken. This blood pressure served as the 30-minute measurement. Additional resting blood pressures were taken at 60, 90, and 120 minutes. In the control trial, blood pressure was measured every 30 minutes after the initial measurement. Measurement of blood pressure was terminated at 120 minutes, corresponding to the protocol of the authors' previous study.¹⁰

Statistical analysis was performed using repeated measures analysis of variance (ANOVA) to examine change differences in systolic and diastolic blood pressures from rest to 30, 60 (30 after exercise), 90, and 120 minutes, and to assess for an interaction between exercise intensity and change in blood pressure. The alpha value indicating statistical significance was set at 0.05 or less. Tukey's post hoc tests were used to evaluate pairwise differences when justified by ANOVA.

RESULTS

The average exercise intensity during the exercise bouts, on the basis of RPE, were as follows: 70% VT = 3.0, 80% VT = 3.5, 90% VT = 4.0, and 100% VT = 5.0. These values represent perceived intensities ranging from "moderate" to "hard" on the RPE scale. The heart rate and blood pressure responses during the exercise bouts are presented in Figure 1. The percentage of max-

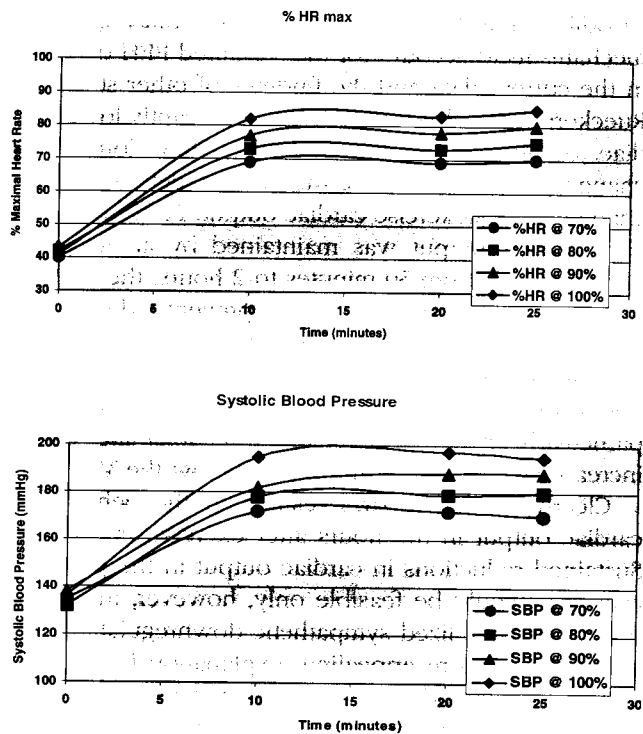


Figure 1. Pattern of heart rate response expressed as percentage of heart rate max) and systolic blood pressure during the four exercise bouts.

imal heart rate (%HRmax) data were consistent with conventional guidelines for exercise prescription.

After all four interventions (70%, 80%, 90%, and 100% of the VO_2 at the VT), there was a significant decrease in systolic blood pressure after exercise period, which was defined as measurements at times of 60 minutes and greater. In general, the magnitude of PEH was greater during the early postexercise period after the harder exercise bouts. However, by the 120-minute measurement, there was no difference in the magnitude of PEH related to exercise intensity (Figure 2). There were no statistically significant differences in diastolic blood pressure from rest after any of the interventions.

Postexercise hypotension was documented at all levels of exercise training intensity. There was a significant interaction between intensities and time of onset with PEH, magnitude of PEH, and duration of PEH, and the most dramatic early reductions in systolic blood pressure were found after the higher-intensity exercise bouts. The results showed the largest magnitude of PEH after exercise at 100% VT, with a reduction in systolic blood pressure 30, 60, and 90 minutes after exercise. Systolic blood pressure was lower for the entire recovery period, with the greatest reduction (-15 mmHg) at the 60-minute measurement. At the 90- and 120-minute measurements, there was a gradual return toward resting values, but systolic blood pressure still was 10 mm

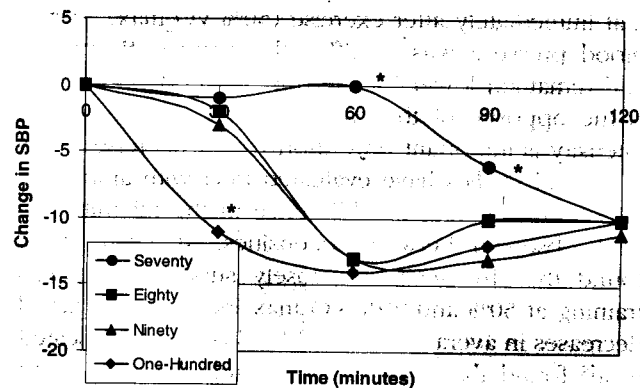


Figure 2. Control-corrected changes in systolic blood pressure after 25-minute exercise bouts of varying intensity. At the 30-minute measurement, the reduction in systolic blood pressure was significantly greater in the 100% ventilatory threshold (VT) exercise bout than in the other three bouts. At the 60- and 90-minute measurements, the reduction in systolic blood pressure was significantly less for the 70%VT exercise bout than for the other exercise bouts. At the 120-minute measurement, the reduction in systolic blood pressure was not significantly different from that for any of the exercise bouts.

Hg lower than the control at the 120-minute measurement. After the 90% VT intensity, systolic blood pressure was significantly reduced at the 60-minute measurement and stayed lower for the remainder of the recovery period (90- and 120-minute measurements). After the 80% VT exercise bout, there was a significant decrease in systolic blood pressure at the 90-minute measurement, which remained lower for the remainder of the recovery period. After the 70% VT exercise bout, there was no significant decrease in diastolic blood pressure until the 120-minute measurement.

DISCUSSION

The findings of this study suggest that PEH occurs after a wide range of exercise intensities from fairly easy to quite hard, and that the development of PEH appears to follow a pattern consistent with at least two independent mechanisms: an intensity-dependent mechanism and an intensity-independent mechanism. The greater magnitude of PEH shortly after the more intense exercise bouts is consistent with expectations based on nitric oxide-mediated vasodilation. The smaller and more consistent PEH observed at the 120-minute measurement after exercise is consistent with a non-nitric oxide-mediated mechanism. These observations are consistent with the findings of Hagberg et al,⁶ who reported that at higher intensities (70% vs 50% VO_2 max), there was a greater decrease in systolic blood pressure (-13 vs -8 mm Hg) for a longer period (3 hours vs 1 hour). However, these authors also found

that immediately after exercise (50% $\text{VO}_{2\text{max}}$), systolic blood pressure was significantly lower (-8 mm Hg) and remained lower for the duration of 1 hour, which is the opposite of the current results, suggesting that intensity is important only shortly after exercise.

Several studies have evaluated PEH with ambulatory monitoring, finding no difference in the magnitude of PEH observed between intensities. Marceau et al¹³ found that 10 weeks of closely supervised exercise training at 50% and 70% $\text{VO}_{2\text{max}}$ induced comparable decreases in average 24-hour blood pressure. Pescatello et al⁸ found that postexercise systolic blood pressure was reduced for 8 hours, apparently independently of exercise intensity (40% vs 70% $\text{VO}_{2\text{max}}$). However, these studies focused on the sustained effect of exercise on blood pressure, and thus may have missed the more transient effect evident within the first 30 minutes after exercise.

Most of the previous studies observed a significant reduction in diastolic blood pressure after exercise, whereas this was not the case in the current study. The authors have no explanation for this finding, although the current results are consistent with the earlier report of Schuster-Decker et al¹⁰ from the authors' laboratory, suggesting a predominantly systolic blood pressure response of PEH.

A reduction in blood pressure can be accomplished only by a reduction in cardiac output, total peripheral resistance, or both. Rueckert et al¹⁴ found that vascular resistance immediately after exercise was significantly lower than before exercise. As total peripheral resistance gradually normalized, these authors found a significant decrease in cardiac output, which contributed to the decline in blood pressure. This early decrease in peripheral resistance may be attributable to a number of factors including the effect of exercise-induced metabolites, flow-mediated vasodilation, reduced alpha-adrenergic responsiveness, reduced sympathetic outflow, and thermodilation.³ Floras and Wesche¹⁵ recently reported reductions in systolic, diastolic, and mean arterial pressures; calf vascular resistance; and total peripheral resistance, together with tachycardia-mediated increases in cardiac output after submaximal exercise. Sustained decreases in limb vascular and total peripheral resistances have been reported after exercise in both normotensive and hypertensive subjects.

These observations suggest that sustained vasodilation in skeletal muscle and other arterial beds may contribute to PEH. The factors that cause a decline in peripheral resistance could account for the greater reduction in PEH found at higher intensities in the current study. Higher intensities may mediate a greater response in exercise-induced metabolites, likely mediated by nitric oxide.³ The augmentation of PEH by pre-exercise ingestion of L-arginine suggests the importance of this mechanism.¹⁰ However, because this type of

vasodilation is time dependent, there must be other mechanisms to account for the sustained PEH observed in the current data and the findings of other studies.^{4,9} Rueckert et al¹⁴ also found that a significantly lower cardiac output contributed to PEH. They found that changes in heart rate were the primary determinants of changes in postexercise cardiac output. Early after exercise, cardiac output was maintained by an increased heart rate, but from 50 minutes to 2 hours, the increase in heart rate was not sufficient to prevent a decrease in cardiac output. It seems unlikely that significant reductions in beta-receptor density could occur in the short period of the single exercise bout used in this study,¹⁴ particularly given that circulating catecholamines do not increase significantly at intensities below the VT.

Clearly, further studies relating to the behavior of cardiac output in the hours after exercise are needed. Sustained reductions in cardiac output in the postexercise state would be feasible only, however, in the setting of a generalized sympathetic downregulation. The simpler, and more appealing, explanation for the sustained intensity-independent PEH involves a resetting of either intrinsic vascular tone or modulation of adrenergic output. How this might be accomplished remains unclear.

In summary, the magnitude of PEH response observed in the mildly hypertensive subjects in this study was dependent on exercise intensity only in the first 30 to 60 minutes after exercise, but the magnitude of the response was sufficient to be clinically important. It would be of interest to know whether multiple daily bouts of exercise could be used as a short-acting agent to control mild hypertension without the need for a resort to pharmacologic therapy.

References

1. Pescatello LS, Franklin B, Fagard R, Farquhar W, Kelly GA, Ray C. American College of Sports Medicine position stand: exercise and hypertension. *Med Sci Sports Exerc.* 2004;36:533-553.
2. Cordain L, Eades MR, Eades MD. Hyperinsulinemic diseases of civilization: more than just syndrome X. *Comp Biochem Physiol A.* 2003;136:95-112.
3. Halliwill JR. Mechanisms and clinical implications of postexercise hypotension in humans. *Exerc Sport Sci Rev.* 2001;65-70.
4. Beaulieu M, Nadeau A, Lacourciere Y, Cleroux J. Postexercise reduction in blood pressure in hypertensive subjects: effects of angiotensin converting enzyme inhibition. *Br J Clin Pharmacol.* 1993;36:331-338.
5. DiCarlo SE, Collins HL, Howard MG, et al. Postexercise hypotension: a brief review. *Sports Med.* 1994;5:17-27.
6. Hagberg JM, Montain SJ, Martin WH. Blood pressure and hemodynamic responses after exercise in older hypertensives. *J Appl Physiol.* 1987;63:270-276.
7. Kenny LJ, Seals DR. Postexercise hypotension: key features, mechanisms, and clinical significance. *Hypertension.* 1993;22:653-664.
8. Pescatello LS, Fargo AE, Leach CN, Scherzer HH. Short-term effect of dynamic exercise on arterial blood pressure. *Circulation.* 1991;83:1557-1561.

9. Pescatello LS, Kulikowich JM. The after effects of dynamic exercise on ambulatory blood pressure. *Med Sci Sports Exerc.* 2001;33:1855-1861.
10. Schuster-Decker R, Foster C, Porcari JP, Maher MA. Acute effects of dynamic exercise and nutritional supplementation on blood pressure in mildly hypertensive patients. *Clin Exerc Physiol.* 2002;4:17-21.
11. Halliwill JR, Minson CT, Joyner MJ. Effect of systemic nitric oxygen synthase inhibition on postexercise hypotension in humans. *J Appl Physiol.* 2000;89:1830-1836.
12. Foster C, Schrage M, Snyder AC. Blood lactate and respiratory methods of evaluating the capacity for sustained exercise. In: Maud PJ, Foster C, eds. *Physiological Assessment of Human Fitness.* Champaign, Ill: Human Kinetics Publishers; 1995: 57-72.
13. Marceau M, Kouame N, Lacourci Y, Cleroux J. Effects of different training intensities on 24-hour blood pressure in hypertensive subjects. *Circulation.* 1993;88:2803-2811.
14. Rueckert PA, Slane PR, Lissis DL, Hanson P. Hemodynamic patterns and duration of postexercise hypotension in hypertensive humans. *Med Sci Sports Exerc.* 1996;28:24-32.
15. Floras JS, Wesche J. Hemodynamic contributions to postexercise hypotension in young adults with hypertension and rapid resting heart rates. *J Hum Hypertens.* 1992;6:265-269.