Influence of endurance exercise training status and gender on postexercise hypotension

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Senitko, Annette N., Nisha Charkoudian, and John R. Halliwill. Influence of endurance exercise training status and gender on postexercise hypotension. J Appl Physiol 92: 2368–2374, 2002.—In sedentary individuals, postexercise hypotension after a single bout of aerobic exercise is due to a peripheral vasodilation. Endurance exercise training has the potential to modify this response and perhaps reduce the degree of postexercise hypotension. We tested the hypothesis that endurance exercise-trained men and women would have blunted postexercise hypotension compared with sedentary subjects but that the mechanism of hypotension would be similar (i.e., vasodilation). We studied 16 endurance-trained and 16 sedentary men and women. Arterial pressure, cardiac output, and total peripheral resistance were determined before and after a single 60-min bout of exercise at 60% peak oxygen consumption. All groups exhibited a similar degree of postexercise hypotension (~4–5 mmHg; P < 0.05 vs. preexercise). In sedentary men and women, hypotension was the result of vasodilation (Aresistance: ~8.9 ± 2.2%). In endurance-trained women, hypotension was also the result of vasodilation (~8.1 ± 4.1%). However, in endurance-trained men, hypotension was the result of a reduced cardiac output (~5.2 ± 2.4%; P < 0.05 vs. all others) and vasodilation was absent (~0.7 ± 3.3%; P < 0.05 vs. all others). Thus we conclude the magnitude of postexercise hypotension is similar in sedentary and endurance-trained men and women but that endurance-trained men and women achieve this fall in pressure via different mechanisms.

Cardiovascular deconditioning; blood pressure; cardiac output; vasodilator agents

After a single bout of dynamic exercise, there are profound changes in the mechanisms that regulate and determine arterial pressure, resulting in a postexercise hypotension that lasts nearly 2 h in sedentary but otherwise healthy individuals (8, 11). Whereas shorter or less vigorous exercise protocols elicit inconsistent changes in arterial pressure in normotensive subjects (3–5, 13, 23), postexercise hypotension is consistently elicited after longer (30–60 min) bouts of moderate-intensity [50–60% peak aerobic capacity (V02peak)] exercise (10–12, 16, 18, 30, 33). It is generally accepted that, in most subjects, postexercise hypotension is due to a persistent drop in peripheral vascular resistance that is not completely offset by increases in cardiac output (8–11).

Although the mechanisms of the vasodilation underlying postexercise hypotension are poorly understood, the sympathetic nervous system (10, 13), baroreflex resetting (10), nitric oxide (9, 22), and an unknown vasodilator have all been implicated (8, 9). Exercise training has the potential to impact each of these putative mechanisms. Furthermore, chronic endurance exercise training is associated with changes in vasodilator capacity (20, 29) and regulation of arterial pressure (26, 28). However, whether the phenomenon of postexercise hypotension is present in endurance-trained individuals is currently unknown. In this context, much of the information on postexercise hypotension comes from studies in sedentary or recreationally active individuals. It is possible that postexercise hypotension is an epiphenomenon of a sedentary lifestyle, unrelated to the physiology of exercise per se, but instead due to the imposition of a novel stress (exercise) on a sedentary cardiovascular system. Thus one could predict that postexercise hypotension would be blunted, or even absent, in individuals chronically exposed to the stress of exercise.

To date, studies on postexercise hypotension have not addressed the potential influence of training status on postexercise hypotension. Furthermore, although studies have not reported an influence of gender on postexercise hypotension (8), it is clear that gender can influence the cardiovascular response to both acute exercise (24) and exercise training (20), perhaps secondary to the effects of female reproductive hormones on cardiovascular function and regulation (2). Thus there is the potential for an interaction between endurance training status and gender affecting postexercise hypotension.

Therefore, the goal of this study was to determine the prevalence and extent of postexercise hypotension in endurance exercise-trained compared with sedentary men and women. We tested the hypothesis that endurance exercise-trained men and women would have blunted postexercise hypotension compared with...
sedentary subjects but that the mechanism of hypotension would be similar (i.e., vasodilation).

METHODS

This study was approved by the Institutional Review Board of the Mayo Clinic and Foundation, and each subject gave his or her informed, written consent before participation.

Subjects

A total of thirty-two healthy, nonsmoking, normotensive subjects between the ages of 18 and 35 yr participated in this study. On the basis of their exercise habits over the prior 12 mo, subjects were classified as “sedentary” (no regular physical activity) or “endurance exercise trained” (strenuous endurance exercise ≥4 days/wk). Sixteen subjects (8 men and 8 women) were sedentary, performing <1 h of aerobic exercise per week. Sixteen subjects (8 men and 8 women) were endurance exercise trained, exercising ≥4 days/wk for ≥1 h/day for at least 1 yr. The latter subjects all performed leg exercise, primarily running on the order of 20–60 miles/wk. Subject characteristics are shown in Table 1.

None of the subjects was taking medications other than oral contraceptives. All female subjects had a negative serum pregnancy test on the screening day. Because the effects of the menstrual cycle on postexercise hypotension are unknown, female subjects were studied during the early follicular phase (1–4 days after the onset of menstruation) or during the placebo phase of oral contraceptives to control for this potential influence.

Screening Visit

On a screening day, scheduled 7–10 days before the study day, \( V_{O_2 \text{peak}} \) and maximal heart rate were determined with a graded maximal cycle ergometer test consisting of 1- to 2-min workload increments. After a 5-min warm-up period of light cycling, workload increased by 20, 25, or 30 W every minute for sedentary subjects and increased by 50 W every minute up to 250 or 300 W, at which time workload increased every 2 min for trained subjects. Selection of the workload increment was subjective, with the goal of producing exhaustion within 8–12 min. All subjects achieved exhaustion within this time range. All subjects reached a respiratory exchange ratio of at least 1.17 during testing, and peak heart rates were 84–107% of age-predicted maximal heart rates. This test was used to determine the exercise workloads used on the study day (60% \( V_{O_2 \text{peak}} \)). Subjects also completed two standard questionnaires to further quantify exercise training status (1, 17) (Table 1).

Study Day Protocol

Subjects reported for the study at least 2 h postprandial, and they had abstained from caffeine for 12 h and from exercise for 24 h before the study. Subjects were instrumented on a tilt table for measurement of heart rate (electrocardiogram) and standard arterial pressure via an automated auscultometric device (Dinamap blood pressure monitor, model 1846SX, Critikon, Tampa, FL).

Measurements. Figure 1 shows the timeline for measurements during the study. Preexercise measurements consisted of triplicate recordings of arterial pressure, heart rate, and cardiac output (see below) in the supine position separated by 5 min to allow for \( C_2H_2 \) washout. Preexercise measurements of heart rate and arterial pressure were also recorded after 5 min of 70° head-up tilt. Exercise measurements of heart rate and arterial pressure were recorded every 10 min throughout the exercise period. After exercise, triplicate measurements of arterial pressure, heart rate, and cardiac output in the supine position were recorded at both 30 and 60 min postexercise. Heart rate and arterial pressure were also recorded after 5 min of 70° head-up tilt at 45 min postexercise. Measurements in the upright position were conducted to assess whether postexercise hypotension was exacerbated in any of the groups by the superimposition of orthostatic stress.

Exercise. Subjects underwent a 60-min period of seated upright cycling at 60% \( V_{O_2 \text{peak}} \). Exercise of this intensity and duration produces a sustained (≈2 h) postexercise hypotension (8). During exercise, subjects were allowed to drink

### Table 1. Subject characteristics

<table>
<thead>
<tr>
<th></th>
<th>Sedentary Women</th>
<th>Sedentary Men</th>
<th>Endurance Trained Women</th>
<th>Endurance Trained Men</th>
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<tr>
<td>n</td>
<td>8</td>
<td>8</td>
<td>8</td>
<td>8</td>
</tr>
<tr>
<td>Age, yr</td>
<td>25.8 ± 5.4</td>
<td>24.9 ± 5.3</td>
<td>28.4 ± 3.6</td>
<td>26.6 ± 4.6</td>
</tr>
<tr>
<td>Height, m</td>
<td>1.68 ± 0.07</td>
<td>1.75 ± 0.06†</td>
<td>1.64 ± 0.07</td>
<td>1.77 ± 0.04†</td>
</tr>
<tr>
<td>Weight, kg</td>
<td>64.4 ± 10.8</td>
<td>73.8 ± 5.6†</td>
<td>58.7 ± 6.1</td>
<td>75.0 ± 5.4†</td>
</tr>
<tr>
<td>Body mass index, kg/m²</td>
<td>22.8 ± 3.2</td>
<td>24.9 ± 1.9</td>
<td>21.8 ± 1.9</td>
<td>23.9 ± 1.3†</td>
</tr>
<tr>
<td>( V_{O_2 \text{peak}} ), ml·kg⁻¹·min⁻¹</td>
<td>27.1 ± 4.2</td>
<td>34.4 ± 1.7†</td>
<td>42.0 ± 6.2×</td>
<td>47.2 ± 2.8×†</td>
</tr>
<tr>
<td>Workload at 60% of ( V_{O_2 \text{peak}} ), W</td>
<td>64 ± 15</td>
<td>90 ± 23†</td>
<td>110 ± 30×</td>
<td>153 ± 15*†</td>
</tr>
<tr>
<td>Baecke sport index, arbitrary units</td>
<td>1.6 ± 0.2</td>
<td>1.7 ± 0.2</td>
<td>6.0 ± 0.5×</td>
<td>4.8 ± 0.6×</td>
</tr>
<tr>
<td>Index of physical activity, MET·h/wk</td>
<td>40.5 ± 6.0</td>
<td>42.0 ± 12.4</td>
<td>148.2 ± 15.3×</td>
<td>151.5 ± 32.2×</td>
</tr>
</tbody>
</table>

Values are means ± SE; n, no. of subjects. \( V_{O_2 \text{peak}} \), peak oxygen consumption; MET, metabolic equivalents. *P < 0.05 vs. sedentary within same gender. †P < 0.05 vs. women within same physical activity status.

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![Fig. 1. Timeline for study. Arterial pressure, cardiac output, and total peripheral resistance were determined before and after a single 60-min bout of cycling at 60% peak oxygen consumption by 16 endurance-trained and 16 sedentary men and women.](http://japphysiol.org)
Table 2. *Preexercise hemodynamics*

<table>
<thead>
<tr>
<th></th>
<th>Sedentary</th>
<th></th>
<th>Endurance Trained</th>
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<tbody>
<tr>
<td></td>
<td>Women</td>
<td>Men</td>
<td>Women</td>
<td>Men</td>
</tr>
<tr>
<td>Heart rate, beats/min</td>
<td>65.7 ± 1.5</td>
<td>64.7 ± 3.1</td>
<td>54.0 ± 4.2 č</td>
<td>57.5 ± 3.5</td>
</tr>
<tr>
<td>Systolic arterial pressure, mmHg</td>
<td>106.3 ± 4.0</td>
<td>119.8 ± 4.4†</td>
<td>110.0 ± 3.6</td>
<td>116.3 ± 2.5</td>
</tr>
<tr>
<td>Diastolic arterial pressure, mmHg</td>
<td>68.8 ± 2.9</td>
<td>66.4 ± 2.7</td>
<td>67.5 ± 2.4</td>
<td>70.1 ± 3.2</td>
</tr>
<tr>
<td>Mean arterial pressure, mmHg</td>
<td>81.3 ± 3.1</td>
<td>84.2 ± 3.1</td>
<td>81.7 ± 2.6</td>
<td>85.5 ± 2.8</td>
</tr>
<tr>
<td>Cardiac output, l/min</td>
<td>5.37 ± 0.30</td>
<td>6.10 ± 0.38</td>
<td>5.31 ± 0.43</td>
<td>6.45 ± 0.38†</td>
</tr>
<tr>
<td>Stroke volume, ml/beat</td>
<td>82 ± 4</td>
<td>95 ± 5†</td>
<td>100 ± 8 č</td>
<td>113 ± 3 č</td>
</tr>
<tr>
<td>Total peripheral resistance, units</td>
<td>15.4 ± 0.9</td>
<td>14.2 ± 1.1</td>
<td>15.9 ± 1.0</td>
<td>13.7 ± 1.1</td>
</tr>
</tbody>
</table>

Values are means ± SE. *P < 0.05 vs. sedentary within same gender. †P < 0.05 vs. women within same physical activity status.

water ad libitum. Ambient temperature was controlled between 22 and 24°C.

**Cardiac output.** Cardiac output was estimated by using an open-circuit acetylene washin method as developed by Stout et al. (31), modified by Gan et al. (7), and recently validated in humans vs. the direct Fick approach (15). This method allows the noninvasive estimation of cardiac output. We chose an open-circuit method because subjects are exposed to stable oxygen and carbon dioxide levels throughout the measurement in contrast to rebreath techniques. Subjects breathed a gas mixture containing 0.6% acetylene-9.0% helium-20.9% oxygen-balance nitrogen for 8–10 breaths via a two-way non-rebreathing valve attached to a custom-built pneumatic sliding valve. During the washin phase, breath-by-breath acetylene and helium uptake were measured by a respiratory mass spectrometer (Perkin-Elmer MGA 1100) and tidal volume was measured via a pneumotach (model 3700, Hans Rudolph, Kansas City, MO) linearized by the technique of Yeh et al. (34) and calibrated by using test gas before each study. The pneumotach and valve system had a combined dead space of 24 ml. Cardiac output calculations have been described previously (15). Total vascular resistance was calculated as mean arterial pressure/cardiac output and expressed as arbitrary units.

**Data Analysis.** The results were analyzed with a repeated-measures two-way ANOVA (gender vs. training status). Significant effects were further tested with Fisher’s least significant difference test, and differences were considered significant when P < 0.05. All values are reported as means ± SE.

**RESULTS**

Preexercise hemodynamics for the subject groups are shown in Table 2. As expected, endurance-trained subjects had larger stroke volumes at rest and a resting bradycardia, and mean arterial pressures were similar between the endurance-trained and sedentary groups.

The goal was to have each subject exercise for 60 min at 60% \( \dot{V}_O_2_{\text{peak}} \). The average workloads were higher for endurance-trained subjects than sedentary subjects within each gender (P < 0.05) and higher for men than women with the same physical activity status (P < 0.05; Table 1). The percentage of heart rate reserve (heart rate reserve is defined as maximal heart rate achieved during \( \dot{V}_O_2_{\text{peak}} \) testing minus the resting supine heart rate) reached during exercise by endurance-trained women (59.2 ± 7.7%) and men (62.0 ± 9.7%) and sedentary women (56.7 ± 8.6%) and men (55.9 ± 14.5%) did not differ and were consistent with the target workloads.

Figure 2 compares preexercise mean arterial pressures with values postexercise in all groups. Endurance-trained and sedentary men and women exhibited a similar degree of postexercise hypotension (~4–5 mmHg; P < 0.05 vs. preexercise). In addition, Table 3 shows values for systolic and diastolic pressures averaged across 30 and 60 min postexercise. As with mean arterial pressure, systolic and diastolic pressures were lower after exercise (P < 0.05 for all groups). Responses were similar in magnitude between the supine and upright position, as reported previously (25). Fig-

![Figure 2](http://jap.physiology.org/)
ure 3 shows the changes in hemodynamics that underlie postexercise hypotension in each group. In sedentary men and women, hypotension was the result of vasodilation with increased cardiac output, as previously reported (8). In endurance-trained women, hypotension was also the result of vasodilation with increased cardiac output. However, in endurance-trained men, hypotension was the result of a reduced cardiac output (see Fig. 3, top) and vasodilation was absent (Fig. 3, bottom).

Figure 4 shows the cardiac response associated with postexercise hypotension in each group. Within each gender, heart rate responses were similar independent of training status. There was a tendency for men to have a greater rise in heart rate than women ($P < 0.12$). In contrast to heart rate, there was a gender by training status interaction such that endurance-trained men had a greater fall in stroke volume than any other group ($P < 0.05$).

DISCUSSION

In this study, we tested the hypothesis that endurance exercise-trained men and women would have blunted postexercise hypotension compared with sedentary subjects but that the hemodynamics underlying postexercise hypotension would be qualitatively similar. In other words, we expected both endurance exercise-trained men and women and their sedentary counterparts to exhibit a postexercise vasodilation that was not completely offset by increases in cardiac output (8–11). Our results suggest that the magnitude of postexercise hypotension is similar in endurance-trained and sedentary men and women but that endurance-trained men achieve this fall in pressure via a different hemodynamic balance. In sedentary men and women, hypotension was the result of vasodilation as our laboratory has reported previously (8–11). In endurance-trained women, hypotension was also the result of vasodilation. However, in endurance-trained

### Table 3. Postexercise hemodynamics

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<thead>
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<th>Sedentary</th>
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<th>Endurance Trained</th>
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<tbody>
<tr>
<td></td>
<td>Women</td>
<td>Men</td>
<td>Women</td>
<td>Men</td>
</tr>
<tr>
<td>Systolic arterial pressure, mmHg</td>
<td>100.7 ± 3.4</td>
<td>114.2 ± 3.8†</td>
<td>105.3 ± 3.6</td>
<td>110.2 ± 2.3</td>
</tr>
<tr>
<td>Diastolic arterial pressure, mmHg</td>
<td>66.4 ± 2.1</td>
<td>62.7 ± 2.5</td>
<td>63.3 ± 2.7</td>
<td>66.0 ± 2.3</td>
</tr>
<tr>
<td>Mean arterial pressure, mmHg</td>
<td>77.8 ± 2.3</td>
<td>79.9 ± 2.8</td>
<td>77.3 ± 2.9</td>
<td>80.7 ± 2.1</td>
</tr>
</tbody>
</table>

Values are means ± SE. †$P < 0.05$ vs. women within same physical activity status.
men, hypotension was the result of a reduced cardiac output, whereas total peripheral resistance did not change in this group. These results add to the growing body of evidence that adaptations in cardiovascular regulation after chronic endurance exercise training are gender specific. Furthermore, these data provide new information that postexercise hypotension occurs in endurance exercise-trained humans and that the mechanisms of postexercise hypotension appear to depend on gender and training status.

Prior Studies on the Hemodynamics of Postexercise Hypotension

Prior studies have established that postexercise hypotension is characterized by a persistent drop in systemic vascular resistance that is not completely offset by increases in cardiac output (see Ref. 8 for review). Forearm and calf vascular resistances are decreased in parallel with systemic vascular resistance; thus the vasodilation that underlies postexercise hypotension is not restricted to the sites of active skeletal muscles (i.e., the legs) but involves inactive regions as well (e.g., the arms). The associated rise in arterial blood inflow through the vasodilated regions contributes to an increase in venous pooling of blood. The increase in venous pooling, in conjunction with the loss of plasma volume associated with exercise, leads to a reduction in central venous pressure (~2 mmHg supine) and cardiac filling pressure (preload) (9). Despite this fall in cardiac preload, stroke volume is maintained because of the reduction in cardiac afterload and a probable increase in cardiac contractility (8). The net result of these influences on the blood vessels and heart is that cardiac output is elevated (heart rate is higher and stroke volume is unchanged compared with before exercise). Thus it is generally accepted that, in sedentary individuals, postexercise hypotension is due to a persistent drop in systemic vascular resistance. Our data suggest endurance-trained women exhibit postexercise hypotension involving similar mechanisms.

Mechanism for Postexercise Fall in Cardiac Output in Trained Men

An obvious question is: What changes underlie the reduced cardiac output observed postexercise in endurance-trained men in the present study? An acceptable answer to this question should address the fact that 1) the endurance-trained men had a heart rate response similar to that of the sedentary men and 2) the endurance-trained women did not show the fall in stroke volume and cardiac output seen in the endurance-trained men.

Differences in the cardiac contractility response from pre- to postexercise might play a role in the divergent cardiac output responses between endurance-trained and sedentary men. However, both groups had a similar heart rate response during postexercise hypotension. If we consider the chronotropic response as a crude index of autonomic control of the heart, it would suggest that there would be no difference between the trained and sedentary men in terms of the inotropic response as well. Thus it seems unlikely that a change in cardiac contractility explains the fall in cardiac output. A more likely explanation is that the fall in central venous pressure, and thus cardiac preload, is greater in the endurance-trained men than in the sedentary men. If a greater fall in central venous pressure does exist in the endurance-trained men, it could be due to a greater loss of plasma volume (more sweating?; see below) or due to a redistribution of cardiac output from less compliant to more compliant vascular beds (27). It is also plausible that the same fall in preload pressure produces a greater fall in stroke volume due to the effect of cardiac hypertrophy in the endurance-trained men on the Frank-Starling pressure-volume curve for the heart (19). However, this would likely apply to the endurance-trained women as well (21), who did not show the fall in stroke volume seen in the endurance-trained men. Thus it seems the most likely explanation is that of a greater fall in central venous pressure in endurance-trained men, although we are unable to explain why there would be a greater loss of plasma volume (or a redistribution of cardiac output to more compliant vascular beds) selectively in this group of individuals. Along these lines, sweating rates are generally lower in women than in men (6). Thus it is possible that the trained men had greater sweating-related fluid loss, subsequently losing more plasma volume and undergoing greater reductions in central venous pressure. Unfortunately, sweat loss was not assessed in the present study.

Although all subjects exercised at the same relative workload (60% \( \dot{V}O_2 \text{peak} \)), endurance-trained men exercised at higher absolute workloads (153 W) than either sedentary men (90 W) or endurance-trained women (110 W). Although most cardiovascular and metabolic responses during exercise are related to relative and not absolute workload, it is feasible that a response linked to absolute workload mediates the proposed decline in central venous pressure in endurance-trained men. In support of this notion, Fig. 5 shows the relationship between stroke volume responses and ab-

![Fig. 5. Percent change in stroke volume from preexercise to 30 min postexercise as a function of absolute workload. •, Sedentary women; ▼, sedentary men; ○, endurance-trained women; ▲, endurance-trained men.](image-url)
solute workload across all subjects ($r^2 = 0.37$, $P < 0.05$). It is unclear at this time whether this correlation is causal or secondary to some other effect. One might speculate that elite athletes with greater aerobic capacity than our endurance-trained subjects might have greater falls in stroke volume.

**Unchanged Total Peripheral Resistance Postexercise in Endurance-trained Men**

The mechanisms of the vasodilation underlying postexercise hypotension in the sedentary man and women are poorly understood, although the sympathetic nervous system (10, 13), baroreflex resetting (10), nitric oxide (9, 22), and an unknown vasodilator have all been implicated (8, 9). Because we do not have data on regional vascular resistance, it is unclear whether vasodilation still occurred in some vascular regions (e.g., the legs), but was masked by vasoconstriction elsewhere, or whether vasodilation was absent postexercise in endurance-trained men. One possibility is that whatever vasodilator signal mediates postexercise vasodilation in sedentary men and women is not activated by exercise in endurance-trained men. However, why this response would be absent in the endurance-trained men but present in the endurance-trained women is not clear. It is also possible that, unlike sedentary men and women, trained men have increased sympathetic vasoconstrictor outflow during recovery from exercise that would oppose peripheral vasodilation. This could arise as a result of baroreflex compensation for the fall in cardiac output in the endurance-trained men. Although speculative, it could be that baroreflex resetting to lower pressures (10) occurs in sedentary and trained individuals alike, but that it results in sympathetic withdrawal in individuals who maintain high cardiac outputs after exercise and results in sympathetic activation in individuals who are unable to maintain adequate cardiac output as a result of plasma volume loss. A similar suggestion has been made by Raine et al. (25) and Takahashi et al. (92) to explain differences in cardiac output and vascular resistance between supine and upright recovery from exercise. Clearly, more studies are necessary to address many of these issues.

**Perspectives**

There is growing evidence to support the concept that postexercise hypotension is mediated by multiple and redundant mechanisms. In addition, it now appears that this phenomenon occurs equally in sedentary and endurance-trained individuals. Taken together, this suggests that postexercise hypotension is more than an epiphenomenon of a sedentary lifestyle but instead represents a highly regulated physiological response associated with exercise. As such, it is interesting to ponder whether postexercise hypotension serves a meaningful purpose. Along these lines, Hayes et al. (14) have suggested that postexercise hypotension may play a role in plasma volume recovery after exercise. If this is true, postexercise hypotension may represent a homeostatic mechanism and an essential part of the cardiovascular response to an acute bout of exercise.

**Conclusion**

The magnitude of postexercise hypotension is similar between sedentary and endurance-trained men and women, but endurance-trained men achieve this fall in pressure via a different mechanism. The explanation for this divergent hemodynamic response is unclear at this time and merits further study.

We thank Shelly K. Roberts and Karen P. Krucker for technical assistance. We especially thank the subjects who volunteered for this study.

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**REFERENCES**

13. Johnson BD, Beck KC, Proctor DN, Miller J, Dietz NM, and Joyner MJ. Cardiac output during exercise by the open


