Postexercise Hypotension and Sympathoinhibition in Borderline Hypertensive Men

John S. Floras, Christine A. Sinkey, Philip E. Aylward, Douglas R. Seals, Peter N. Thoren, and Allyn L. Mark

To determine if there would be a decrease in blood pressure after exercise in patients with borderline hypertension and if this decrease would be accompanied by a decrease in sympathetic nerve activity to muscle, we recorded multifiber postganglionic muscle sympathetic activity from the peroneal nerve at rest in nine men with borderline hypertension (age 25 ± 1 years, mean ± SEM) before and 60 minutes after 45 minutes of submaximal treadmill exercise. In addition, responses to a cold pressor test, handgrip, and the Valsalva maneuver were recorded before and after exercise. Four subjects were also studied before and after "sham" exercise. Sham exercise had no effect on blood pressure or sympathetic nerve activity whereas resting systolic blood pressure was lower after treadmill exercise in seven subjects (from 136 ± 4 before to 123 ± 2 mm Hg 60 minutes after exercise; p < 0.01). Sixty minutes after exercise, sympathetic nerve activity was lower in all seven subjects (from 19 ± 2 to 11 ± 2 bursts/min, p < 0.015; or from 27 ± 3 to 14 ± 2 bursts/100 heartbeats, p < 0.005) but was slightly increased in the two subjects without postexercise hypotension. Heart rate and pressor and sympathoneural responses to the cold pressor test, handgrip, and the Valsalva maneuver were not altered by prior exercise. When nitroprusside was infused in five subjects to produce a reduction in systolic blood pressure similar to that seen 60 minutes after exercise, this drug increased sympathetic discharge from 37 ± 6 to 57 ± 4 bursts/100 heartbeats (p < 0.001). These observations demonstrate 1) that rhythmic exercise can lower blood pressure in men with borderline hypertension and 2) that postexercise hypotension is associated with a decrease, rather than a reflex increase, in sympathetic discharge to muscle. Postexercise hypotension may be mediated in part by inhibition of sympathetic nerve activity. (Hypertension 1989;14:28–35)

After a single bout of prolonged muscular exercise, systolic and diastolic blood pressure decrease, sometimes for several hours.1–4 The depressor response to exercise is greater in magnitude and duration in hypertensive than in normotensive subjects and is also elicited after shorter periods of exercise in hypertensive individuals.5 Indeed, even brief (10 minutes) exercise can reduce the blood pressure of hypertensive subjects.

Proposed mechanisms for postexercise decreases in blood pressure include decreased blood volume, thermoregulatory vasodilatation, and accumulation of ischemic vasodilatory metabolites, but it is unlikely that these phenomena are greater in hypertensive than in normotensive subjects.

Heart rate does not increase after prolonged exercise in hypertensive subjects.2–4 The absence of the anticipated reflex tachycardia in response to this decrease in blood pressure suggests that neural regulatory mechanisms have been altered by exercise. Indeed, in spontaneously hypertensive rats (SHR), decreases in blood pressure after exercise

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are mediated by inhibition of efferent sympathetic nerve activity (SNA). Therefore, we postulated that the reduction in blood pressure seen after exercise in hypertensive subjects is associated with a reduction in SNA. To test this hypothesis, we used a microneurographic technique to measure multifiber postganglionic muscle SNA directly from the peroneal nerve of borderline hypertensive subjects before and after prolonged treadmill exercise.

Subjects and Methods

Subjects

We studied two groups of subjects. SNA was studied before and after treadmill exercise in the first group and before and during an intravenous infusion of nitroprusside in the second. The first group comprised nine men with borderline hypertension. Their mean age was 25 ± 1 (mean ± SEM) years. The second group consisted of five men, aged 24 ± 2 years. Two of the five were borderline hypertensive subjects who had participated in the exercise protocol, and three were normotensive subjects. Cuff blood pressure was measured on three or more occasions more than 1 week apart. Cuff pressure was measured in the laboratory in which these studies were to be performed. Subjects sat quietly for 15 minutes, and then three readings were taken during 6 minutes. Korotkoff Phase V was used to represent diastolic blood pressure. Subjects whose diastolic blood pressure readings were intermittently above 90 mm Hg on one or more visits, yet below 90 mm Hg on one or more other visits, were considered to have borderline hypertension. None of the subjects had received drug treatment for hypertension.

Informed, written consent was obtained after the rationale, nature, and potential risks of this research were explained. This protocol was approved by the Institutional Committee on Human Investigation at the University of Iowa.

Procedures

Subjects were seated in a comfortable reclining chair. The electrocardiogram and respiratory excursions were recorded continuously and inscribed onto paper by an ink recorder. Blood pressure was measured from the left arm with a standard sphygmomanometer attached to a mercury column.

Multifiber recordings of postganglionic SNA were obtained from a muscle fascicle of a peroneal nerve posterior to the fibular head by a microneurographic technique. Recordings were made with tungsten microelectrodes with a 200 μm-diameter shaft tapering to a 1-5-μm uninsulated tip. A reference electrode was inserted subcutaneously 1-3 cm from the recording electrode. Neural activity was amplified 30- to 80,000-fold and then passed through a band-pass filter with a bandwidth of 700-2,000 Hz. The filtered neurogram was routed through an amplitude discriminator to a storage oscilloscope and a loudspeaker. For recording and analysis, the filtered neurogram was passed through a resistance-capacitance integrating network (time constant, 0.1 seconds) to obtain a mean voltage neurogram of SNA.

There were three criteria for an acceptable recording of muscle SNA. First, weak electrical stimulation through the electrode in the peroneal nerve elicited involuntary muscle contraction, but not paresthesia. Second, tapping or stretching the muscle or tendons supplied by the impaled fascicle elicited afferent mechanoreceptor discharge whereas stroking the skin in the distribution of the peroneal nerve did not. Third, the neurogram contained spontaneous, intermittent, pulse-synchronous bursts that increased during held expiration and during phases two and three of a Valsalva maneuver. Evidence that such activity represents efferent postganglionic SNA has been summarized by Valbo et al.6 Neurograms that revealed spontaneous activity characteristic of cutaneous SNA were not accepted. An arousal stimulus that triggers cutaneous activity, but not muscle sympathetic activity, was used to test for cutaneous SNA.

Inadverent contraction of the leg muscles adjacent to the recording electrode elicits electromyographic activity that causes a sudden rise in baseline noise level on the mean voltage neurogram and a characteristic repetitive nerve discharge. These electromyographic artifacts are infrequent, are readily distinguished from sympathetic bursts, and are excluded from analysis. Microneurographic recordings are stable and are not affected by time or infusion of vehicle.

Exercise Protocol

This protocol had three parts: preexercise, exercise, and postexercise.

Preexercise. Once a stable sympathetic nerve recording was obtained from the left peroneal nerve, we measured blood pressure in the left arm each minute and recorded heart rate and SNA continuously for 10 minutes to establish control or baseline values. To test reflex responsiveness, subjects then performed a 90-second cold pressor test (immersion of the right hand in ice water) and 2 minutes of isometric handgrip at 30% of their maximum voluntary capacity (right hand). These interventions were followed by a Valsalva maneuver. Blood pressure was measured every 30 seconds during a control period (2 minutes), the reflex stimuli, and a 2-minute recovery period.

Exercise. Treadmill speed and grade were adjusted to elicit 70% of each subject’s resting heart rate reserve calculated as

\[
\text{resting heart rate} + 0.7[(220 - \text{age}) - \text{resting heart rate}]
\]

Subjects then exercised 45 minutes with the treadmill speed or grade adjusted as necessary to main-
tain this heart rate. The electrocardiogram was recorded continuously, and blood pressure was recorded every 5 minutes during exercise.

**Postexercise.** Subjects were seated as before exercise. Blood pressure (left arm) and heart rate were recorded every 5 minutes. The recording electrode was placed in the right peroneal nerve. We switched to the contralateral nerve after exercise to minimize the possibility of nerve injury from impaling the same nerve twice in the same study. An acceptable neurogram was obtained from this nerve by 36±5 minutes after exercise. Values obtained 60 minutes after exercise were compared with pre-exercise measurements. Next, we repeated the cold pressor test and handgrip, again using the right hand. The study concluded with a second Valsalva maneuver. The Valsalva maneuver permitted us to compare the quality and stability of the microneurographic recordings obtained before and after exercise.

**"Sham" exercise.** On a separate day, four of these subjects followed the protocol described above except that they did not perform treadmill exercise. Instead, they walked leisurely about the laboratory at will. Paired recordings of blood pressure, heart rate, and SNA were obtained before and 60 minutes after this sham exercise. Sham exercise preceded the exercise session in one subject and followed it in three subjects.

**Nitroprusside Protocol**

Subjects did not exercise during this session. SNA was obtained from the right peroneal nerve with subjects recumbent. Blood pressure was measured by sphygmomanometer, and heart rate was recorded continuously before and during an intravenous infusion of sodium nitroprusside, 1.2 µg/kg/min. Comparisons were made between baseline values and values during the third minute of the nitroprusside infusion.

**Analysis of Mean Voltage Neurogram**

Recordings were analyzed without knowledge of experimental conditions. Sympathetic bursts were identified by inspection of the mean voltage neurogram by two observers. Based on a systematic evaluation of 19 consecutive microneurographic records by two independent observers, Mark et al reported a mean intraobserver variability of 4.3% and a mean interobserver variability of 9.1%. Because these bursts are pulse synchronous, burst frequency may vary with heart rate. To adjust for anticipated changes in heart rate with exercise and nitroprusside, burst frequency was expressed both as bursts per minute and as bursts per 100 heartbeats as recommended.

**Statistics**

Results are expressed in terms of mean±SEM. Paired comparisons (Student’s t test) resulting in values of p<0.05 were considered significant.

**Results**

**Effect of Recent Exercise on Blood Pressure, Heart Rate, and Sympathetic Nerve Activity**

Systolic blood pressure increased, and diastolic blood pressure decreased from 135±3/83±3 mm Hg to 185±8/74±5 mm Hg within 5 minutes of starting treadmill exercise; blood pressure declined slowly thereafter. After 45 minutes of exercise, the mean blood pressure was 168±5 mm Hg systolic and 49±7 mm Hg diastolic. Heart rate increased from 70±3 beats/min to a peak of 168±3 beats/min after 25 minutes of exercise. This rate was sustained for the duration of exercise.

Sixty minutes after exercise, systolic blood pressure was significantly reduced from preexercise values (125±2 vs. 135±3 mm Hg; p<0.03), whereas heart rate remained elevated (84±4 vs. 70±3 beats/min; p<0.005) and diastolic blood pressure was unchanged (83±2 vs. 83±3 mm Hg). SNA, which
TABLE 1. Hemodynamics and Sympathetic Nerve Activity in Borderline Hypertensive Subjects With Postexercise Hypotension

<table>
<thead>
<tr>
<th>Variable</th>
<th>Preexercise</th>
<th>Sixty minutes after exercise</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Systolic blood pressure (mm Hg)</td>
<td>136±4</td>
<td>123±2</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Diastolic blood pressure (mm Hg)</td>
<td>82±4</td>
<td>83±2</td>
<td>NS</td>
</tr>
<tr>
<td>Heart rate (beats/min)</td>
<td>70±3</td>
<td>82±5</td>
<td>&lt;0.02</td>
</tr>
<tr>
<td>Sympathetic nerve activity (bursts/min)</td>
<td>19±2</td>
<td>11±2</td>
<td>&lt;0.015</td>
</tr>
<tr>
<td>Sympathetic nerve activity (bursts/100 heartbeats)</td>
<td>27±3</td>
<td>14±2</td>
<td>&lt;0.005</td>
</tr>
</tbody>
</table>

n=7. Values are mean±SEM.

was 28±2 bursts/100 heartbeats before exercise, declined to 18±4 bursts/100 heartbeats 60 minutes after exercise (p<0.005) (Figure 1).

Postexercise reductions in systolic blood pressure were seen in seven subjects. All seven also had lower SNA 60 minutes after exercise than before (Table 1 and Figures 2 and 3). In contrast, in the two subjects without postexercise hypotension (130 mm Hg before vs. 134 mm Hg 60 minutes after exercise), SNA was unchanged or slightly increased (24 bursts/min before vs. 32 bursts/min 60 minutes after exercise; 34 bursts/100 heartbeats before and 34 bursts/100 heartbeats after exercise).

Although prior exercise lowered systolic blood pressure and SNA, it did not affect the pressor or sympathetic nerve responses to the cold pressor test, to isometric exercise, or to the increase in SNA during the Valsalva maneuver (54±6 before and 43±5 bursts/100 heartbeats after exercise, n=6, p=NS) in those subjects with postexercise hypotension (Figure 4).

Effect of Sham Exercise on Blood Pressure, Heart Rate, and Sympathetic Nerve Activity

In contrast to treadmill exercise, sham exercise had no effect on systolic blood pressure or SNA. Blood pressure before and 60 minutes after sham exercise was 128±4/82±6 mm Hg and 134±9/88±10 mm Hg, respectively (n=4). Heart rate before and 60 minutes after sham exercise was 65±1 and 64±1 beats/min, respectively. SNA was 40±8 before and 36±4 bursts/100 heartbeats 60 minutes after sham exercise (p=NS). By way of comparison, in these same four subjects, blood pressure before and after treadmill exercise was 137±8/6±6 mm Hg and 122±3/84±4 mm Hg, respectively. Despite the increase in blood pressure with sham exercise, which would be expected to decrease SNA reflexively, and despite the decrease in blood pressure after treadmill exercise, which would be expected to increase SNA reflexively, the reduction in muscle SNA was more than threefold greater after treadmill exercise (28±3 vs. 18±2 bursts/100 heartbeats or −36%) than after sham exercise (−11%).

Effect of Nitroprusside on Blood Pressure, Heart Rate, and Sympathetic Nerve Activity

When nitroprusside was infused in five subjects to produce a reduction in systolic blood pressure similar to that seen after exercise, this drug increased heart rate (from 65±6 to 83±7 beats/min, p<0.001) and SNA (from 24±4 to 46±5 bursts/100 heartbeats, p<0.005; from 37±6 to 57±4 bursts/100 heartbeats, p<0.001) (Figure 1).

Nitroprusside also decreased diastolic blood pressure from 71±4 to 62±5 mm Hg. Although treadmill exercise did not influence average diastolic blood pressure in the entire group, four of the borderline hypertensive subjects had lower diastolic blood pressure 60 minutes after exercise than before. Indeed, these four subjects had greater reductions in both systolic and diastolic blood pressure (from 139±3/88±5 mm Hg to 124±3/84±4 mm Hg) than were produced by nitroprusside.
FIGURE 3. Tracings of muscle sympathetic nerve activity (SNA) at rest (30 seconds) and during a Valsalva maneuver before (30 seconds) and 60 minutes after (lower panel) treadmill exercise. SNA and systolic blood pressure (mm Hg) at rest are lower after exercise. SNA increased substantially with the Valsalva maneuver before and after exercise. Thus, the lower SNA after exercise could not be attributed to inadequate placement of the microneurographic electrode because the Valsalva maneuver elicited a marked increase in the amplitude and frequency of sympathetic nerve discharge before and after exercise.

FIGURE 4. Bar graphs of increases in systolic blood pressure (SBP, mm Hg) and muscle sympathetic nerve activity (MSNA, bursts/100 heartbeats) during the cold pressor test (CPT, n=6) and isometric handgrip (HG) at 30% of maximum voluntary capacity (n=4) before (C) and 60 minutes after exercise in borderline hypertensive subjects with postexercise hypotension. Exercise did not significantly affect these responses.

Discussion

This study demonstrates that a sustained bout of exercise sufficient to produce a postexercise decrease in systolic blood pressure also lowers postganglionic muscle SNA. Our key findings were as follows. First, SNA decreased in the seven subjects with postexercise hypotension but did not change in the two subjects whose blood pressure was not reduced by exercise. Second, sham exercise tended to increase, not decrease, blood pressure. Although this increase might be expected to decrease SNA reflexively, we saw more than three times greater inhibition of SNA after treadmill exercise than after sham exercise. These findings suggest that postexercise hypotension may be mediated in part by inhibition of SNA to muscle vessels. Third, in contrast to exercise, a decrease in systolic blood pressure produced by administration of nitroprusside increased, rather than decreased, SNA. Fourth, this reduction in blood pressure and SNA 60 minutes after exercise was associated with an increase in heart rate. Fifth, despite the reduction in baseline SNA, the pressor and sympathoneural responses to handgrip and the cold pressor test were preserved. Thus, mechanisms that increase blood pressure and efferent SNA reflexively do not appear to be attenuated after a bout of exercise. Finally, the depressor effect of exercise in borderline hypertensive subjects appears intermediate between that reported...
In normotensive subjects and in patients with established hypertension.\textsuperscript{2,3}

In the remainder of this discussion, we focus on the following points: 1) limitations of our methods and protocol, 2) potential mechanisms to account for this sympathoinhibition after exercise, 3) relation to previous studies of postexercise decreases in blood pressure, and 4) implications for the management of hypertensive patients.

**Limitations of Protocol**

Although mean values for systolic blood pressure and SNA were reduced 60 minutes after exercise, only seven of the nine subjects demonstrated postexercise hypotension. Because the principal aim of these experiments was to determine whether postexercise hypotension was associated with a reduction in SNA in humans as it is in SHR, we focused specifically on the seven subjects who displayed this phenomenon. The significant reduction in SNA seen in these seven subjects cannot be dismissed as a statistical consequence of “subgroup selection” because the calculated nominal \( p \) values (\( p < 0.015 \) for bursts/min and \( p < 0.005 \) for bursts/100 heartbeats) remain significant at less than the 5% level if corrected conservatively for subgroup analysis. The presence of sympathoinhibition in all seven subjects with postexercise hypotension and the absence of sympathoinhibition in the two subjects whose blood pressure did not decrease after exercise support the concept that postexercise hypotension is associated with a reduction in SNA.

An important consideration is the potential limitation of comparing muscle SNA from two separate sites before and after exercise. However, we do not believe this to be a significant limitation of our protocol for several reasons. First, previous investigations by Sundlof and Wallin\textsuperscript{9} have demonstrated a high degree of congruence between neurograms recorded simultaneously from the left and right peroneal nerves, and we have recently confirmed this important observation in our laboratory in simultaneous neurograms from a peroneal and a radial nerve.\textsuperscript{10} Second, burst frequency is highly reproducible within a given individual on repeat recordings.\textsuperscript{9} Third, sympatheural responses to standard stimuli such as the cold pressor test, handgrip, and the Valsalva maneuver were comparable before and after exercise despite the lower value of SNA at rest during the second recording. Finally, the contrasting effects of sham and treadmill exercise on SNA indicate that the responses we observed after exercise cannot be explained adequately by temporal or technical considerations.

The sham exercise studies were also useful in that they indicated that habituation to the laboratory environment over time, as suggested by Kaufman et al,\textsuperscript{4} was not responsible for the decrease in blood pressure observed after exercise. Habituation to the laboratory during the course of these experiments also cannot explain our findings because a previous study from our laboratory demonstrated that recordings of muscle SNA in such subjects is stable over time.\textsuperscript{7}

To counter the possibility that the marked increase in SNA we observed in the nitroprusside experiments was due to a reduction in diastolic rather than systolic blood pressure, we identified those subjects who also had a decrease in diastolic pressure after exercise. All four of these subjects had a decrease, rather than an increase, in SNA after exercise despite similar reductions in mean arterial pressure that were achieved with nitroprusside. Thus, we cannot attribute the contrasting effects of exercise and nitroprusside on SNA to their effects on diastolic blood pressure.

Similarly, the contrasting effects of exercise and nitroprusside on SNA cannot be attributed to our expression of burst frequency as bursts per 100 heartbeats (Figure 1). The key point to be emphasized is that uncorrected burst frequency (bursts/min) increased when nitroprusside was infused but was lower 60 minutes after exercise, whereas the effects of these two interventions on heart rate were virtually identical.

**Potential Mechanisms**

We suggest three potential mechanisms by which exercise may decrease muscle SNA in humans. First, prolonged increases in systolic blood pressure during exercise may suppress efferent SNA. Sustained increases in blood pressure produced by infusion of phenylephrine and angiotensin inhibit renal SNA in rabbits for up to 90 minutes after these vasoconstrictors have been stopped and blood pressure has returned to control values.\textsuperscript{11}

Postexercise facilitation of inhibitory cardiopulmonary reflexes may be a second potential mechanism. Studies in conscious sinoaortic baroreceptor deafferented dogs by Daskalopoulos et al\textsuperscript{12} have demonstrated that cardiopulmonary baroreceptors with vagal afferents act to decrease systemic vascular resistance reflexively after exercise. Bennett et al\textsuperscript{13} addressed this issue in humans by studying reflex forearm vasoconstrictor responses to deactivation of cardiopulmonary baroreceptors with lower body negative pressure in hypertensive subjects. These authors reported enhanced forearm vasoconstrictor responses to this stimulus 60 minutes after treadmill exercise and interpreted this observation as suggesting that the tonic inhibitory influence of cardiopulmonary baroreceptors might be greater at this time. Augmentation of inhibitory cardiopulmonary baroreceptor reflexes might explain the reduction in muscle SNA seen after exercise.

Third, experiments performed on SHR by one of us (P.N.T.) indicate that prolonged stimulation of somatic afferents during exercise activates opioid and serotonergic systems that inhibit sympathetic outflow by modulating baroreceptor reflexes centrally.\textsuperscript{5,13}
Relation to Previous Studies of Postexercise Decreases in Blood Pressure

Our observations are not entirely consistent with those of Wilcox et al.²⁻⁴ and Bennett et al.³ who followed the same protocol as these workers, also did not detect a decrease in diastolic blood pressure 60 minutes after exercise.

The decline in blood pressure after exercise was not accompanied by the expected reflex tachycardia in three previous studies,²⁻⁴ whereas we observed an increase in heart rate. The faster heart rates in our study may reflect our more intense exercise protocol. These other groups used a protocol of 10-minute bouts of exercise interrupted by rest.²⁻⁴ Exercise heart rates in these studies averaged only 135 beats/min. An alternative possibility is that the persistent increase in heart rate after exercise is a characteristic of borderline hypertensive subjects, but not of normotensive subjects or patients with established hypertension. In this regard, Somers et al.,¹⁴ who also studied borderline hypertensive subjects, observed an increase in heart rate after prolonged exercise, whereas Hagberg et al.,¹⁵ who exercised older subjects with essential hypertension, did not. A third possibility is that the intensity of this exercise led to greater increases in plasma epinephrine than in the previous studies. When taken up by sympathetic nerve endings¹⁶ and subsequently discharged, neurally released epi-

We cannot comment definitively on the possible effect of postexercise changes in hemodynamics on muscle SNA in our subjects. Prolonged bicycle exercise in elderly hypertensive subjects¹⁵ results in sustained reductions in cardiac output and stroke volume. A sustained reduction in cardiac output is also seen in young normotensive subjects after prolonged strenuous treadmill exercise.¹⁹ Because muscle vascular resistance is tightly linked to efferent muscle SNA,⁶ our observations would suggest that muscle vascular resistance is also decreased after exercise; thus, if systemic vascular resistance is higher overall as suggested by Hagberg et al.,¹⁵ this may be due to increases in resistance in other vascular beds. The increase in heart rate and the dissociation between changes in muscle SNA and diastolic blood pressure we observed are consistent with this concept. Moreover, it is known that there is selectivity of arterial baroreceptor control of heart rate and regional vascular resistance particularly of the splanchnic circulation.²⁰ Thus, an increase in heart rate and in splanchnic vascular resistance would be appropriate reflex responses to the lower systolic blood pressure after exercise. Further, there is experimental and clinical evidence that the tonic inhibitory influence of cardiopulmonary baroreceptors, which appear to selectively control muscle SNA,²⁰,²¹ might be greater after exercise.³,¹²

Implications

Evidence from recent studies²²⁻²⁴ supports the concept that chronic exercise training can lower blood pressure, sympathetic tone, and (in contrast to these acute aftereffects of exercise) heart rate. Although our observations suggest that exercise may lower the blood pressure of selected young men with borderline or mild essential hypertension through inhibition of the sympathetic nervous system for an hour or more, it is not yet clear as to whether the acute and chronic hypertensive effects of exercise are mediated through the same mechanism.

This study demonstrates that blood pressure declines after prolonged exercise in most subjects with borderline hypertension. This reduction in blood pressure is associated with a decrease, rather than an anticipated increase, in muscle SNA.

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