

Regional hemodynamics during postexercise hypotension.

I. Splanchnic and renal circulations

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Pricher, Mollie P., Lacy A. Holowatz, Jay T. Williams, Jennifer M. Lockwood, and John R. Halliwill. Regional hemodynamics during postexercise hypotension. I. Splanchnic and renal circulations. *J Appl Physiol* 97: 2065–2070, 2004. First published August 13, 2004; doi:10.1152/japplphysiol.00465.2004.—Moderate exercise elicits a relative postexercise hypotension that is caused by an increase in systemic vascular conductance. Previous studies have shown that skeletal muscle vascular conductance is increased postexercise. It is unclear whether these hemodynamic changes are limited to skeletal muscle vascular beds. The aim of this study was to determine whether the splanchnic and/or renal vascular beds also contribute to the rise in systemic vascular conductance during postexercise hypotension. A companion study aims to determine whether the cutaneous vascular bed is involved in postexercise hypotension (Wilkins BW, Minson CT, and Halliwill JR. *J Appl Physiol* 97: 2071–2076, 2004). Heart rate, arterial pressure, cardiac output, leg blood flow, splanchnic blood flow, and renal blood flow were measured in 13 men and 3 women before and through 120 min after a 60-min bout of exercise at 60% of peak oxygen uptake. Vascular conductances of leg, splanchnic, and renal vascular beds were calculated. One hour postexercise, mean arterial pressure was reduced (79.1 ± 1.7 vs. 83.4 ± 1.8 mmHg; $P < 0.05$), systemic vascular conductance was increased by $\sim 10\%$, leg vascular conductance was increased by $\sim 65\%$, whereas splanchnic (16.0 ± 1.8 vs. 18.5 ± 2.4 ml \cdot min $^{-1}\cdot$ mmHg $^{-1}$; $P = 0.13$) and renal (20.4 ± 3.3 vs. 17.6 ± 2.6 ml \cdot min $^{-1}\cdot$ mmHg $^{-1}$; $P = 0.14$) vascular conductances were unchanged compared with preexercise. This suggests there is neither vasoconstriction nor vasodilation in the splanchnic and renal vasculature during postexercise hypotension. Thus the splanchnic and renal vascular beds neither directly contribute to nor attenuate postexercise hypotension.

exercise; sympathetic nervous system; vascular resistance; baroreflex; vascular conductance

AFTER AN ACUTE BOUT OF dynamic exercise, arterial pressure is decreased by ~ 5 – 10 mmHg in the supine position relative to preexercise levels for up to 2 h (11, 15). In most individuals, this drop in pressure is caused by a rise in systemic vascular conductance that is not completely offset by increases in cardiac output, although endurance-trained men achieve this reduced mean arterial pressure via reductions in cardiac output (31).

Studies on the mechanisms of postexercise hypotension thus far have focused largely on the vasodilation of skeletal muscle vascular beds. Both exercising and nonexercising skeletal muscle vascular beds have increased conductance after exercise and thus contribute to the net rise in systemic vascular conductance (5, 11, 12, 15, 31). However, it is unclear whether vasodilation in skeletal muscle vascular beds can account for

the entire rise in systemic vascular conductance postexercise. On the basis of a retrospective analysis of available data, we have estimated that skeletal muscle vascular beds can only account for $\sim 30\%$ of the increase in systemic vascular conductance observed after a bout of exercise (unpublished observations). This begs the question: What other vascular beds are likely to contribute to the increased systemic vascular conductance during postexercise hypotension?

Possible sites of vasodilation include the cutaneous, splanchnic, renal, and cerebral circulations. However, a recent study suggests that global cerebral blood flow is unaltered after exercise sufficient to elicit postexercise hypotension and that blood flow to some brain regions (e.g., the insular cortex) is decreased postexercise (35). Therefore, it is unlikely that the cerebral circulation plays a role in the increase in systemic vascular conductance that underlies postexercise hypotension.

The splanchnic and renal circulations have large conductances at rest, each receiving $\sim 20\%$ of cardiac output (30). These circulations are capable of greatly changing vascular tone in response to changes in sympathetic nerve activity. It has been well established that, during exercise, splanchnic and renal conductances are considerably reduced to supply blood to the exercising muscle and, most importantly, maintain adequate perfusion pressure to the working muscle (7, 10, 27–30). It is unclear, however, how these vascular beds react following a bout of moderate exercise.

During orthostatic stress, the splanchnic and renal vascular beds respond to sympathetic influences to defend arterial pressure. With increasing levels of lower body negative pressure, there is splanchnic vasoconstriction followed by renal vasoconstriction, contributing to a rise in systemic vascular resistance and decreasing venous pooling (17, 28). In patients who have had splanchnic sympathectomy, orthostasis results in severe hypotension (25), illustrating the importance of the splanchnic vascular bed in blood pressure regulation. Moderate levels of lower body negative pressure do not decrease renal blood flow, but renal blood flow decreases after prolonged or extreme levels of lower body negative pressure, and this response becomes crucial during hypovolemia or heat stress. However, if heat stress is superimposed or a subject is dehydrated, renal blood flow is decreased during orthostasis sooner than in normal conditions (17, 22).

It is plausible that the postexercise drop in mean arterial pressure induces baroreflex-mediated vasoconstriction in vascular beds such as the renal and splanchnic regions. Alternatively, it is feasible that these two vascular beds vasodilate after exercise. It has previously been shown that, after a bout of

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dynamic exercise, skeletal muscle vascular beds are dilated due to reductions in sympathetic nerve activity, reduced vascular responsiveness to sympathetic nerve activity, and release of an unidentified vasodilator substance (11, 12, 14). These same changes may also be occurring in the splanchnic and renal vascular beds, leading to splanchnic and renal vasodilation and consequently contributing to postexercise hypotension.

Therefore, the purpose of this study was to determine the role of the splanchnic and renal vascular beds during postexercise hypotension in humans. Specifically, we tested the hypothesis that the splanchnic and/or renal vascular beds are vasodilated during recovery from moderate-intensity exercise, contributing to the net rise in systemic vascular conductance that underlies postexercise hypotension. In a second study, reported in the companion article (34), we investigated the role of the cutaneous vascular bed during postexercise hypotension.

METHODS

This study was approved by the Institutional Review Board of the University of Oregon. Informed consent was obtained from each subject before involvement.

Subjects. Sixteen subjects (13 men, 3 women, age 18–30 yr) participated in this study. Subjects were healthy, normotensive, non-smokers, and taking no medications other than oral contraceptives. Subjects were either sedentary or recreationally active. Female subjects were studied during the early follicular stage of their menstrual cycle or during the placebo phase of the oral contraceptive cycle. Female subjects had a negative pregnancy test on both the screening and study days.

Screening day. Subjects performed an incremental bicycle exercise test (Lode Excaliber, Groningen, The Netherlands) consisting of 1-min workload increments to determine peak oxygen uptake ($\dot{V}O_{2\text{ peak}}$). Specifically, after a 2-min warm-up period of easy cycling (20–30 W), workload increased at 20, 25, or 30 W every minute. Selection of the workload increment was subjective, with the goal of producing exhaustion within 8–12 min. Whole body oxygen uptake ($\dot{V}O_2$) was measured via a mixing chamber (Parvomedics, Sandy, UT) integrated with a mass spectrometry system (Marquette MGA 1100, MA Tech Services, St. Louis, MO). All subjects reached subjective exhaustion [Borg (2) rating of perceived exertion = 19–20] within the 8- to 12-min period. After the subjects rested for 15–20 min, they returned to the cycle ergometer for assessment of the workload corresponding to a steady-state $\dot{V}O_2$ of 60% of $\dot{V}O_{2\text{ peak}}$. This workload was used on the study day for the 60-min exercise bout. Subjects self-reported activity levels on two questionnaires (1, 19). Subject characteristics are presented in Table 1.

Study day. Subjects arrived between 7 and 9 AM having fasted overnight. Per instruction, they had refrained from exercise, alcohol, and caffeine for the 24 h before the study. After a prestudy weight was obtained, subjects were instrumented while lying supine. Heart rate was assessed by a five-lead electrocardiogram (model Q710, Quinton

Instruments, Bothell, WA), and mean arterial pressure was attained by an automated sphygmomanometer (Dinamap Pro100 vital signs monitor, Critikon, Tampa, FL). Venous catheters were inserted into each arm in the antecubital region. The left arm catheter was used for infusion, and the right arm catheter was used for blood draws. During the protocol, 11 ml of blood were taken every 20 min for the analysis of splanchnic blood flow, renal blood flow, hematocrit, and hemoglobin. All other measurements were taken every 10 min for 30 min before exercise and through 120 min after exercise. All pre- and postexercise measurements were made while the subjects were in the supine position.

Exercise. Subjects exercised upright on a stationary cycle at 60% of $\dot{V}O_{2\text{ peak}}$ for 60 min. Exercise of this intensity and duration consistently produces a sustained (~2 h) postexercise hypotension in healthy normotensive subjects (11). During the 60 min of exercise, heart rate and arterial pressure were determined every 10 min. On the basis of initial body weight, subjects drank between 400 and 600 ml of water to offset volume loss during exercise. Subjects were then laid supine for postexercise measurements.

Measurement of splanchnic and renal blood flow. Splanchnic blood flow was determined by the clearance of indocyanine green (ICG) (Akorn, Buffalo Grove, IL), and renal blood flow was determined by the clearance of *p*-aminohippurate (PAH; Clinalfa, Laeufelfingen, Switzerland) as previously described (3, 22, 23).

A blank blood sample was drawn before administration of bolus doses of 0.10 mg/kg of ICG and 8.0 mg/kg of PAH. A constant infusion of 0.5 mg/ml of ICG and 12 mg/ml of PAH was started immediately after bolus dose administration. To allow plasma concentrations of ICG and PAH to achieve steady-state levels, preexercise measurements began 45 min after the start of the infusion. Subsequent blood sampling at 20-min intervals was used to determine steady-state plasma concentrations of ICG and PAH. Blood samples were collected in prechilled tubes containing heparin for ICG analysis or EDTA for PAH analysis. Samples were then centrifuged for 10 min at 1,300 RCF in a refrigerated centrifuge (4°C). Plasma samples for PAH analysis were stored at –80°C for subsequent analysis while samples for ICG were kept on ice for same-day analysis.

ICG plasma concentrations were measured with a spectrophotometer (model DU-7400, Beckman Instruments, Fullerton, CA) at 800 nm. Splanchnic plasma flow was calculated as infusion rate/(extraction ratio·plasma concentration). We assumed an extraction ratio of 0.85 for young healthy individuals (7). Splanchnic blood flow was calculated as splanchnic plasma flow/(1 – hematocrit) in milliliters per minute, and splanchnic conductance was calculated as splanchnic blood flow/mean arterial pressure and expressed as milliliters per minute per millimeters of Hg.

In an analogous fashion, plasma concentrations of PAH were measured with the reagent *N*-(1-naphthyl)ethylenediammonium dichloride (3, 23) and measured with a spectrophotometer at 550 nm. Renal plasma flow was calculated as infusion rate/(extraction ratio·plasma concentration). We assumed an extraction ratio of 0.91 for young healthy individuals (10). Renal blood flow was calculated as renal plasma flow/(1 – hematocrit) in milliliters per minute and renal vascular conductance was calculated as renal blood flow/mean arterial pressure and expressed as milliliters per minute per millimeters of Hg.

Extensive use of ICG and PAH for the measurement of splanchnic and renal blood flow has shown that extraction of these compounds does not change in healthy young subjects under a wide range of stresses and provocations such as lower body negative pressure, heat stress, and exercise (3, 4, 10, 22, 23, 26, 27, 29, 30).

Measurement of leg blood flow. Femoral artery diameter and velocity were measured by using an ultrasound probe (10-MHz linear-array vascular probe, GE Vingmed System 5, Horton, Norway). The entire width of the artery was insonated with an angle of 60°. Velocity measurements were taken immediately after diameter measurements. Leg blood flow was calculated as artery cross-sectional area multiplied by femoral mean blood velocity, doubled to represent

Table 1. Subject characteristics

Age, yr	20.9 ± 2.9
Height, m	1.76 ± 0.12
Weight, kg	83.3 ± 20.8
Body mass index, kg/m ²	26.4 ± 3.8
$\dot{V}O_{2\text{ peak}}$, ml·kg ^{−1} ·min ^{−1}	40.9 ± 5.7
Workload at 60% $\dot{V}O_{2\text{ peak}}$, W	144 ± 35
Baecke sport index, arbitrary units	8.6 ± 1.6
Index of physical activity, MET·h ^{−1} ·wk ^{−1}	92.2 ± 57.8

Values are means ± SD; 16 subjects. $\dot{V}O_{2\text{ peak}}$, peak oxygen consumption; MET, metabolic equivalents.

both legs, and reported as milliliters per minute. Leg vascular conductance was calculated as flow for both legs/mean arterial pressure and expressed as milliliters per minute per millimeters of Hg.

Measurement of cardiac output. The noninvasive method of open-circuit acetylene washin was used to assess cardiac output as developed by Stout et al. (32), Gan et al. (8), and Johnson et al. (18). Subjects breathed a mixture of 0.6% acetylene, 9.0% helium, 20.9% oxygen, and balance nitrogen for 8–10 breaths via a two-way nonrebreathing valve. Breath-by-breath acetylene and helium uptake were measured by a respiratory mass spectrometer (Marquette, MGA 1100, MA Tech Services) and pneumotach (model 3700, Hans Rudolph, Kansas City, MO) interfaced with a custom-designed data-acquisition system. Systemic vascular conductance was calculated as cardiac output/mean arterial pressure and expressed as milliliters per minute per millimeters of Hg.

Plasma volume. Percent change in plasma and blood volume were calculated from changes in hemoglobin and hematocrit by the method of Dill and Costill (6).

Statistics. Data were found to be normally distributed; thus parametric statistics were used. The results were analyzed with repeated-measures ANOVA. Significant effects were further tested with Fischer's least significant difference test, and differences were considered significant when $P < 0.05$. All values are reported as means \pm SE.

RESULTS

Exercise. Heart rate increased during exercise from 60.3 ± 2.3 to 153.8 ± 3.6 beats/min (average over 60 min; $P < 0.05$). The goal was to have each subject exercise for 60 min at 60% $\dot{V}O_{2\text{ peak}}$. 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 ($71.5 \pm 2.4\%$) was consistent with the target workload.

Postexercise. Preexercise and postexercise hemodynamics are illustrated in Fig. 1. Mean arterial pressure remained lower ($P < 0.05$) through 80 min postexercise, whereas cardiac output tended to be elevated during recovery from exercise (Table 2). As a result, systemic vascular conductance was elevated ($P < 0.05$) postexercise. In parallel with systemic vascular conductance, leg vascular conductance was increased through 100 min ($P < 0.05$). Splanchnic vascular conductance and renal vascular were not changed from preexercise levels, although both decreased during exercise ($P < 0.05$).

Plasma and blood volume. During exercise, both plasma and blood volume decreased from baseline by $9.5 \pm 0.7\%$ ($P < 0.05$). However, at 20 min postexercise, both plasma and blood volume had returned to preexercise levels ($-1.1 \pm 0.7\%$; $P = 0.13$).

DISCUSSION

In most populations, postexercise hypotension is typified by an increase in systemic vascular conductance that is not completely offset by an increase in cardiac output (11). Explorations before this have measured the increase in systemic vascular conductance and skeletal muscle conductance. In this study, we tested the hypothesis that the splanchnic and/or renal vascular beds have increased conductance postexercise and contribute to the relative hypotension seen after a bout of moderate exercise. In the companion paper (34), we investigated the contribution of the cutaneous vascular bed to postexercise hypotension. Our findings indicate that the splanchnic

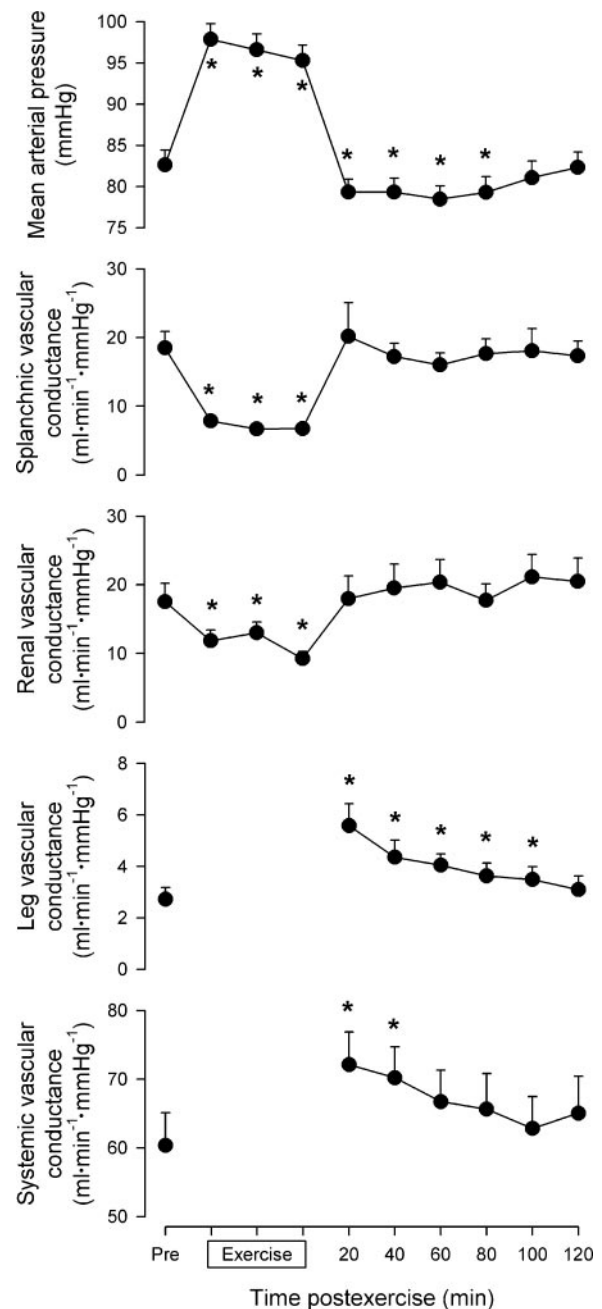


Fig. 1. Systemic and regional hemodynamics before (Pre), during, and after exercise. Values are means \pm SE for 16 subjects. * $P < 0.05$ vs. preexercise.

and renal vascular beds are neither vasodilated nor vasoconstricted after exercise when compared with preexercise values. On face value, it would seem that neither of these vascular beds contributes to postexercise hypotension.

It is well known that, during exercise, the arterial baroreflex is reset to maintain a higher arterial pressure, such that the baroreflex-mediated relation between arterial pressure and sympathetic outflow is shifted upward and to the right, so that, for a given arterial pressure, sympathetic nerve activity is greater. It appears that this resetting affects control of sympathetic nerve activity to the heart as well as the skeletal muscle, splanchnic, and renal circulations. This resetting of the baroreflex and activation of sympathetic vasoconstrictor nerve activ-

Table 2. Systemic and regional hemodynamics before and after exercise

	Preexercise	60 min Postexercise	120 min Postexercise
Heart rate, beats/min	59.8±2.4	67.7±2.8*	62.1±3.1
Stroke volume, ml/beat	87.0±9.0	79.3±6.1*	87.5±8.0
Cardiac output, l/min	5.02±0.40	5.27±0.36	5.29±0.40
Leg blood flow, ml/min	221.4±32.4	313.6±32.7*	250.3±41.6
Splanchnic blood flow, ml/min	1,494±176	1,256±144	1,435±193
Renal blood flow, ml/min	1,468±217	1,623±271	1,705±282

Values are means ± SE; for 16 subjects for heart rate, stroke volume, cardiac output, leg blood flow, and splanchnic blood flow and for 12 subjects for renal blood flow. * $P < 0.05$ vs. preexercise.

ity contributes to the redistribution of blood flow to the active skeletal muscle by vasoconstriction in inactive regions and maintaining perfusion pressure to active regions (25). The splanchnic and renal vascular beds are important contributors to this response. Because they both receive ~20% of the resting cardiac output, but extract only a minimal amount of oxygen (10, 26), substantial vasoconstriction is possible. The extent to which the splanchnic and renal vascular beds constrict is related to the intensity of exercise; in other words, the more intense the exercise, the more these regions constrict to redistribute blood and maintain perfusion pressure to skeletal muscle vascular beds (26). Suzuki et al. (33) reported that, immediately after a session of exhaustive exercise, renal blood flow was diminished 53% compared with preexercise levels. However, Castenfors (4) reported that renal blood flow was near preexercise levels by 30 min postexercise after an extended exercise session. These prior observations are consistent with the findings of the present study, suggesting that renal blood flow is reduced immediately after exercise but returns to preexercise levels within 20–30 min.

Prior work on postexercise hypotension has found that, after exercise, the baroreflex is reset to defend a lower pressure, such that the baroreflex-mediated relation between arterial pressure and sympathetic outflow is shifted downward and to the left (relative to preexercise) so that, for a given arterial pressure, sympathetic nerve activity is less. This resetting affects muscle sympathetic nerve activity (14) but, interestingly, does not extend to control of the heart (14, 15). Thus, during postexercise hypotension, there is an inhibition of sympathetic nerve activity to the skeletal muscle vascular beds concomitant with pressure reductions, but heart rate remains elevated. Because there is a resetting of baroreflex control of muscle sympathetic nerve activity during postexercise hypotension (14), it is possible this resetting extends to other vascular beds, particularly those that play critical roles in blood pressure regulation such as the splanchnic and renal circulations (25, 28). Along these lines, recent evidence in rats supports the notion that baroreflex control of renal sympathetic nerve activity is reset during postexercise hypotension (21). If this is also the case in humans, the absence of constriction in the splanchnic and renal vascular beds after exercise may be indicative of this resetting. Of note, our laboratory has previously suggested that the resetting of arterial baroreflex control of muscle sympathetic nerve activity likely occurs within the central nervous system (and not at the level of the baroreceptors), because this resetting is not extended to baroreflex control of the heart (14). This

raises the possibility of a differential resetting of sympathetic outflow to different vascular beds (e.g., visceral vs skeletal muscle). As such, it is conceivable that sympathetic outflow to skeletal muscle vascular beds is reset to a greater degree than that to the splanchnic or renal vascular beds; hence, there is frank vasodilation in skeletal muscle but negligible changes in vascular tone in splanchnic or renal beds. In addition, we must recognize that, despite clear evidence of reduced sympathetic outflow to skeletal muscle vascular beds, blockade of α -adren-ergic receptors has not been found to reproduce the magnitude of postexercise vasodilation in skeletal muscle (13). Thus some of the skeletal muscle vasodilation postexercise is due to an unknown vasodilator signal that persists in that region for a considerable time postexercise. This vasodilator signal may function in a paracrine fashion, being restricted to skeletal muscle vascular beds and not affecting the splanchnic and renal vascular beds.

Our present data suggest that the splanchnic and renal vascular beds do not contribute directly to postexercise hypotension. However, because there is an absence of vasoconstriction in these vascular beds, despite reductions in arterial and central venous pressure, one can view these vascular beds as being complicitous in the creation of postexercise hypotension. In other words, if one were to lower arterial and central venous pressures to the same degree as seen in postexercise hypotension, profound splanchnic and renal vasoconstriction would be expected. By not vasoconstricting, these vascular beds allow a greater degree of postexercise hypotension than if they had vasoconstricted measurably. Thus, looking deeper, our results suggest that when muscle blood flow is elevated during recovery from exercise, arterial pressure appears to be controlled around a lower pressure by maintaining splanchnic and renal blood vascular tone at near preexercise levels.

It is unclear what other vascular beds might be involved in the elevation of systemic vascular conductance after exercise. Coronary vascular conductance is tightly regulated by myocardial \dot{V}_{O_2} , which correlates with the rate-pressure product. In the present study, rate-pressure product did not differ before ($5,009.5 \pm 230.1$ mmHg·beats/min) vs. 80 min postexercise ($5,256.1 \pm 235.9$ mmHg·beats/min; $P = 0.21$). Therefore, it seems unlikely that the coronary circulation, which only accounts for ~5% of resting cardiac output, is a major contributor of the increased systemic vascular conductance during postexercise hypotension. Similarly, the cerebral circulation does not appear to be a major contributor, because some regions of the brain have diminished blood flow after exercise (35). In a companion paper (34), we provide evidence that cutaneous vascular beds in nonglabrous skin do not contribute to postexercise hypotension, but like the splanchnic and renal vasculatures, may be complicitous in the generation of postexercise hypotension.

Our previous calculations indicated that skeletal muscle vascular beds only accounted for up to ~30% of the increase in systemic vascular conductance postexercise. Although these calculations were performed retrospectively on two separate sets of data from two different studies that used different techniques to measure blood flow and cardiac output, both relied on extrapolation to the whole body of muscle blood flow values derived from venous occlusion plethysmography of the calf. It is likely that changes in blood flow in the thigh are substantially greater than in the calf. If so, our prior predictions

could severely underestimate skeletal muscle's contribution to postexercise hypotension. On the basis of the data gathered in this present study, the leg vasculature accounts for 34% of the rise in systemic vascular conductance. Extrapolation of the changes in leg vascular conductance to whole body muscle could account for ~56% of the rise in systemic vascular conductance [extrapolation to whole body muscle is based on the assumption that the legs comprise ~60% of whole body muscle mass (20, 24)]. Renal and splanchnic vascular conductances, although not significantly altered, would account for ~8% of the change, but an additional 36% remains unaccounted for. Thus, whereas our new data suggest a greater contribution from skeletal muscle vasodilation than previously demonstrated, they continue to support the notion that other regions are vasodilated during postexercise hypotension.

Perspectives. If, in fact, baroreflex control of the renal and splanchnic vascular beds is reset subsequent to exercise, what purpose might this serve? It has been suggested that postexercise hypotension plays a significant role in the allowance of plasma volume recovery. Shortly after exercise, plasma volume returns to preexercise levels and, within hours, has exceeded preexercise levels (9). This expansion is accompanied by a decline in free water clearance (36) and by an increase in albumin content, most likely due to an increase in the flow of lymph that occurs postexercise. Hayes et al. (16) reported that infusion of phenylephrine to prevent postexercise hypotension also attenuated the normal recovery of plasma volume over the first 90 min postexercise. This suggests the possibility that postexercise hypotension is an important mechanism in at least the initial stages of plasma volume recovery and expansion. Resetting of baroreflex control of splanchnic and renal vascular beds after a session of moderate, dynamic exercise, and the resulting absence of vasoconstriction, may be important in achieving these plasma volume changes.

Limitations. The magnitude of postexercise hypotension seen in this study was slightly less than seen in our prior studies, despite tight experimental control over most of the factors recognized to modulate postexercise hypotension (11). Thus it is unclear what factor(s) are responsible for this observation. Nonetheless, we were able to document substantial long-lasting vasodilation in terms of both leg and system vascular conductance in this protocol, supporting the notion that our results are comparable with prior studies on postexercise hypotension.

We did not have direct measurements of the sympathetic nerve activity of the splanchnic or renal vascular beds, so we cannot be certain what balance of vasoconstrictor and vasodilator influences are at work. In the absence of net changes in vascular conductance in these regions, it is possible that sympathetic nerve activity to these two vascular beds is, in fact, increased, but that the vasoconstriction is either prevented by changes in vascular transduction or by release of an unidentified vasodilatory signal.

We also did not measure central venous pressure in the present study. Central venous pressure is reduced postexercise (13), and it is established that a decrease in central venous pressure produces splanchnic and renal vasoconstriction via cardiopulmonary baroreflexes. Furthermore, we did not directly assess baroreflex control of the splanchnic or renal vascular beds. This raises the issue that the cardiopulmonary as well as arterial baroreflexes may be reset after exercise, but we

have merely inferred such changes on the basis of measures of vascular tone. Therefore, future studies are needed to assess the response of the splanchnic and renal vascular beds to perturbations in arterial and central venous pressures during postexercise hypotension to address this issue.

Conclusion. In summary, we assessed splanchnic and renal vascular conductances after a bout of moderate-intensity, dynamic exercise. The conductance of both vascular beds was unchanged from preexercise levels. These observations suggest that the previously identified resetting of baroreflex control of skeletal muscle vascular beds likely extends to the renal and splanchnic vascular beds. In essence, although not contributing directly to postexercise hypotension, the lack of baroreflex-mediated vasoconstriction in these vascular beds can be viewed as being complicitous in the generation of postexercise hypotension.

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REFERENCES

1. Baecke JAH, Burema J, and Frijters JER. A short questionnaire for the measurement of habitual physical activity in epidemiological studies. *Am J Clin Nutr* 36: 936–942, 1982.
2. Borg G. Perceived exertion as an indicator of somatic stress. *Scand J Rehabil Med* 2–3: 92–98, 1970.
3. Bratton AC and Marshall EK. A new coupling component for sulfanilamide determination. *J Biol Chem* 128: 537–550, 1938.
4. Castenfors J. Renal function during prolonged exercise. *Ann NY Acad Sci* 301: 151–159, 1977.
5. Cléroutx J, Kouamé N, Nadeau A, Coulombe D, and Lacourcière Y. Baroreflex regulation of forearm vascular resistance after exercise in hypertensive and normotensive humans. *Am J Physiol Heart Circ Physiol* 263: H1523–H1531, 1992.
6. Dill DB and Costill DL. Calculation of percentage changes in volumes of blood, plasma, and red cells in dehydration. *J Appl Physiol* 37: 247–248, 1974.
7. Escourrou P, Raffestin B, Papelier Y, Pussard E, and Rowell LB. Cardiopulmonary and carotid baroreflex control of splanchnic and forearm circulations. *Am J Physiol Heart Circ Physiol* 264: H777–H782, 1993.
8. Gan K, Nishi I, Chin I, and Slutsky AS. On-line determination of pulmonary blood flow using respiratory inert gas analysis. *IEEE Trans Biomed Eng* 40: 1250–1259, 1993.
9. Gillen CM, Lee R, Mack GW, Tomaselli CM, Nishiyasu T, and Nadel ER. Plasma volume expansion in humans after a single intense exercise protocol. *J Appl Physiol* 71: 1914–1920, 1991.
10. Grimby G. Renal clearances during prolonged supine exercise at different loads. *J Appl Physiol* 20: 1294–1298, 1965.
11. Halliwill JR. Mechanisms and clinical implications of post-exercise hypotension in humans. *Exerc Sport Sci Rev* 29: 65–70, 2001.
12. Halliwill JR, Dineno FA, and Dietz NM. α -Adrenergic vascular responsiveness during postexercise hypotension in humans. *J Physiol* 550: 279–286, 2003.
13. Halliwill JR, Minson CT, and Joyner MJ. Effect of systemic nitric oxide synthase inhibition on postexercise hypotension in humans. *J Appl Physiol* 89: 1830–1836, 2000.
14. Halliwill JR, Taylor JA, and Eckberg DL. Impaired sympathetic vascular regulation in humans after acute dynamic exercise. *J Physiol* 495: 279–288, 1996.

15. Halliwill JR, Taylor JA, Hartwig TD, and Eckberg DL. Augmented baroreflex heart rate gain after moderate-intensity, dynamic exercise. *Am J Physiol Regul Integr Comp Physiol* 270: R420–R426, 1996.
16. Hayes PM, Lucas JC, and Shi X. Importance of post-exercise hypotension in plasma volume restoration. *Acta Physiol Scand* 169: 115–124, 2000.
17. Hirsch AT, Levenson DJ, Cutler SS, Dzau VJ, and Creager MA. Regional vascular responses to prolonged lower body negative pressure in normal subjects. *Am J Physiol Heart Circ Physiol* 257: H219–H225, 1989.
18. Johnson BD, Beck KC, Proctor DN, Miller J, Dietz NM, and Joyner MJ. Cardiac output during exercise by the open circuit acetylene washin method: comparison with direct Fick. *J Appl Physiol* 88: 1650–1658, 2000.
19. Kohl HW, Blair SN, Paffenbarger RS Jr, Macera CA, and Kronenfeld JJ. A mail survey of physical activity habits as related to measured physical fitness. *Am J Epidemiol* 127: 1228–1239, 1988.
20. Lawler LA, Halliwill JR, Summer JM, Joyner MJ, and Mulvagh SL. Leg mass and lower body negative pressure tolerance in men and women. *J Appl Physiol* 85: 1471–1475, 1998.
21. Miki K, Yoshimoto M, and Tanimizu M. Acute shifts of baroreflex control of renal sympathetic nerve activity induced by treadmill exercise in rats. *J Physiol* 548: 313–322, 2003.
22. Minson CT, Wladkowski SL, Pawelczyk JA, and Kenney WL. Age, splanchnic vasoconstriction, and heat stress during tilting. *Am J Physiol Regul Integr Comp Physiol* 276: R203–R212, 1999.
23. Nagashima K, Wu J, Kavouras SA, and Mack GW. Increased renal tubular sodium reabsorption during exercise-induced hypervolemia in humans. *J Appl Physiol* 91: 1229–1236, 2001.
24. Rein H and Schneider M. *Einführung in die Physiologie des Menschen*. Berlin: Springer-Verlag, 1956.
25. Rowell LB. *Human Cardiovascular Control*. New York: Oxford University Press, 1993.
26. Rowell LB, Blackmon JR, and Bruce RA. Indocyanine green clearance and estimated hepatic blood flow during mild to maximal exercise in upright man. *J Clin Invest* 43: 1677–1690, 1964.
27. Rowell LB, Blackmon JR, Martin RH, Mazzarella JA, and Bruce RA. Hepatic clearance of indocyanine green in man under thermal and exercise stresses. *J Appl Physiol* 20: 384–394, 1965.
28. Rowell LB, Detry JR, Blackmon JR, and Wyss C. Importance of the splanchnic vascular bed in human blood pressure regulation. *J Appl Physiol* 32: 213–220, 1972.
29. Rowell LB, Detry JR, Profant GR, and Wyss C. Splanchnic vasoconstriction in hyperthermic man—role of falling blood pressure. *J Appl Physiol* 31: 864–869, 1971.
30. Rowell LB, Marx HJ, Bruce RA, Conn RD, and Kusumi F. Reductions in cardiac output, central blood volume, and stroke volume with thermal stress in normal men during exercise. *J Clin Invest* 45: 1801–1816, 1966.
31. Senitko AN, Charkoudian N, and Halliwill JR. Influence of endurance exercise training status and gender on postexercise hypotension. *J Appl Physiol* 92: 2368–2374, 2002.
32. Stout RL, Wessel HU, and Paul MH. Pulmonary blood flow determined by continuous analysis of pulmonary N₂O exchange. *J Appl Physiol* 38: 913–918, 1975.
33. Suzuki M, Sudoh M, Matsubara S, Kawakami K, Shiota M, and Ikawa S. Changes in renal blood flow measured by radionuclide angiography following exhausting exercise in humans. *Eur J Appl Physiol* 74: 1–7, 1996.
34. Wilkins BW, Minson CT, and Halliwill JR. Regional hemodynamics during postexercise hypotension. II. Cutaneous circulation. *J Appl Physiol* 97: 2071–2076, 2004.
35. Williamson JW, McColl R, and Mathews D. Changes in regional cerebral blood flow distribution during postexercise hypotension in humans. *J Appl Physiol* 96: 719–724, 2004.
36. Yang RC, Mack GW, Wolfe RR, and Nadel ER. Albumin synthesis after intense intermittent exercise in human subjects. *J Appl Physiol* 84: 584–592, 1998.