Postexercise hypotension causes a prolonged perturbation in esophageal and active muscle temperature recovery

Glen P. Kenny,1 Ollie Jay,1 Wytek M. Zaleski,3 Mark L. Reardon,2 Ronald J. Sigal,2 W. Shane Journeay,1 and Francis D. Reardon1

1Faculty of Health Sciences, School of Human Kinetics, and 2Department of Medicine, University of Ottawa, Ottawa, Ontario, Canada; 3London Health Sciences Centre, London, Ontario, Canada; and 4Department of Veterinary Biomedical Sciences, Western College of Veterinary Medicine, University of Saskatchewan, Saskatoon, Canada

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Postexercise hypotension causes a prolonged perturbation in esophageal and active muscle temperature recovery. Am J Physiol Regul Integr Comp Physiol 291: R580–R588, 2006. First published March 2, 2006; doi:10.1152/ajpregu.00918.2005.—We examined the effect of two levels of exercise-induced hypotension on esophageal (Tes) and active and nonactive muscle temperatures during and following exercise. Seven males performed an incremental isotonic test on a KinCom isokinetic apparatus to determine their peak oxygen consumption during bilateral knee extensions (VO2peak). This was followed on separate days by 15-min of isolated bilateral knee extensions at moderate (60% VO2peak) (MEI) and high (80% VO2peak) (HEI) exercise intensities, followed by 90 min of recovery. Muscle temperature was measured with an intramuscular probe inserted in the left vastus medialis (Tvm) and triceps brachii (Ttb) muscles under ultrasound guidance. The deepest sensor (tip) was located ~10 mm from the femur and deep femoral artery and from the superior ulnar collateral artery and humerus for the Tvm and Ttb, respectively. Additional sensors were located 15 and 30 mm from the tip with an additional sensor located at 45 mm for the Tvm measurements only. Following exercise, mean arterial pressure (MAP) remained significantly below preexercise rest, was observed at the end of exercise for both HEI and MEI, and throughout 90 min of recovery for MEI. The magnitude of this decrease in MAP is more pronounced and prolonged subsequent to exercise of increasing intensity (29, 30). These findings suggest that the magnitude and duration of the postexercise esophageal temperature elevation are associated with the marked cardiovascular changes that occur after dynamic exercise.

After cessation of exercise, there are changes in the factors that determine mean arterial pressure (MAP), which result in hypotension that is both vascular and neural in origin (17, 25). MAP decreases rapidly during the early stages of recovery, as do stroke volume and cardiac output, whereas total peripheral resistance tends to increase toward preexercise resting values (6). The magnitude of this decrease in MAP is more pronounced and prolonged subsequent to exercise of increasing intensity (13) and is greater with the orthostatic influence of upright posture (17, 25, 38). Postexercise hypotension is thought to occur in part as a result of venous pooling in the previously active musculature (7, 34, 38). It has been shown that resistance vessels in skeletal muscle remain dilated after a bout of cycling exercise, and the resultant hyperemia persists well into recovery (37). Such pooling of blood in the lower extremities tends to reduce cardiac filling and unload baroreceptors (17). Changes in hemodynamic response, such as an increase in stroke volume and MAP, induced by the application of positive pressure to the lower limbs in the upright position postexercise were shown to reverse the postexercise increase in the esophageal temperature at which onset of cutaneous vasodilation and sweating occurs (22), and 2) favor the decay of esophageal temperature to preexercise values (23). Thus the perturbations result in a prolonged elevation in postexercise esophageal temperature (28, 41) paralleled by a rapid decrease in sweating (28), skin blood flow, and skin temperature (28, 41) to preexercise baseline values within the early stages of recovery. Kenny and Neidre (28) showed that the magnitude of increase in esophageal temperature increases with exercise intensity. At higher exercise intensity, they observed 1) a greater decrease in postexercise mean arterial pressure (MAP); 2) an overall decrease in the rate of heat loss; and, 3) an increase in the esophageal temperature recovery time. More recently, studies have shown that exercise results in a residual increase in the esophageal temperature threshold at which onset for skin blood flow (29) and sweating (30) are initiated. This effect is also relatively greater during recovery after exercise of increasing intensity (29, 30). These findings suggest that the magnitude and duration of the postexercise esophageal temperature elevation are associated with the marked cardiovascular changes that occur after dynamic exercise.

Recovery from dynamic exercise has been shown to result in significant perturbations of thermoregulatory control. These

Address for reprint requests and other correspondence: G. P. Kenny, Univ. of Ottawa, School of Human Kinetics, 125 Univ. Priv., Montpetit Hall (367), PO Box Stn. A, Ottawa, ON, Canada.

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same conditions that give rise to these cardiovascular phenomena postexercise appear to attenuate heat loss during the postexercise period, eliciting a prolonged elevation in esophageal temperature.

In an attempt to better understand the timeline response of the prolonged esophageal temperature elevation, recent studies examined the nonactive (i.e., contralateral inactive leg) (31) and active (33) muscle tissue temperature responses after a single bout of moderate-intensity single leg (31) and bilateral knee (33) extension exercise. It was shown that during isolated muscle activity, convective heat transfer by the blood to muscle tissue results in a significant residual heat load in both nonactive (31, 33) and active (33) muscle during subsequent recovery. These studies showed that postexercise decay of esophageal temperature is influenced by convective heat transfer between both the previously active (33) and nonactive (31) muscles, which may increase recovery time to reestablish normal resting temperatures. These observations provide preliminary evidence in support of previous suggestions that during sustained postexercise hypotension, the postexercise esophageal temperature is a direct function of the residual heat load of previously active musculature (22, 28). To date, however, the effect of exercise intensity on postexercise tissue temperature responses has not been explored. Moreover, there remains a paucity of information relating the temperature response between exercised and nonexercised tissue compartments. In this study, two exercise intensities were used to elicit different magnitudes of postexercise hypotension (28), during which muscle temperature and the decay of esophageal temperature were measured. We hypothesized that after exercise, the elicited decrease in MAP would result in an increase in the postexercise esophageal temperature and that of inactive and active muscle and that these effects will be exacerbated by exercise of greater intensity.

METHODS

Subjects. Subsequent to approval of the project by the University of Ottawa Human Research Ethics Committee, seven healthy males consented to participate in the study. Mean values ± SD of the subject’s age, height, body mass, VO₂peak during bilateral concentric knee extensions and body fat content were 25 ± 5 years, 1.8 ± 0.4 m, 86.8 ± 6.1 kg, 2.3 ± 0.8 l/min and 12.7 ± 2.6%, respectively.

Instrumentation. In each trial, esophageal temperature (Tₑ) was measured using a pediatric nasopharyngeal thermocouple temperature probe (Size 9Fr, Mon-a-therm, Mallinckrodt Medical, Hazelwood, MO) inserted through a nostril, into the esophagus to the level of the heart. Regional muscle temperature was measured by a flexible multisensor intramuscular temperature probe (Physitemp Instruments, Clifton, NJ, model IT-17:3, thermal constant of 0.25 s) inserted into the vastus medialis and triceps brachii (model IT-17:3) muscles by ultrasound guidance (32). Using an aseptic technique, we anesthetized the skin, subcutaneous tissue, and muscle to a maximum depth of 50 mm by infiltrating ~2 ml of 1% lidocaine without epinephrine, using a 25-gauge needle. The tip of this needle was placed at the proposed site for the deepest temperature probe. We used the anesthetic needle as a guide and inserted an 18-gauge, 50-mm polyethylene catheter (Cathlon; Critikon, Markham, ON, Canada) into the anesthetized tract to the required depth. The anesthetic needle and the catheter stylet were withdrawn, and the temperature probe was inserted into the catheter shaft. When the probe was fully inserted into the vastus medialis, the catheter was withdrawn, leaving the tip of the temperature probe ~10 mm (Tvån) equidistant from the femur and deep femoral artery (Tvån+10) with 3 sensors located at 15 (Tvån+25), 30 (Tvån+40), and 45 (Tvån+55) mm from the tip. The implant site was approximately midway between and medial to a line joining the anterior superior iliac spine and the superior aspect of the midline of the patella. In the case of the triceps brachii, the deepest temperature sensor (Tvån+10) was positioned at ~10 mm from the humerus and superior ulnar collateral artery with two sensors located at 15 (Tvån+25) and 30 (Tvån+40) mm from the tip. The implant site was approximately midway and medial to a line joining the acromion process of the scapula and olecranon process of the ulna. The catheter was withdrawn and the final position of the probe was verified using the ultrasound imaging.

The average depth of the probe from the surface was 47.9 mm and within 11.4 mm of both the deep femoral artery and femur and 34.2 mm and within 11.5 mm of both the humerus and superior ulnar collateral artery for Tvån and Tvår, respectively. The probe assembly was secured to the skin with sterile, waterproof transparent dressing (3M 1622W Tegaderm transparent dressing) and tape (total surface coverage ~25 cm²). The Tegaderm transparent dressing consisted of a thin polyethylene membrane coated with a layer of an acrylic adhesive. The dressing, which is permeable to both water vapor and oxygen, is impermeable to microorganisms, and once in position, it provides an effective barrier to external contamination.

The internal position of the temperature probe relative to the skin surface was calculated based on the ratio of the known depth of the probe (r) from the skin surface measured by ultrasound imaging and the radius of the thigh (ru). Thus r/ru is the relative radius (9, 11, 33). Although it was not possible to verify the final position of the probe after completion of the experimental trial, the length of the probe within limb tissue was measured during removal of the probe. The depth of the probe was verified with the preexperiment depth as determined by ultrasound imaging.

Skin temperature was monitored at 12 sites using Type T thermocouples integrated into heat flow sensors (Concept Engineering, Old Saybrook, CT). The area-weighted mean skin temperature (Tₛ) and heat flux (HFₛ) were calculated by assigning the following regional percentages: head 6%, upper arm 9%, forearm 6%, finger 2%, chest 19%, upper back 9.5%, lower back 9.5%, anterior thigh 10%, posterior thigh 10%, anterior calf 9.5%, posterior calf 9.5% (20). Temperature and heat flux data were collected and digitized (data acquisition module, model 3497A, Hewlett Packard) at 5-s intervals, simultaneously displayed and recorded in spreadsheet format on a hard disk (model PC-312, 9000, Hewlett Packard).

Heart rate (HR) was monitored using a Polar coded transmitter, recorded continuously, and stored with a Polar Advantage interface and Polar Precision Performance software (Polar Electro Oy, Kempele, Finland). MAP was estimated from the integration of a noninvasive recording of blood pressure at the middle digit of the left hand (Finapres 2300, Ohmeda, Madison, WI) fixed at heart level (the third intercostal space). The Finapres system is based on the Penaz volume clamp method (dynamic unloaded arterial wall principle). MAP was verified periodically throughout the protocol by auscultation.

Pulmonary V̇O₂ was estimated using a metabolic cart (model CPX/D; Medgraphics, St. Paul, MN) during V̇O₂peak assessment preceding the experimental trials. Cardiac output (CO) was estimated using the CPX/D computerized version of the CO₂ rebreathe technique of Defares (8). It has been shown that Doppler-derived aortic blood flow (CO) measurements correlate well with the indirect carbon dioxide rebreathe method (21). The Defares method has also been shown to work well in “unsteady state” testing (16). Each measure took ~20–25 s to perform. Subjects performed one rebreathing protocol per designated time point. Stroke volume (SV) was calculated as CO/HR. Total peripheral resistance (TPR) was calculated as MAP/CO.

Microcirculation in the triceps brachii muscle and skin of the anterior thigh and posterior upper arm was measured continuously by laser-Doppler flowmetry (PeriFlux System 5000, main control unit; PFS5010 LDPM, operating unit; Perimed, Stockholm, Sweden). Mus-
cle microcirculation was measured by means of an optic fiber (a diameter of 0.5 mm and a length of 170 mm) passed through a needle catheter inserted perpendicular to the skin surface. The median depth from the skin surface to the fiber tip in the muscle was 3 cm. For the measurement of skin blood flow, the laser-Doppler flow probes (PR 401 Angled Probe, Perimed) were taped to cleaned skin in an area that superficially did not appear to be highly vascular and from where consistent readings were noted (35). Cutaneous vascular conductance (CVC) was calculated throughout the experimental protocol by using the ratio of 30-s averages of laser-Doppler flux and MAP.

Sweat rate was estimated from a 5.0 cm² ventilated capsule placed on the upper back. Anhydrous compressed air was passed through the capsule over the skin surface at a rate of 1 l/min. Water content of the effluent air was measured at known barometric pressure using the readings from an Omega HX93 humidity and temperature sensor (Omega Engineering, Stamford, CT). Sweat rate was calculated from the product of the difference in water content between effluent and influent air and the flow rate. This value was normalized for the skin surface area under the capsule and expressed in milligrams per minute per centimeters square.

Experimental protocol. Subjects performed an incremental isotonic test (constant angular velocity, increases in force output) on the Kin-Com isokinetic apparatus to determine their maximal capacity (V\textsubscript{O₂peak}). The exercise consisted of bilateral, concentric knee extension over a range of 70° from perpendicular with the subject in an upright seated position (hip angle between 90° and 110°) and the long axis of the thigh in the horizontal plane. The force output was increased by 15 N every 2 min until fatigue, while the angular velocity was maintained at 58.3°/s throughout the test. The results of the test were used to establish the work rate for the subsequent experimental submaximal exercise trials.

Each subject performed a total of two experimental trials carried out in random order. The experimental trials were conducted in the morning after a 24-h period without heavy or prolonged physical activity. Upon arrival at the laboratory at 0800, subjects clothed in shorts and athletic shoes were appropriately instrumented. On separate days, subjects rested in an upright seated position for 90 min at an ambient temperature of 25°C, of which the final 15 min were recorded as representative of the baseline resting values. At 2 min before exercise, the subjects were secured to the Kin-Com isokinetic apparatus at the level of the torso and ankles. Subjects then performed 15 min of exercise as described above consisting of bilateral, concentric knee extension at a moderate exercise intensity (MEI; 60% V\textsubscript{O₂peak}) and heavy exercise intensity (HEI; 80% V\textsubscript{O₂peak}) on a Kin-Com followed by 90 min of recovery.

Statistical analysis. The data were first evaluated using a two-way repeated-measures ANOVA. The repeated factors of exercise intensity (levels: MEI and HEI) and postexercise recovery time (levels: 0, 10, 30, and 90 min) were used with the dependent variables of changes from preexercise rest in T\textsubscript{es}, T\textsubscript{vm10}, T\textsubscript{vm25}, T\textsubscript{vm40}, T\textsubscript{vm55}, T\textsubscript{tb10}, T\textsubscript{tb25}, T\textsubscript{tb40}, CO, SV, HR, MAP, TPR, T\textsubscript{a}, H\textsubscript{F}ak, thigh CVC, forearm CVC, triceps brachii muscle blood flow, and sweat rate.

Subsequent analyses of the muscle temperature profiles across the vastus medialis and triceps brachii were conducted by comparing the relationship of muscle temperature with probe depth at 0, 10, 30, and 90 min of postexercise recovery to that observed at preexercise rest at each exercise intensity separately; and, 2) 0, 10, 30, and 90 min of postexercise recovery between exercise intensities. For this purpose, we used forward stepwise multiple regression with the following general linear models

\[
T_{vm} \text{ or } T_{tb} (at \ MEI \ or \ HEI) = \beta_0 + \beta_1 [\text{Probe Depth}] + \beta_2 [\text{Experimental Stage}] + \beta_3 [\text{Experimental Stage} \cdot \text{Probe Depth}] \quad (1)
\]

\[
T_{vm} \text{ or } T_{tb} (at \ 0, 10-, 30-, \ and \ 90-min \ postexercise \ recovery) = \beta_0 + \beta_1 [\text{Probe Depth}] + \beta_2 [\text{Exercise Intensity}] + \beta_3 [\text{Exercise Intensity} \cdot \text{Probe Depth}] \quad (2)
\]

where probe depth was represented by the distance in millimeters from the deep femoral artery and femur for T\textsubscript{vm}; and superior ulnar collateral artery and humerus for T\textsubscript{tb}. Experimental stage was represented by a dummy variable (1 for preexercise rest, 2 for the selected data for comparison, i.e., 0, 10, 30, or 90 min postexercise recovery). Exercise intensity was represented by a dummy variable (1 for MEI, 2 for HEI).

Post hoc comparisons were performed using paired-sample t-tests. All analyses were performed using the statistical software package SPSS 11.5 for Windows (SPSS Chicago, IL) and JMP 6.0 for Windows (SAS Institute, Cary, NC). The level of significance was set at an alpha level of 0.05.

RESULTS

Esophageal temperature response. During recovery from exercise, the elevation seen in T\textsubscript{es} from preexercise rest became less with postexercise recovery time [F(1,7, 10.2) = 103.6, P < 0.001] and was significantly different between HEI and MEI trials [F(1,6) = 21.6, P = 0.004]. The elevation in T\textsubscript{es} from preexercise rest remained significantly higher after HEI than MEI (P ≤ 0.05) throughout the 90-min postexercise recovery period. At the end of postexercise recovery, T\textsubscript{es} returned to 0.28°C (SD 0.15) above preexercise rest for HEI, but to 0.05°C (SD 0.09) above preexercise rest for MEI. However, the interaction between exercise intensity and recovery time [F(2.7, 16.2) = 3.5, P = 0.045] demonstrates that the rate of T\textsubscript{es} decay after HEI was greater than after MEI. The mean T\textsubscript{es} responses at HEI and MEI are given in Figs. 1 and 2.

Muscle temperature: effect of exercise intensity. After exercise, elevations in vastus medialis temperature from preexercise rest were significantly greater after HEI compared with MEI for T\textsubscript{vm10} [F(1,6) = 17.9, P = 0.006], T\textsubscript{vm25} [F(1,6) = 28.0, P = 0.002], T\textsubscript{vm40} [F(1,6) = 55.8, P < 0.001] and T\textsubscript{vm55} [F(1,6) = 40.9, P < 0.001]. Despite the temperature elevations becoming less with postexercise recovery time for T\textsubscript{vm10} [F(2.3, 14.0) = 92.5, P < 0.001], T\textsubscript{vm25} [F(2.3, 13.5) = 84.7, P < 0.001], T\textsubscript{vm40} [F(2.3, 14.0) = 142.1, P < 0.001] and T\textsubscript{vm55} [F(2.0, 11.9) = 127.6, P < 0.001], the significant differences between exercise intensities were sustained throughout the 90-min postexercise recovery period for all four measurement sites (P ≤ 0.05). At the end of recovery from HEI, vastus medialis temperature returned to +0.90°C (SD 0.20) for T\textsubscript{vm10}, +1.05°C (SD 0.43) for T\textsubscript{vm25}, +1.22°C (SD 0.49) for T\textsubscript{vm40}, and +1.53°C (SD 0.51) for T\textsubscript{vm55} of preexercise rest values. For MEI, values returned to +0.70°C (SD 0.28) for T\textsubscript{vm10}, +0.80°C (SD 0.36) for T\textsubscript{vm25}, +0.89°C (SD 0.50) for T\textsubscript{vm40}, and +0.91°C (SD 0.42) for T\textsubscript{vm55} of preexercise rest temperature at the end of recovery. However, the significant interactions between exercise intensity and recovery time for T\textsubscript{vm10} [F(3.5, 21.2) = 8.2, P = 0.001], T\textsubscript{vm25} [F(2.4, 14.5) = 8.9, P = 0.002] and T\textsubscript{vm40} [F(3.0, 17.9) = 8.9, P = 0.001] demonstrates that rate of muscle temperature decay was significantly greater after HEI than MEI but not for T\textsubscript{vm55} [F(2.9, 17.1) = 1.4, P = 0.273]. Mean T\textsubscript{es} responses at HEI and MEI are given in Fig. 1.

The elevations in triceps brachii temperature from preexercise rest after exercise were significantly greater after HEI than
Regression analysis using Eq. 1 showed that muscle temperature significantly increased with increasing probe depth ($\beta_1$) and with progressing experimental stage ( Hacker $\beta_3$), as evidenced by the significantly shallower relationship between muscle temperature and probe depth for MEI after 0 min ($P = 0.031$), 10 min ($P = 0.036$), and 30 min ($P = 0.043$) postexercise recovery but not 90 min ($P = 0.248$); and for HEI after 0 min ($P < 0.001$), 10 min ($P = 0.007$), 30 min ($P < 0.001$), and 90 min ($P = 0.002$) recovery. Comparison of the vastus medialis temperature profiles between exercise intensities at each individual experimental stage using Eq. 2, showed that a significant difference in profile slope did not occur after 0 min ($P = 0.622$), 10 min ($P = 0.843$), and 30 min ($P = 0.069$) postexercise recovery, but after 90 min recovery, muscle temperature profile was significantly shallower after HEI ($P = 0.027$). All models from Eqs. 1 and 2 were significant ($P \leq 0.05$).

Comparison of the muscle temperature profile across the triceps brachii at different points throughout the postexercise period to preexercise rest is given for MEI (Fig. 4A) and HEI (Fig. 4B). Regression analysis using Eq. 1 showed that following MEI, triceps brachii temperature significantly increased with increasing probe depth ($\beta_1$) for all comparisons, but muscle temperature was only significantly increased after 0 min postexercise ($P \leq 0.05$) compared with preexercise rest ($\beta_2$). After HEI, triceps brachii temperature did not increase (Fig. 4B).

**Muscle temperature: postexercise recovery profiles.** Comparison of the muscle temperature profile across the vastus medialis at different points throughout the postexercise period to preexercise rest is given for MEI (Fig. 3A) and HEI (Fig. 3B). Regression analysis using Eq. 1 showed that muscle temperature significantly increased with increasing probe depth ($\beta_1$) and with progressing experimental stage compared with preexercise rest ($\beta_2$) for both MEI and HEI for all comparisons made (all $P \leq 0.05$). Compared with preexercise rest, muscle temperature profile significantly changed with experimental stage ($\beta_3$), as evidenced by the significantly shallower relationship between muscle temperature and probe depth for MEI after 0 min ($P = 0.031$), 10 min ($P = 0.036$), and 30 min ($P = 0.043$) postexercise recovery but not 90 min ($P = 0.248$); and for HEI after 0 min ($P < 0.001$), 10 min ($P = 0.007$), 30 min ($P < 0.001$), and 90 min ($P = 0.002$) recovery. Comparison of the vastus medialis temperature profiles between exercise intensities at each individual experimental stage using Eq. 2, showed that a significant difference in profile slope did not occur after 0 min ($P = 0.622$), 10 min ($P = 0.843$), and 30 min ($P = 0.069$) postexercise recovery, but after 90 min recovery, muscle temperature profile was significantly shallower after HEI ($P = 0.027$). All models from Eqs. 1 and 2 were significant ($P \leq 0.05$).

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significantly with probe depth ($\beta_1$) for any of the comparisons, but muscle temperature did increase significantly ($P \leq 0.05$) with experimental stages progressing from 0 to 30 min post-exercise ($\beta_2$). Compared with preexercise rest, muscle temperature profile did not significantly change with experimental stage ($\beta_3$) for any of the comparisons made for MEI or HEI ($P > 0.05$). Comparison of the triceps brachii temperature profiles between exercise intensities at each individual experimental stage using Eq. 2, showed that a significantly different profile slope did not occur after 0 min ($P = 0.387$), 10 min ($P = 0.667$), 30 min ($P = 0.762$), or 90 min ($P = 0.908$) postexercise recovery.

Skin temperature, dry heat loss, CVC, and sweating. No significant differences were found between HEI and MEI for changes from preexercise rest of $T_{sk}$ and $HF_{sk}$ ($P > 0.05$). Furthermore, $T_{sk}$ and $HF_{sk}$ decreased to levels not significantly greater than preexercise resting values ($P > 0.05$) within 15–20 min of recovery after both HEI and MEI. Similarly, no significant differences were found between HEI and MEI ($P > 0.05$) for changes from baseline of forearm CVC (Fig. 5A), thigh CVC (Fig. 5B) and sweat rate (Fig. 5D). Both forearm and thigh CVC returned to levels not significantly elevated above baseline values ($P > 0.05$) within 15–20 min of recovery after both HEI and MEI. Sweat rate returned to levels not significantly elevated above baseline values ($P > 0.05$) within 20–30 min recovery after both HEI and MEI.

Hemodynamics and triceps muscle blood flow. All hemodynamics data are summarized in Table 1. Change in MAP from preexercise rest was significantly different between exercise intensities [$F(1,6) = 21.0, P = 0.006$]. Despite the changes in MAP from resting values becoming less with postexercise recovery time [$F(9.7, 48.3) = 198.0, P < 0.001$], a significantly lower MAP was observed after HEI compared with MEI after 5-min postexercise and for the remainder of the recovery period ($P \leq 0.05$). After HEI, the MAP of 91 mmHg (SD 2) at the end of 90-min recovery remained significantly below ($P = 0.015$) the preexercise resting MAP of 97 mmHg (SD 2). In contrast, after MEI, MAP was only significantly different from preexercise rest for 60 min of postexercise recovery ($P \leq 0.05$).

Changes from preexercise rest were significantly different after HEI than MEI for HR [$F(1,6) = 72.0, P < 0.001$] and SV [$F(1,6) = 17.0, P = 0.006$]. These changes from resting values became less with postexercise recovery time for both HR [$F(1.9, 11.5) = 290.7, P < 0.001$] and SV [$F(3.5, 21.1) = 53.6, P < 0.001$]; however, the increase in HR from rest remained significantly greater throughout recovery after HEI than MEI ($P \leq 0.05$), and the decrease in SV from rest was significantly greater after HEI than MEI after 20 min of recovery and onward ($P \leq 0.05$). Change in CO from preexercise rest was significantly different between exercise intensities [$F(1,6) = 7.3, P = 0.036$]; however, the rapid recovery...
of CO to preexercise resting values with postexercise recovery time \[F(1.4, 8.3) = 363.4, P < 0.001\] were such that CO was only significantly different between HEI and MEI trials for only the initial 5 min of postexercise recovery \((P \leq 0.05)\).

Change in TPR from preexercise rest was significantly different between exercise intensities \([F(1.6) = 7.8, P = 0.038]\), but these changes became less with postexercise recovery time \([F(2.9, 14.6) = 104.5, P < 0.001]\). Changes in TPR from rest were significantly greater after HEI than MEI for the initial 60 min of postexercise recovery \((P \leq 0.05)\).

Triceps muscle blood flow was not significantly different \((P > 0.05)\) between HEI and MEI after exercise or at any point during postexercise recovery (Fig. 5C). Significantly greater triceps muscle blood flow, compared with preexercise rest, was only observed for the initial 10 min of recovery \((P < 0.05)\).

**DISCUSSION**

The most striking observation from this study was that the greater level of postexercise hypotension, induced by the higher exercise intensity, was paralleled by an increase in the magnitude and duration of the postexercise elevation in esophageal and active muscle temperatures (Figs. 1 and 2). MAP remained significantly below preexercise resting baseline for the initial 60 min of recovery after MEI and for the duration of the 90-min postexercise period after HEI (Table 1). This exercise-dependent hypotension is further supported by the greater relative changes in the postexercise HR, SV, and TPR measured in HEI compared with MEI (Table 1).

Exercise tissue temperature response. We observed an exercise intensity-dependent increase in the magnitude of the end-exercise esophageal and inactive and active muscle temperatures (Figs. 1 and 2). Although the deep active muscle-to-core temperature gradient was reversed at the end of exercise, increasing from \(-0.55^\circ\text{C}\) to \(0.78^\circ\text{C}\) and \(-0.56^\circ\text{C}\) to \(0.94^\circ\text{C}\) for the MEI and HEI conditions, respectively, no significant change was observed for the nonactive muscle-to-core gradient after exercise (i.e., \(-0.95^\circ\text{C}\) to \(-0.91^\circ\text{C}\) and \(-1.02^\circ\text{C}\) to \(-0.98^\circ\text{C}\) for the MEI and HEI conditions, respectively). In comparing the change in muscle-to-core temperature gradients after exercise between intensities, no significant difference was found between MEI and HEI for the active muscle (\(+1.33^\circ\text{C}\) vs. \(+1.50^\circ\text{C}\)) or for the nonactive muscle (\(+0.04^\circ\text{C}\) vs. \(+0.04^\circ\text{C}\)). This may be explained in part by an exercise intensity-dependent increase in muscle tissue conductive heat loss (3, 9). It has been shown that in conjunction with an exercise-induced increase in the muscle-to-skin temperature difference and a concomitant increase in both limb sweat rate and skin blood flow, conductive heat loss from the limb is increased (3, 9). We recorded a greater muscle-to-skin temperature difference in HEI. Specifically, end-exercise muscle temperature recorded in both the active and nonactive (except T_{b40}) muscles was elevated in HEI compared with MEI, while mean or local skin temperatures remained unchanged. In parallel, local heat flux values were significantly elevated for both the active and inactive muscle temperature measurement sites. Thus the greater muscle-to-skin temperature difference during HEI would serve to enhance surface heat dissipation as the heat of the underlying muscle would be rapidly transferred to the surface veins and lost across the limb surface (15).

The temperature profile across the active muscle (i.e., between the superficial and deep regions) during HEI was significantly reduced at end exercise compared with preexercise rest (Fig. 3A). After MEI, a significantly shallower temperature profile across the active muscle was also apparent at end

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**Fig. 5.** Mean forearm skin blood flow (A), thigh skin blood flow (B), triceps muscle blood flow (C), and upper back sweat rate (D) during rest and throughout 90-min postexercise recovery from high (●)– and moderate (○)–intensity exercise. Error bars indicate SD.
exercise compared with preexercise rest; however, this profile was comparatively steeper relative to HEI (Fig. 3B). Our observations are consistent with those of Saltin et al. (39), who showed that temperatures of the mid- and superficial regions of the muscle remained lower than deep muscle temperature during moderate exercise. Kenny et al. (33) showed that the temperature across the radial axis of the muscle became homogenous during moderate-intensity exercise. However, the tissue temperature responses in the present study were measured over a wider radius (as defined by the r/rsk ratio; see Fig. 3) using four temperature sensors as opposed to three sensors (similarly spaced), which placed the outer sensor (Tmem55) in closer proximity to the skin surface. It is possible that an increase in conductive heat loss associated with the exercise-induced increase in thigh skin blood flow (Fig. 5B) would have a more pronounced effect on the superficial region of the muscle compared with the deeper regions (15).

In contrast to the muscle temperature profile of the vastus medialis, no difference in the triceps brachii deep-to-superficial temperature gradient was measured between end-exercise and preexercise rest. Moreover, no differences in the slope were observed between exercise conditions. However, the end-exercise muscle temperatures of the deep and middle regions of the triceps brachii were significantly greater after HEI compared with MEI (Figs. 3A and 3B). The increase in inactive muscle temperature is consistent with the previous observation by Kenny et al. (31), who reported a 0.23°C increase in end-exercise deep muscle temperature of the inactive contralateral thigh during single-leg knee extension exercise. In contrast, previous studies have reported slight decreases or no changes of inactive muscle temperature during dynamic exercise (1, 2) despite a significant increase in both esophageal temperature and muscle temperature of the exercising legs. The lack of temperature increase in the inactive muscle tissue during exercise (1, 2) was thought to indicate a proportional decrease in muscle tissue perfusion. Previous studies have shown a significant skin vasodilation and simultaneous vasoconstriction in inactive muscle tissue (i.e., as measured in the forearm) with rising core temperatures (4, 24). More recently, however, Tanaka et al. (40) showed that blood flow to nonexercising limbs increases during exercise in proportion to the intensity of exercise. We measured a significant elevation in triceps muscle blood flow (Fig. 5C) during both exercise conditions, although no difference was observed between conditions, which was paralleled by an increase in inactive muscle temperature during exercise. The increase in inactive muscle temperature subsequent to the increase in active muscle and esophageal temperatures provides support for the important role that vascular heat transfer plays in the cooling of active tissue (10, 31).

Postexercise tissue temperature response. Previous studies have shown that under thermoneutral conditions, core body temperature remains elevated after an acute bout of exercise (5, 14, 26, 27, 41, 43). This postexercise elevation in core temperature is intensity dependent, with higher postexercise temperatures associated with higher exercise intensities (28). More recent studies demonstrate that the disturbance in postexercise thermal homeostasis appears to be correlated to the marked cardiovascular changes that occur after dynamic exercise. This hypothesis has evolved from the initial observations that an increase in the postexercise hypotensive response, induced by exercise of increasing intensity, was shown to I) result in a relative increase in the onset thresholds for sweating (30) and cutaneous vasodilation (29), II) an overall decrease in the rate of heat loss (28), and III) a concomitant increase in the postexercise core temperature recovery time (28). In the present study, we observed a similar exercise intensity-dependent effect on core temperature recovery. The greater and more prolonged postexercise core temperature measured in HEI was paralleled by I) a similar temperature response recorded in both the active and inactive skeletal muscle regions (except for Tmem55) (Figs. 1 and 2) and II) a sustained decrease in MAP (Table 1). Specifically, MAP remained significantly below preexercise resting values for the first 60 min after MEI and throughout the 90 min recovery for HEI. The magnitude of reduction was greater after HEI for the duration of 90 min. These trends are consistent with other studies that have examined exercise recovery hemodynamics (5, 13, 34).

It has been recently demonstrated that a single bout of dynamic exercise elicits a persistent reduction in arterial pres-
sure lasting nearly 2 h in healthy individuals (17–19, 43), with postexercise hypotension greater and more prolonged after exercise of a greater intensity (13, 36). Postexercise hypotension is thought to be caused by profound changes in the factors that regulate and determine MAP, resulting in hypotension that is both vascular and neural in origin (17). Research indicates that venous and muscle pooling contributes to the postexercise hypotension in an upright seated posture (37, 38). Piepoli et al. (37) showed that resistance vessels in skeletal muscle remain dilated after a bout of dynamic exercise, and the resultant hyperemia persists well into recovery. Thus, during inactive seated recovery, pooling of blood would tend to trap heat in the previously active muscle (37). Our observation of a prolonged elevated temperature of the previously active musculature lends support to this hypothesis. In parallel to the decrease in MAP, we recorded a prolonged elevation in vastus medialis muscle temperature above preexercise rest. The magnitude and duration of the response was greater with HEI.

In conjunction with a rapid reduction in skin blood flow, sweating, and whole body heat loss after the cessation of exercise (28), a time-dependent transfer of heat from previously active muscle to the central core region (as measured by esophageal temperature) would likely contribute to a prolonged elevation of body core temperature (5, 14, 26, 27, 41, 43). Given that we did not observe a difference in the skin blood flow and sweating response postexercise between exercise conditions, the difference in recovery time of esophageal temperature between MEI and HEI is likely to be primarily caused by differences in the rate of convective heat transfer between muscle and blood and between blood and the core region. This is supported by the recent observations of Journey et al. (23) that the reversal of the postexercise hypotension induced by the application of positive pressure to the upper limbs resulted in an increase in whole body heat loss and core temperature decay. Further studies, however, are required to examine the effect of the redistribution of blood volume after exercise on core temperature change.

Upon close examination of the skeletal muscle, temperature profiles (Figs. 3, A and B and 4, A and B), it is evident that the significant hemodynamic adjustments, such as the postexercise decrease in MAP, has an influence on inactive and active muscle temperature profiles. However, the resultant effect is dissimilar in active and inactive skeletal muscle regions. The temperature profile of the vastus medialis (Fig. 3, A and B) was significantly shallower at the beginning of postexercise recovery compared with preexercise rest for both HEI and MEI. Relative to preexercise rest, this deep-to-superficial muscle temperature profile remained significantly shallower throughout the postexercise recovery (after HEI, but after MEI, this profile was only significantly shallower until the midstages of postexercise recovery. Such differences would be expected given that regional muscle temperature at any point in time is a consequence of regional differences in metabolic rate (10), conductive heat loss to adjacent tissue (10, 12), and deep and peripheral convective blood flow (10, 42).

In conclusion, it was shown that the increase in the postexercise hypotensive response, induced by exercise of increasing intensity, was paralleled by a significantly greater and sustained elevation in esophageal and both active and nonactive muscle temperatures throughout 90 min of postexercise recovery. At the beginning of postexercise recovery, active muscle temperature at all four measurement depths were above esophageal temperature for both exercise protocols. The distinct similarity in the magnitude and duration of the esophageal and previously active muscle temperature recovery throughout the postexercise period for both exercise intensity protocols indicates a time-dependent convective transfer to the core of residual heat from hyperemic previously active muscle. This suggests that the postexercise hypotension-induced heat load from previously active muscle plays an integral role in the attenuation of whole body heat loss and the subsequent sustained elevation of core temperature typically observed after exercise.

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