Is postexercise hypotension related to excess postexercise oxygen consumption through changes in leg blood flow?

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Williams, Jay T., Mollie P. Pricher, and John R. Halliwill. Is postexercise hypotension related to excess postexercise oxygen consumption through changes in leg blood flow? *J Appl Physiol* 98: 1463–1468, 2005. First published December 17, 2004; doi:10.1152/japplphysiol.01211.2004.—After a single bout of aerobic exercise, oxygen consumption remains elevated above preexercise levels [excess postexercise oxygen consumption (EPOC)]. Similarly, skeletal muscle blood flow remains elevated for an extended period of time. This results in a postexercise hypotension. The purpose of this study was to explore the possibility of a causal link between EPOC, postexercise hypotension, and postexercise elevations in skeletal muscle blood flow by comparing the magnitude and duration of these postexercise phenomena. Sixteen healthy, normotensive, moderately active subjects (7 men and 9 woman, age 20–31 yr) were studied before and through 135 min after a 60-min bout of upright cycling at 60% of peak oxygen consumption. Resting and recovery $\dot{V}O_2$ were measured with a custom-built dilution hood and mass spectrometer-based metabolic system. Mean arterial pressure was measured via an automated blood pressure cuff, and femoral blood flow was measured using ultrasound. During the first hour postexercise, $\dot{V}O_2$ was increased by $11 \pm 2\%$, leg blood flow was increased by $51 \pm 18\%$, leg vascular conductance was increased by $56 \pm 19\%$, and mean arterial pressure was decreased by 2.2 ± 1.0 mmHg (all $P < 0.05$ vs. preexercise). At the end of the protocol, $\dot{V}O_2$ remained elevated by $4 \pm 2\%$ ($P < 0.05$), whereas leg blood flow, leg vascular conductance, and mean arterial pressure returned to preexercise levels (all $P > 0.7$ vs. preexercise). Taken together, these data demonstrate that EPOC and the elevations in skeletal muscle blood flow underlying postexercise hypotension do not share a common time course. This suggests that there is no causal link between these two postexercise phenomena.

metabolism; hemodynamics; skeletal muscle; recovery

AFTER A SINGLE BOUT OF DYNAMIC exercise, a reduction in arterial pressure is maintained for nearly 2 h in healthy individuals (21, 25). This postexercise hypotension is consistently elicited after 30- to 60-min bouts of moderate intensity [50–60% peak aerobic capacity (peak oxygen consumption; $\dot{V}O_{2\ peak}$)] exercise, whereas shorter or less vigorous exercise elicits inconsistent changes in arterial pressure in normotensive subjects (13, 21). In most subjects, postexercise hypotension is due to a persistent rise in systemic vascular conductance that is not completely offset by increases in cardiac output (21, 23–25), although some exceptions exist [e.g., endurance-trained men (36)]. Whereas sustained vasodilation has been partially linked to a reduced sympathetic neural outflow to skeletal muscle vascular beds and reduced vascular responsiveness to a given sympathetic outflow (15, 22, 24), additional factors appear to mediate the persistent vasodilation during postexercise hypotension (21, 23, 29).

Skeletal muscle blood flow increases with exercise because of local events in the muscle that continually regulate blood flow to meet the changing metabolic demands (37). Immediately after the cessation of intense muscle activity, skeletal muscle blood flow may be as high as 10–15 times the resting value (37), revealing the high flow capacity of active human muscles (33). A single bout of dynamic exercise also generates an elevated postexercise oxygen uptake ($\dot{V}O_2$) (2, 3, 16, 26, 30). This increase in postexercise $\dot{V}O_2$ is commonly referred to as excess postexercise $\dot{V}O_2$ (EPOC) and is described as the excess $\dot{V}O_2$ above that required to support resting metabolic processes after exercise (16). Several factors have been implicated in the generation of EPOC, including, the metabolism of lactate and replenishment of creatine phosphate (5, 7, 8, 14, 16, 19, 26, 30, 35), muscle glycogen resynthesis (8), increased body temperature (6, 9, 12, 20), increased heart rate (12), increased ventilatory rate (19), increased circulating concentrations of catecholamine hormones (4, 10, 11, 17), and replenishing the body's resting oxygen levels (18, 39).

Skeletal muscle blood flow remains elevated several hours postexercise, for reasons that remain unidentified and via an unknown mechanism. It is possible that this elevation is related to meeting the metabolic demands associated with EPOC. Along these lines, regional oxygen delivery and utilization are the product of blood flow and the arteriovenous oxygen difference (i.e., the Fick equation) (33). Muscular exercise is accompanied by increases in skeletal muscle blood flow from ~1.2 l/min at rest to 20–25 l/min during maximal exercise (37). However, the exact vasodilator mechanism or mechanisms underlying exercise hyperemia remain elusive. It is likely that vasodilator metabolites formed by active skeletal muscles act on the resistance vessels in their vicinity to raise local blood flow to match metabolic demands (34). It seems plausible that the sustained elevation of skeletal muscle blood flow after exercise may similarly subserve a continued elevation in skeletal muscle $\dot{V}O_2$. If so, then elevated oxidative metabolism may be one of the causes of the sustained vasodilation in skeletal muscle vascular beds that underlies postexercise hypotension.

Therefore, this study was undertaken to examine the potential association between EPOC and the sustained increase in skeletal muscle blood flow during postexercise hypotension. Specifically, we tested the hypothesis that EPOC would correlate closely with the changes in leg blood flow during recovery from a single bout of dynamic exercise in humans.

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METHODS

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

Subjects

A total of 16 healthy, moderately active, nonsmoking, normotensive subjects between the ages of 20 and 31 yr participated in this study (7 men, 9 women). 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

Subjects reported to the laboratory for a screening day scheduled at least 2 days before the study day. Subjects reported for this visit at least 2 h postprandial and abstained from caffeine, alcohol, and exercise for 24 h before this visit. Subjects performed an incremental bicycle exercise test (Lode Excaliber, Groningen, The Netherlands) comprised of 1-min workload increments to determine $\dot{V}O_{2\,peak}$. 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 $\dot{V}O_2$ uptake 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 (rating of perceived exertion = 19–20) within 12–16 min. 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 $\dot{V}O_{2\,peak}$. This workload was used on the study day for the 60-min exercise bout. Subjects self-reported activity levels on two questionnaires (1, 27). Finally, subjects were instructed to ingest the temperature sensing pill the night before reporting to the laboratory for the study day visit. Subject characteristics are presented in Table 1.

Study Day Protocol

Subjects reported for the study between 6 and 10 AM at least 8 h postprandial. Subjects abstained from caffeine at least 8 h and from alcohol and exercise 24 h before the study. Ingestion of a temperature-sensing pill (HQInc, Palmetto, FL) the evening before the study was used to assess internal temperature as an index of core body temperature. After visiting the restroom, subjects were instrumented on a table for measurement of heart rate via a five-lead electrocardiogram (model Q710, Quinton Instruments Bothell, WA) and arterial pressure via an automated blood pressure monitor (Dinamap Pro100 vital signs monitor, Critikon, Tampa, FL).

Table 1. *Subject characteristics*

Age, y	21.7±2.6
BMI, kg/m^2	23.8±3.7
$\dot{V}O_{2\,peak}$, ml·kg^{-1}·min^{-1}	40.4±7.4
Exercising workload, W	127.1±42.2
Exercising heart rate, beats/min	139.3±15.7
Percentage of heart rate reserve, %	64.9±13.5
Baecke sport index, arbitrary units	9.3±2.2
Index of physical activity, MET·h/wk	158.4±86.5

Values are means ± SD; n = 16 subjects. BMI, body mass index; $\dot{V}O_{2\,peak}$, peak oxygen consumption; MET, metabolic equivalents.

Measurements. Preexercise and postexercise measurements were made with subjects in the supine position and consisted of consecutive recordings of heart rate, arterial pressure, internal temperature, and leg blood flow every 15 min for 60 min preexercise and 135 min postexercise.

$\dot{V}O_2$ was measured continuously using a custom-built dilution hood and mass spectrometer-based metabolic system (Parvomedics, Sandy, UT). Ambient air was drawn through a loosely fitting hood past the subject's nose and mouth at a rate sufficient to prevent escape of expired gas (i.e., up to 60 l/min). The rate was adjusted to maintain flow-through carbon dioxide concentrations between 1 and 1.5%.

Femoral artery diameter and velocity were measured by using an ultrasound probe (10-MHz linear 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 and were corrected for the angle of insonation. Leg blood flow was calculated as artery cross-sectional area multiplied by femoral mean blood velocity, doubled to represent both legs, and reported as milliliters per minute. Leg vascular conductance was calculated as flow for both legs divided by mean arterial pressure and expressed as milliliters per minute per millimeters of mercury.

Exercise. Subjects exercised upright on a stationary cycle at 60% of $\dot{V}O_{2\,peak}$ for 60 min. Exercise of this intensity and duration consistently produces a sustained (~2 h) postexercise hypotension in healthy normotensive subjects (21). Heart rate, arterial pressure, and internal temperature were recorded every 15 min throughout the exercise period. During exercise, subjects drank 15 ml of water/kg of initial body weight to offset volume loss.

Statistics

The results were analyzed with repeated-measures ANOVA. 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

Exercise

During exercise, heart rate increased from 55.0 ± 2.2 to 139.3 ± 4.0 beats/min (average over 60 min; $P < 0.05$). The goal was to have each subject exercise for 60 min at 60% $\dot{V}O_{2\,peak}$. The percentage of heart rate reserve (heart rate reserve is defined as maximal heart rate achieved during $\dot{V}O_{2\,peak}$ testing minus the resting supine heart rate) reached during exercise (64.9 ± 3.5%) was consistent with the target workload. Systolic blood pressure increased from 108.0 ± 2.8 mmHg during supine rest to 141.3 ± 4.4 mmHg ($P < 0.05$), whereas diastolic blood pressure was unchanged (60.8 ± 1.6 vs. 64.3 ± 2.2 mmHg) over the 60-min bout of exercise. Therefore, mean arterial pressure increased from 77.4 ± 1.5 mmHg during supine rest to 90.7 ± 8.2 mmHg during exercise ($P < 0.05$). Internal body temperature increased from 36.7 ± 0.1°C during supine rest to 37.9 ± 0.1°C by the final min of exercise ($P < 0.05$).

Postexercise

Figure 1 shows mean arterial pressure, whole body $\dot{V}O_2$, and leg blood flow preexercise and through 135 min postexercise. Notably, whole body $\dot{V}O_2$ was increased by 11% during the first hour postexercise compared with preexercise ($P < 0.05$). This elevated $\dot{V}O_2$ was maintained throughout the study protocol, such that $\dot{V}O_2$ at the end of the protocol remained 4%
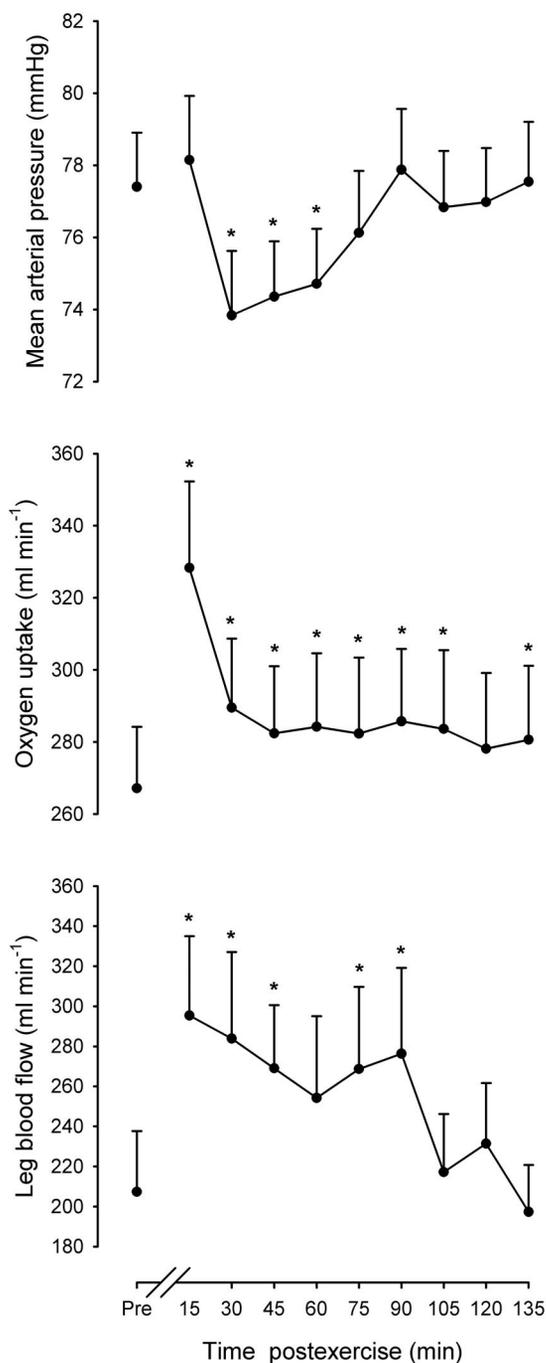
Fig. 1. Mean arterial pressure (*top*), whole body oxygen uptake (*middle*), and leg blood flow (*bottom*) before (Pre) and after exercise. Values are means ± SE; $n = 16$ subjects. * $P < 0.05$ vs. preexercise.

tance was elevated during recovery from exercise but returned to preexercise levels by the end of the protocol.

Figure 2 shows the simultaneous values for $\dot{V}_{O_2}$ vs. leg blood flow throughout the protocol. At 15 min postexercise, both leg blood flow and $\dot{V}_{O_2}$ exhibited high values. However, by 30 min postexercise, there was a substantial decline in $\dot{V}_{O_2}$ with little change in leg blood flow. Through the remainder of the protocol, leg blood flow progressively returned to resting preexercise levels, but $\dot{V}_{O_2}$ remained elevated.

Figure 3 shows the preexercise to postexercise change in mean arterial pressure and leg vascular conductance vs. the change in $\dot{V}_{O_2}$ across individuals for the representative time point of 45 min postexercise. Individual responses were variable. The change in mean arterial pressure ranged from -11.4 to $+3.7$ mmHg, the change in leg vascular conductance ranged from -27.2 to $+209.4\%$, and the change in $\dot{V}_{O_2}$ ranged from -18.0 to $+26.1\%$ at this time point. There did not appear to be an association between the degree of pressure reductions and elevations in $\dot{V}_{O_2}$ ($r = 0.40$, $P = 0.13$). Similarly, there did not appear to be an association between the degree of leg vasodilation and elevations in $\dot{V}_{O_2}$ ($r = 0.37$, $P = 0.16$).

DISCUSSION

The goal of this study was to determine whether an association exists between postexercise hypotension and EPOC. In our study, moderately active subjects were examined to test the hypothesis that EPOC would correlate closely with the changes in leg blood flow over time during recovery from exercise, suggesting a link between the elevated postexercise metabolism and hyperemia in the previously active skeletal muscle (21, 37, 40). In contrast to our hypothesis, no association between these two postexercise phenomena was observed; thus they appear to be unrelated.

We assessed changes in metabolism via changes in $\dot{V}_{O_2}$. These data indicate that the preceding exercise caused a modest but prolonged increase in metabolism, requiring an additional 2.86 ± 0.93 liters of oxygen through 135 min of recovery. This observation is consistent with prior reports on EPOC (2, 3, 16, 26, 30). Several factors have been implicated in the generation of EPOC, including the metabolism of lactate and replenishment of creatine phosphate (5, 7, 8, 14, 16, 19, 26, 30, 35), muscle glycogen resynthesis (8), increased body temperature (6, 9, 12, 20), increased heart rate (12), increased ventilatory rate (19), increased circulating concentrations of catecholamine hormones (4, 10, 11, 17), and replenishing the body's resting oxygen levels (18, 39). Some of the metabolic products created during exercise whose oxidation may contribute to the EPOC may also stimulate increases in blood flow to previously active skeletal muscle (38), creating a "postexercise hyperemia."

Our subjects demonstrated a reduction in blood pressure through 60 min after the cessation of exercise consistent with prior reports on postexercise hypotension (21–25). Underlying this postexercise hypotension, we observed an elevated leg blood flow through 90 min postexercise. This rise in skeletal muscle blood flow after exercise is well documented but poorly understood. Although there is clear evidence of reduced sympathetic outflow to skeletal muscle vascular beds in humans (15, 24) and rats (28) during postexercise hypotension, blockade of α-adrenergic receptors is unable to reproduce the mag-

greater than preexercise values ($P < 0.05$). Leg blood flow increased from 207 ± 30 ml/min before exercise to 280 ± 39 ml/min during the first hour postexercise ($P < 0.05$). By 105 min postexercise, leg blood flow decreased to within preexercise levels ($P = 0.82$).

Table 2 presents combined hemodynamic and metabolic observations at several key time points during the protocol. Notably, arterial pressures were decreased at 45 min postexercise relative to preexercise ($P < 0.05$) but returned to preexercise levels by the end of the protocol. Leg vascular conduc-

Table 2. *Hemodynamics and metabolism before and after exercise*

	Preexercise	15 min Postexercise	45 min Postexercise	135 min Postexercise
Heart rate, beats/min	55.0 ± 2.2	65.9 ± 2.9*	61.1 ± 2.6*	56.1 ± 2.4
Systolic arterial pressure, mmHg	108.0 ± 2.8	109.4 ± 2.5	104.3 ± 2.4*	109.3 ± 3.1
Diastolic arterial pressure, mmHg	60.8 ± 1.6	62.5 ± 2.1	59.4 ± 1.6	61.8 ± 1.8
Mean arterial pressure, mmHg	77.4 ± 1.5	78.1 ± 1.8	74.7 ± 1.5*	77.5 ± 1.7
Leg blood flow, ml/min	207 ± 30	295 ± 40*	269 ± 31*	197 ± 24
Leg vascular conductance, $ml\cdot min^{-1}\cdot mmHg^{-1}$	2.78 ± 0.47	3.76 ± 0.49*	3.60 ± 0.38*	2.52 ± 0.28
$\dot{V}O_2$, ml/min	267 ± 17	328 ± 24*	282 ± 19*	281 ± 21*
$\dot{V}CO_2$, ml/min	228 ± 15	306 ± 27*	223 ± 15	224 ± 15
Respiratory exchange ratio	0.85 ± 0.01	0.92 ± 0.02*	0.79 ± 0.01*	0.80 ± 0.01*
Internal temperature, °C	36.7 ± 0.1	37.2 ± 0.1*	36.9 ± 0.1*	36.8 ± 0.1

Values are means $\pm$ SE; $n = 16$ subjects; $\dot{V}O_2$, oxygen consumption; $\dot{V}CO_2$, carbon dioxide production. *$P < 0.05$ vs. preexercise.

nitude of postexercise vasodilation in skeletal muscle. There is evidence of vascular α-adrenergic hyporesponsiveness in rats (32), but α_1- and α_2-adrenergic vascular responsiveness is intact in humans (22). Finally, although nitric oxide contributes to postexercise vasodilation in rats (31), independent inhibition of either nitric oxide synthase (23) or cyclooxygenase (29) does not reduce the postexercise vasodilation in humans. Thus additional factors appear to mediate the persistent vasodilation during postexercise hypotension and could be related to ongoing release of metabolic signals from the previously exercised muscle.

We have considered the possibility that this elevation in blood flow to previously active muscles could subserve a continued demand for oxygen delivery and perhaps be linked to EPOC. However, our data appear inconsistent with this model for the following reasons. First, if the elevation in leg blood flow were related to an increase in oxygen utilization in the leg, we would have expected a linear relation between $\dot{V}O_2$ and leg blood flow, which we did not find (Fig. 2). Second, individual differences in the metabolic and hemodynamic responses to exercise exist (Fig. 3), yet we found no association between changes in $\dot{V}O_2$ and changes in either arterial pressure or leg vascular conductance after exercise. In fact, some pairs of subjects had very similar reductions in mean arterial pressure despite dissimilar metabolic responses after the exercise.
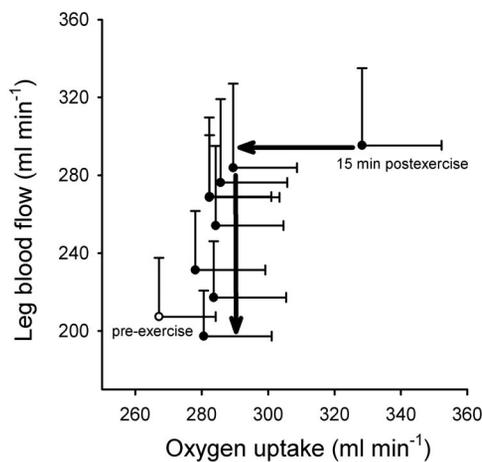
Some subjects exhibited very high EPOC with virtually no reductions in blood pressure, whereas others had significant blood pressure reductions with no elevation in $\dot{V}O_2$. Taken together, these data suggest that EPOC and the elevation in skeletal muscle blood flow underlying postexercise hypotension are not associated and that it is unlikely there is a causal link between these two postexercise phenomena.

We did not measure the arteriovenous oxygen difference across the leg vascular bed, so we cannot be certain that
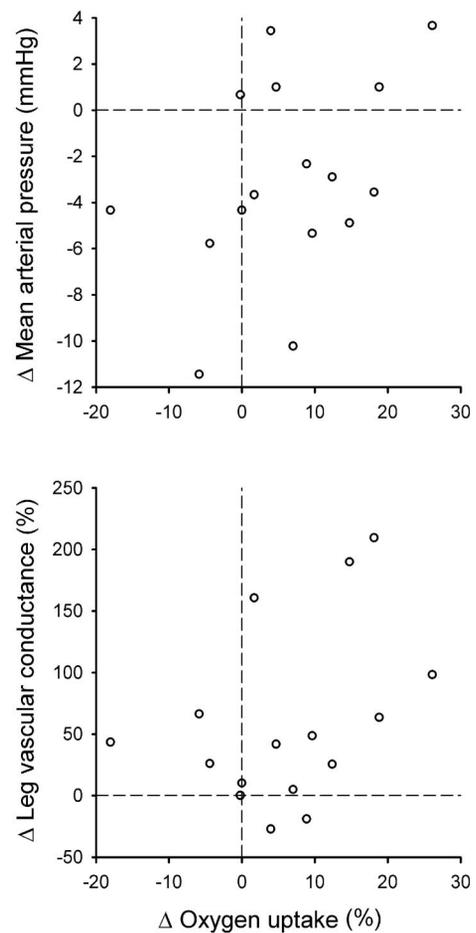

Fig. 2. Leg blood flow vs. oxygen uptake across time. ○, Preexercise; ●, measurements every 15 min postexercise. Superimposed arrows indicate the time course of postexercise measurements, moving from 15 to 30 min postexercise and from 30 to 135 min postexercise. Values are means $\pm$ SE; $n = 16$ subjects.

Fig. 3. *Top*: individual change (Δ) in mean arterial pressure vs. percent change in oxygen uptake from preexercise to 45 min postexercise. *Bottom*: individual percent change in leg vascular conductance vs. percent change in oxygen uptake from preexercise to 45 min postexercise. Values are means $\pm$ SE; $n = 16$ subjects.

changes in leg blood flow were not accompanied by simultaneous changes in local $\dot{V}_{O_2}$. On the basis of the Fick equation relating $\dot{V}_{O_2}$, blood flow, and the arteriovenous oxygen difference, and our observation that leg blood flow returns to resting values while $\dot{V}_{O_2}$ remained elevated, the only way the excess oxygen could be utilized by the legs would be if there were a progressive rise in the arteriovenous oxygen difference over the 135-min postexercise period. Because this seems highly unlikely, it would appear that the sustained rise in $\dot{V}_{O_2}$ was the result of an increased oxygen utilization in some other vascular bed and not in the previously exercised skeletal muscles of the leg.

We were able to document vasodilation in terms of leg vascular conductance in this protocol, despite what can be considered only a modest postexercise hypotension. The magnitude of postexercise hypotension is greater in hypertensive individuals than the normotensive subjects we studied, and we note that our conclusions regarding the absence of an association between EPOC and postexercise hypotension may be limited to healthy individuals of average fitness. Thus it could be that in different populations (e.g., older individuals with hypertension), elevated metabolism during recovery from exercise contributes to the vasodilation that underlies postexercise hypotension.

Conclusion

In summary, we assessed the potential relationship between postexercise hypotension and EPOC after a bout of moderate-intensity, dynamic exercise. Our observations suggest that the elevated leg blood flow after exercise is not the result of an increased oxygen utilization in the previously exercised skeletal muscle of the legs. These data also demonstrate that EPOC and the elevations in skeletal muscle blood flow underlying postexercise hypotension do not share a common time course. This suggests that there is no causal link between these two postexercise phenomena.

REFERENCES

1. **Baeke 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. **Bahr R, Ingnes I, Vaage O, Sejersted OM, and Newsholme EA.** Effect of duration of exercise on excess postexercise O_2 consumption. *J Appl Physiol* 62: 485–490, 1987.
3. **Bahr R and Sejersted OM.** Effect of feeding and fasting on excess postexercise oxygen consumption. *J Appl Physiol* 71: 2088–2093, 1991.
4. **Barnard JR and Foss ML.** Oxygen debt: effect of beta-adrenergic blockade on the lactacid and alactacid components. *J Appl Physiol* 27: 813–816, 1969.
5. **Bassett DR Jr, Merrill PW, Nagle FJ, Agre JC, and Sampedro R.** Rate of decline in blood lactate after cycling exercise in endurance-trained and -untrained subjects. *J Appl Physiol* 70: 1816–1820, 1991.
6. **Brehm BA and Gutin B.** Recovery energy expenditure for steady state exercise in runners and nonexercisers. *Med Sci Sports Exerc* 18: 205–210, 1986.
7. **Brooks GA, Brauner KE, and Cassens RG.** Glycogen synthesis and metabolism of lactic acid after exercise. *Am J Physiol* 224: 1162–1166, 1973.
8. **Brooks GA and Gaesser GA.** End points of lactate and glucose metabolism after exhausting exercise. *J Appl Physiol* 49: 1057–1069, 1980.
9. **Brooks GA, Hittelman KJ, Faulkner JA, and Beyer RE.** Temperature, liver mitochondrial respiratory functions, and oxygen debt. *Med Sci Sports Exerc* 3: 72–74, 1971.
10. **Cain SM.** Exercise O_2 debts of dogs at ground level and at altitude with and without β-block. *J Appl Physiol* 30: 838–843, 1971.
11. **Cain SM and Chapler CK.** Effects of norepinephrine and α-block on O_2 uptake and blood flow in dog hindlimb. *J Appl Physiol* 51: 1245–1250, 1981.
12. **Claremont AD, Nagle F, Reddan WD, and Brooks GA.** Comparison of metabolic, temperature, heart rate and ventilatory responses to exercise at extreme ambient temperatures (0° and 35°C). *Med Sci Sports Exerc* 7: 150–154, 1975.
13. **Cléroux 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.
14. **Di Prampero PE, Peeters L, and Margaria R.** Alactic O_2 debt and lactic acid production after exhausting exercise in man. *J Appl Physiol* 34: 628–632, 1973.
15. **Floras JS, Sinkey CA, Aylward PE, Seals DR, Toren PN, and Mark AL.** Postexercise hypotension and sympathoinhibition in borderline hypertensive men. *Hypertension* 14: 28–35, 1989.
16. **Gaesser GA and Brooks GA.** Metabolic bases of excess post-exercise oxygen consumption: a review. *Med Sci Sports Exerc* 16: 29–43, 1984.
17. **Gladden LB, Stainsby WN, and MacIntosh BR.** Norepinephrine increases canine skeletal muscle $\dot{V}_{O_2}$ during recovery. *Med Sci Sports Exerc* 14: 471–476, 1982.
18. **Gutierrez G, Lund N, Acero AL, and Marini C.** Relationship of venous P_{O_2} to muscle P_{O_2} during hypoxemia. *J Appl Physiol* 67: 1093–1099, 1989.
19. **Hagberg JM, King DS, Rogers MA, Montain SJ, Jilka SM, Kohrt WM, and Heller SL.** Exercise and recovery ventilatory and $\dot{V}_{O_2}$ responses of patients with McArdle's disease. *J Appl Physiol* 68: 1393–1398, 1990.
20. **Hagberg JM, Mullin JP, and Nagle FJ.** Effect of work intensity and duration on recovery O_2. *J Appl Physiol* 48: 540–544, 1980.
21. **Halliwill JR.** Mechanisms and clinical implications of post-exercise hypotension in humans. *Exerc Sport Sci Rev* 29: 65–70, 2001.
22. **Halliwill JR, Dinenno FA, and Dietz NM.** α-Adrenergic vascular responsiveness during postexercise hypotension in humans. *J Physiol* 550: 279–286, 2003.
23. **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.
24. **Halliwill JR, Taylor JA, and Eckberg DL.** Impaired sympathetic vascular regulation in humans after acute dynamic exercise. *J Physiol* 495: 279–288, 1996.
25. **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.
26. **Hill AV and Lupton H.** Muscular exercise, lactic acid and the supply and utilization of oxygen. *QJM* 16: 135–171, 1923.
27. **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.
28. **Kulics JM, Collins HL, and DiCarlo SE.** Postexercise hypotension is mediated by reductions in sympathetic nerve activity. *Am J Physiol Heart Circ Physiol* 276: H27–H32, 1999.
29. **Lockwood JM, Pricher MP, Wilkins BW, Holowatz LA, and Halliwill JR.** Postexercise hypotension is not explained by a prostaglandin-dependent peripheral vasodilation. *J Appl Physiol* 98: 447–453, 2004.
30. **Margaria R, Edwards HT, and Dill DB.** The possible mechanisms of contracting and paying the oxygen debt and the role of lactic acid in muscular contraction. *Am J Physiol* 106: 689–715, 1933.
31. **Patil RD, DiCarlo SE, and Collins HL.** Acute exercise enhances nitric oxide modulation of vascular response to phenylephrine. *Am J Physiol Heart Circ Physiol* 265: H1184–H1188, 1993.

32. **Rao SP, Collins HL, and DiCarlo SE.** Postexercise α-adrenergic receptor hyporesponsiveness in hypertensive rats is due to nitric oxide. *Am J Physiol Regul Integr Comp Physiol* 282: R960–R968, 2002.

33. **Rowell LB.** Control of blood flow to dynamically active muscles. In: *Human Cardiovascular Control*. New York: Oxford University Press, 1993, p. 255–301.

34. **Rowell LB.** Ideas about control of skeletal muscle and cardiac muscle blood flow (1876–2003): cycles of revision and new vision. *J Appl Physiol* 97: 384–392, 2004.

35. **Rowell LB, Kraning KK II, Evans TO, Kennedy JW, Blackmon JR, and Kusumi F.** Splanchnic removal of lactate and pyruvate during prolonged exercise in man. *J Appl Physiol* 21: 1773–1783, 1966.

36. **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.

37. **Shepherd JT.** Circulation to skeletal muscle. In: *Handbook of Physiology. The Cardiovascular System. Peripheral Circulation and Organ Blood Flow*. Bethesda, MD: Am. Physiol. Soc., 1983, sect. 2, vol. III, pt. 1, chapt. 11, p. 319–370.

38. **Shepherd JT and Vanhoutte PM.** *The Human Cardiovascular System: Facts and Concepts*. New York: Raven, 1979.

39. **Stainsby WN and Barclay JK.** Exercise metabolism: O_2 deficit, steady level O_2 uptake and O_2 uptake for recovery. *Med Sci Sports Exerc* 2: 177–181, 1970.

40. **Van Beekvelt MCP, Shoemaker JK, Tschakovsky ME, Hopman MTE, and Hughson RL.** Blood flow and muscle oxygen uptake at the onset and end of moderate and heavy dynamic forearm exercise. *Am J Physiol Regul Integr Comp Physiol* 280: R1741–R1747, 2001.