Sex differences in postexercise esophageal and muscle tissue temperature response

Glen P. Kenny and Ollie Jay
Laboratory of Human Bioenergetics and Environmental Physiology, School of Human Kinetics, Faculty of Health Sciences, University of Ottawa, Ottawa, Ontario, Canada

Submitted 11 September 2006; accepted in final form 25 November 2006

Kenny GP, Jay O. Sex differences in postexercise esophageal and muscle tissue temperature response. Am J Physiol Regul Integr Comp Physiol 292: R1632–R1640, 2007. First published November 30, 2006; doi:10.1152/ajpregu.00638.2006.—Factors associated with blood pressure regulation during recovery from exercise dramatically influence core temperature regulation. However, it is unknown whether sex-related differences in postexercise hemodynamics affect core and muscle temperature response. Sixteen participants (8 males, 8 females) completed an incremental isotonic test on a Kin-Com isokinetic apparatus to determine their activity-specific peak oxygen consumption during bilateral knee extensions (VO₂peak). On a separate day, participants performed 15 min of isolated bilateral knee extensions at a moderate (60% VO₂peak) exercise intensity followed by a 90-min recovery. Esophageal temperature (Tₑ), mean arterial pressure (MAP), muscle temperature at four depths in the active vastus medialis (TVₐ), and three depths in the inactive triceps brachii (TVᵢ) were measured concurrently with sweat rate and cutaneous vascular conductance (CVC). Relative to the preexercise resting Tₑ of 36.7°C (SD 0.1), between 10 and 50-min of recovery Tₑ was 0.19°C (SD 0.02) higher for females than males (P = 0.037). All measurements of TVₐ (0.036 > P > 0.014) and TVᵢ (0.048 > P > 0.008) were higher for females during the initial 30 min of recovery by between 0.46°C and 0.64°C for TVₐ and by between 0.53°C and 0.70°C for TVᵢ. In parallel, females showed a 5 to 7 mmHg greater reduction in MAP during recovery relative to males (P = 0.002) and a significantly lower CVC (P = 0.020) and sweat rate (P = 0.034). Therefore, it is concluded that females demonstrate a greater and more prolonged elevation in postexercise esophageal temperature and active and inactive muscle temperatures, which is paralleled by a greater post-exercise hypotensive response.

DURING RECOVERY FROM DYNAMIC exercise, a prolonged elevation in postexercise esophageal temperature is paralleled by a decrease in sweating, skin blood flow, and skin temperature to preexercise baseline values within the early stages of recovery (21, 22, 24, 26, 42). Previous research supports a possible relationship between hemodynamic changes following exercise and the responses facilitating the potential for heat dissipation (19–21, 26, 35).

The majority of the studies investigating the postexercise changes in core temperature and mean arterial pressure (MAP) have been conducted in males. However, while the temporal pattern of postexercise hypotension is similar in both sexes, a more pronounced depression in blood pressure has been reported in females relative to males following exercise bouts of 5 and 35 min duration (2, 8, 25). It has been proposed that females demonstrate a lower responsiveness in systems that regulate arterial pressure, with the predominant mechanism potentially differing between sexes (5). This is supported by the observation of females responding to orthostatic challenges with a vagally mediated heart rate increase, whereas males respond with a greater sympathetic stimulation to the peripheral vasculature (10).

The ability to modulate the rate of heat loss through adjustments in vasoactivity and sudomotor activity is a fundamental mechanism of thermoregulatory homeostasis. Thus, differences in postexercise hemodynamics in females may therefore translate into altered core temperature regulation during recovery. Indeed, a sex-dependent increase in the magnitude of postexercise hypotension has been associated with a significantly greater esophageal temperature onset threshold for cutaneous vasodilation in females (25).

Other work has also endeavored to investigate the association between postexercise hemodynamics and the kinetics of heat exchange between muscle and the core of the body in males. An increase in the postexercise hypotensive response, induced by exercise of increasing intensity, resulted in 1) a concomitant greater elevation in esophageal and both active and inactive muscle temperatures throughout 90 min of post-exercise recovery; and 2) different temperature profiles as measured by a significant decrease in the deep-to-superficial muscle temperature gradient (24). At present, however, it is not known whether the aforementioned sex differences in postexercise hemodynamics similarly influence muscle tissue and core temperatures following dynamic exercise.

Thus, the purpose of this study was to examine possible differences in the postexercise tissue temperature response in active and inactive tissue compartments between males and females. Specifically, we tested the hypothesis that females would demonstrate a greater and more prolonged elevation in postexercise esophageal and muscle tissue temperatures and concurrently a greater reduction in MAP.

METHODS

Participants. Subsequent to approval of the experimental protocol by the University of Ottawa Human Research Ethics Committee, 16 healthy, physically active, nonsmoking normotensive participants (8 males, 8 females) consented to participate in the study. Participants’ characteristics, categorized according to sex are given in Table 1. Female participants were eumenorrheic with regular menstrual cycles, 28 days long. To control for hormonal variation, each female participant was tested during the early follicular phase (2–5 days after the onset of menstruation) of their menstrual cycle.

The costs of publication of this article were defrayed in part by the payment of page charges. The article must therefore be hereby marked “advertisement” in accordance with 18 U.S.C. Section 1734 solely to indicate this fact.
Table 1. Mean participant physical characteristics

<table>
<thead>
<tr>
<th></th>
<th>Males</th>
<th>Females</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, yr</td>
<td>24±3</td>
<td>23±2</td>
</tr>
<tr>
<td>Height, m</td>
<td>1.77±0.41</td>
<td>1.66±0.20*</td>
</tr>
<tr>
<td>Body mass, kg</td>
<td>83.2±5.7</td>
<td>67.9±5.1*</td>
</tr>
<tr>
<td>BSA/body mass, m²/kg</td>
<td>0.024±0.001</td>
<td>0.026±0.001</td>
</tr>
<tr>
<td>Body fat, %</td>
<td>13.0±2.7</td>
<td>14.6±3.3</td>
</tr>
</tbody>
</table>

Maximal oxygen consumption,
VO₂peak, ml·min⁻¹·kg⁻¹  | 50.3±4.4 | 47.0±5.2 |
Maximal oxygen consumption,
VO₂peak, ml·min⁻¹·kg FFM⁻¹ | 57.9±4.7 | 55.0±5.0 |

Data are reported for n = 16 (8 males, 8 females) as means ± SD; BSA, body surface area; FFM, fat-free mass; body fat was determined according to Siri (39); *Significant difference between males and females. Significance to an alpha level of 0.05.

**Instrumentation.** In each trial, esophageal temperature (Tₑₑ) was measured using a pediatric nasopharyngeal thermocouple temperature probe (Size 9 Fr; Mon-a-Therm; Mallinkrodt Medical, St. Louis, MO) inserted through a nostril, into the esophagus, estimated to be positioned at the level of the heart. Regional muscle temperature was measured by a multisensor intramuscular temperature probe inserted into the vastus medialis (model IT-17:4; Physiometrics, Clifton, NJ) and medial head of the triceps brachii (model IT-17:3) muscles by ultrasound guidance.

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. Using the anesthetic needle as a guide, we then inserted an 18-gauge, 50-mm polyethylene catheter (Cathlon, 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 in 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 (TV₅₀₀₀) equidistant from the femur and deep femoral artery with three sensors located at 15 (TV₂₅₀₀), 30 (TV₁₅₀₀), and 45 (TV₃₅₀₀) 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 (TV₁₀₁₀) was positioned at ~10 mm from the humerus and superior ulnar collateral artery with two sensors located at 15 (TV₂₅₁₀) and 30 (TV₁₅₁₀) 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 probe assembly was secured to the skin with sterile, waterproof, transparent dressing and tape.

The internal position of the temperature sensor relative to the skin surface was calculated on the basis of the ratio of the known depth of the probe (r) from the skin surface measured by ultrasound imaging and the radius of the thigh (rtₕ). Thus r₁₀₁₀ is the relative radius (24, 27). At the completion of the experimental trial, the length of the probe within the limb tissue was verified with the preexperimentation depth, as determined by ultrasound imaging.

Skin temperature was measured at 12 sites using Type T thermocouples integrated into heat-flow sensors (Concept Engineering, Old Saybrook, CT). The area-weighted mean skin temperature (Tskin) and mean nonevaporative heat flux (Hₑₑ) 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%, and posterior calf 9.5% (24).

Temperature and heat flux data were collected and digitized (Hewlett Packard data acquisition module, model 3497A) at 5-s intervals and simultaneously displayed and recorded in spreadsheet format on a hard disk (Hewlett Packard, model PC-312, 9000).

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, 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) and fixed at heart level (the third intercostal space). MAP was verified periodically throughout the protocol by auscultation.

Pulmonary VO₂ was estimated using a metabolic cart (model CPX/D; Medgraphics, St. Paul, MN) during maximal capacity (VO₂peak) assessment preceding the experimental trials. Cardiac output (Q) was estimated using the CPX/D computerized version of the CO₂-rebreathing technique of Defares (6). Each measure took ~20–25 s to perform. Participants performed one rebreathing protocol per designated time-point. Stroke volume (SV) was calculated as Q/HR. Total peripheral resistance (TPR) was calculated as MAP/Q.

Muscle microcirculation was measured in the inactive triceps brachii muscle (MbF) only, while skin blood flow was recorded at the nondominant anterior forearm. Muscle and skin blood flow were recorded continuously by laser-Doppler flowmetry (Peri Flux System 5000, main control unit; PF5010 LDPM, operating unit; Perimed AB, Stockholm, Sweden). Muscle microcirculation was measured by an optic fiber (diameter: 0.5 mm, length: 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 30 mm. The probe assembly was secured to the skin surface using the same method as described for the muscle temperature probes. For the measurement of skin blood flow, the laser-Doppler flow probes (PR 401 Angled Probe; Perimed AB, Stockholm, Sweden) were taped to cleaned skin, in an area that superficially, did not appear to be highly vascular and from where consistent readings were noted (32). Cutaneous vascular conductances (CVC) was calculated throughout the experimental protocol by using the ratio of 30-s averages of laser-Doppler flow and MAP and was expressed in terms of percentage of maximum CVC (% of CVCmax). At the end of the experiment, local skin temperature at the skin site was raised to 42°C using a heating element (PF 5020 Temperature Unit; Perimed AB, Stockholm, Sweden), housing the laser-Doppler flow probe. The element was activated until maximum CVC was attained (~30 min). Maximum CVC was determined as a sustained elevated plateau in local skin blood flow. To minimize the occurrence of movement artifacts in the muscle and skin blood recordings, the subject’s left arm was supported with a narrow arm sling secured to the side of the KIN-COM apparatus. The forearm was maintained in 90° flexion with the hand in the pronated position.

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 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 mg·min⁻¹·cm⁻².

**Preliminary testing.** On separate days, body adiposity and VO₂peak were measured. The hydrostatic weighing technique was used to determine body density. Calculation of the percentage of the percentage of body fat was based on the Siri equation (39). VO₂peak was determined using a progressive incremental cycling protocol performed on a Monark cycle ergometer and consisted of continuous cycling at a pedaling cadence of 80 rpm. The resistance was increased by 0.5 kilopond until the subject could no longer maintain the pedaling cadence and/or volitional exhaustion. On the basis of the data, the male and female participants in the present study were...
matched for body composition and physical fitness (based on VO2peak expressed per kilogram of fat-free mass) (Table 1).

Following a minimum of 48 h, participants performed an incremental isotonic test (constant angular velocity, increases in force output) on the KIN-COM isokinetic apparatus (KIN-COM500H; Chatteck, Hixson, TN) to determine their activity-specific peak oxygen consumption (VO2peak). The exercise consisted of synchronous bilateral, concentric knee extensions over a range of 70° from perpendicular with the participant 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 volitional exhaustion and/or until the angular velocity of 58.3°/s could not be maintained. The results of the test were used to establish the work rate for the subsequent experimental submaximal exercise trial.

The experimental trials were conducted between early November and early March. All trials were conducted in the morning after a 24-h period without heavy or prolonged physical activity. Upon arrival at the laboratory at 0800, participants dressed in shorts and athletic shoes were appropriately instrumented. Each participant 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 participant was secured to the KIN-COM isokinetic apparatus at the level of the torso and ankles. Each participant then performed 15 min of exercise, as described above consisting of synchronous bilateral, concentric knee extension at 60% of VO2peak. The workload resistance was adjusted for each participant according to their individual mechanical efficiency of the leg-extension exercise (average was 11.2% for males and 10.7% for females). Exercise was followed by 90 min of recovery. At the end of each exercise bout, CVCmax was determined using the local heating protocol described above.

Statistical analysis. The data were evaluated using a mixed model design ANOVA. The nonrepeated factor of sex (Levels: male and female) and the repeated factor of postexercise recovery time (Levels: 5, 10, 20, 30, 50, 70, and 90 min) were employed with the dependent variables of changes from preexercise rest in Te, Tvm10, Tvm25, Tvm40, Tvm55, Tbr10, Tbr25, Tbr40, Q, SV, HR, MAP, TPR, % of CVCmax, triceps brachii Mfib and upper back sweat rate; and absolute Ta and HFa.

Significant main effects were analyzed further with post hoc tests for between-sex comparisons and independent-sample t-tests for between-sex comparisons. All analyses were performed using the statistical software package SPSS 11.5 for Windows (SPSS Chicago, IL). The level of significance was set at 0.05 and the alpha level was adjusted by the Bonferroni correction method (P ≤ 0.05/n-1; n = number of comparisons).

RESULTS

Postexercise hemodynamics. Change in MAP from preexercise rest was significantly different between sexes (F1,14 = 15.5, P = 0.002), and, regardless of sex, increased significantly with postexercise recovery time (F2,9,41,1 = 216.6, P < 0.001). The interaction between sex and recovery time (F2,9,41,1 = 6.7, P = 0.001) was evidenced by greater decrease in MAP for females between 5 min and 70 min of postexercise recovery (Table 2).

Changes from preexercise rest were significantly different between sexes for HR (F1,14 = 25.2, P < 0.001) and SV (F1,14 = 14.7, P = 0.002). These changes from resting values became less with postexercise recovery time for both HR (F2,9,41,4 = 535.1, P < 0.001) and SV (F4,4,58.0 = 130.9, P < 0.001). Heart rate was significantly more elevated for females after 20 min postexercise and for the remainder of recovery, and SV was significantly lower for females after 20 min postexercise and for the remainder of recovery (Table 2). Change in Q from preexercise rest was not significantly different between sexes (F1,14 = 0.1, P = 0.718) but became rapidly less with postexercise recovery time (F1,5,21,0 = 724.6, P < 0.001) (Table 2). Change in TPR from preexercise rest was significantly different between sexes (F1,14 = 4.9, P = 0.044) and changes in TPR due to exercise became progressively less with postexercise recovery time (F4,7,65.6, = 320.1, P < 0.001). A significant difference between sexes in TPR was observed between 5 min and 50 min of recovery time (Table 2).

Esophageal temperature response. No significant difference was observed in the preexercise resting Te, of 36.74°C (SD 0.16) for TVM10, 35.88°C (SD 0.31) for TVM25, 35.70°C (SD 0.38) for TVM40, and 35.35°C (SD 0.34) for TVM55. For females, resting values were 35.99°C (SD 0.22) for TVM10.

Table 2. Mean changes in MAP, Q, SV, HR, and TPR for males and females at preexercise rest, following exercise, and throughout 90-min postexercise recovery

<table>
<thead>
<tr>
<th>Measure</th>
<th>Sex</th>
<th>Preexercise</th>
<th>End exercise</th>
<th>5-min</th>
<th>10-min</th>
<th>20-min</th>
<th>30-min</th>
<th>50-min</th>
<th>70-min</th>
<th>90-min</th>
</tr>
</thead>
<tbody>
<tr>
<td>MAP, mmHg</td>
<td>M</td>
<td>96 (2)</td>
<td>+15 (4)</td>
<td>-3 (3)</td>
<td>-5 (2)</td>
<td>-7 (3)</td>
<td>-8 (2)</td>
<td>-4 (2)</td>
<td>-2 (1)</td>
<td>-2 (2)</td>
</tr>
<tr>
<td>Q, l/min</td>
<td>F</td>
<td>92 (2)</td>
<td>+17 (6)</td>
<td>-8 (3)**</td>
<td>-10 (3)*</td>
<td>-12 (3)*</td>
<td>-13 (3)*</td>
<td>-11 (3)*</td>
<td>-8 (2)*</td>
<td>-2 (2)</td>
</tr>
<tr>
<td>SV, ml</td>
<td>M</td>
<td>58.0 (2.1)</td>
<td>+10.9 (1.3)</td>
<td>+1.1 (0.3)</td>
<td>+0.8 (0.5)</td>
<td>+0.7 (0.3)</td>
<td>+0.5 (0.2)</td>
<td>+0.4 (0.2)</td>
<td>+0.2 (0.2)</td>
<td>+0.2 (0.2)</td>
</tr>
<tr>
<td>HR, beats/min</td>
<td>F</td>
<td>59.0 (3.0)</td>
<td>+10.8 (1.2)</td>
<td>+1.6 (0.5)</td>
<td>+1.1 (0.4)</td>
<td>+0.6 (0.3)</td>
<td>+0.5 (0.3)</td>
<td>+0.3 (0.3)</td>
<td>+0.3 (0.4)</td>
<td>+0.1 (0.4)</td>
</tr>
<tr>
<td>TPR, mmHg</td>
<td>M</td>
<td>66.4 (3.8)</td>
<td>+16.8 (1.0)</td>
<td>-18.5 (6.4)</td>
<td>-17.5 (6.9)</td>
<td>-19.6 (9.9)*</td>
<td>-20.2 (10.3)*</td>
<td>-21.7 (10.4)*</td>
<td>-20.2 (10.0)*</td>
<td>-17.2 (11.1)*</td>
</tr>
<tr>
<td>1/min</td>
<td>F</td>
<td>60.5 (4.0)</td>
<td>+24.8 (8.3)</td>
<td>+27 (8)</td>
<td>+24 (7)</td>
<td>+24 (7)</td>
<td>+24 (6)</td>
<td>+24 (6)</td>
<td>+24 (6)</td>
<td>+24 (6)</td>
</tr>
</tbody>
</table>

Data are reported for n = 16 (8 males, 8 females) as means (SD). *Significant difference between sexes. MAP, mean arterial pressure; Q, cardiac output; SV, stroke volume; HR, heart rate; TPR, total peripheral resistance.
35.70°C (SD 0.14) for TVM25, 35.37°C (SD 0.31) for TVM40, and 35.07°C (SD 0.30) for TVM55. No significant differences between sexes was observed for any of these preexercise resting values. Elevations in vastus medialis temperature from preexercise rest were significantly greater for females for TVM10 ($F_{1,14} = 7.8$, $P = 0.014$), TVM25 ($F_{1,14} = 6.1$, $P = 0.027$), TVM40 ($F_{1,14} = 5.4$, $P = 0.036$), and TVM55 ($F_{1,14} = 6.1$, $P = 0.027$). Furthermore, elevation in vastus medialis temperature became less with postexercise recovery time for TVM10 ($F_{2.4,33.3} = 175.4$, $P < 0.001$), TVM25 ($F_{2.2,31.0} = 188.2$, $P < 0.001$), TVM40 ($F_{2.8,39.9} = 231.1$, $P < 0.001$), and TVM55 ($F_{3.7,51.8} = 292.5$, $P < 0.001$). Changes in vastus medialis temperature from preexercise rest were significantly greater for females than males between 5 and 70 min postexercise for TVM10 (Fig. 1B), between 20 and 50 min postexercise for TVM25, between 20 and 30 min postexercise for TVM40 and TVM55. Within sex, TVM10, TVM25, TVM40, and TVM55 were all significantly elevated above preexercise resting values throughout the 90-min postexercise recovery period for both males and females. Mean TVM responses for males and females are given in Fig. 2, A–D.

Inactive muscle temperature (triceps brachii). Preexercise resting triceps brachii temperatures for males were 35.76°C (SD 0.75) for TTB10, 35.43°C (SD 0.77) for TTB25, and 34.91°C (SD 0.76) for TTB55. No significant differences between sexes was observed for any of these preexercise resting values. The changes in triceps brachii temperature were not significantly different between males and females. The mean responses for males and females are given in Fig. 2, A–D.
No significant differences were found between sexes for preexercise resting values. Elevations in triceps brachii temperature from preexercise rest were significantly greater for females for $T_{TB10}$ $(F_{1,14} = 4.7, P = 0.048)$, $T_{TB25} (F_{1,14} = 9.6, P = 0.008)$, and $T_{TB40} (F_{1,14} = 8.2, P = 0.012)$. Furthermore, elevation in triceps brachii temperature changed significantly with postexercise recovery time for $T_{TB10}$ $(F_{1,27.0} = 80.0, P < 0.001)$, $T_{TB25} (F_{2,2.30.2} = 31.2, P < 0.001)$, and $T_{TB40} (F_{1,8,24.8} = 27.1, P < 0.001)$. A significantly greater elevation for females was observed between 0 and 10 min of recovery for $T_{TB10}$ (Fig. 1C), between 0 and 30 min recovery for $T_{TB25}$, and between 0 and 20 min recovery for $T_{TB40}$. Within sex, females remained significantly elevated above preexercise resting values for 10, 20, and 30 min of postexercise recovery for $T_{TB10}$, $T_{TB25}$, and $T_{TB40}$, respectively. However, males remained significantly elevated above rest for 5 min of recovery for $T_{TB10}$ and $T_{TB25}$, and for 20 min of recovery for $T_{TB40}$. Mean $T_{TB}$ responses for males and females are given in Fig. 3.

Skin temperature, non-evaporative heat loss, forearm CVC, triceps muscle blood flow, and upper back sweating. No significant differences were found between males and females for $T_{sk}$ $(F_{1,14} = 3.7, P = 0.075)$ and HF$_{sk}$ $(F_{1,14} = 3.6, P = 0.078)$. Furthermore, $T_{sk}$ $(F_{3,2,44.8} = 42.1, P < 0.001)$ and HF$_{sk}$ $(F_{3,6,50.9} = 121.7, P < 0.001)$ decreased with recovery time. Mean skin temperature remained significantly greater than preexercise resting values for 50 and 70 min of recovery for males and females, respectively (Fig. 4A). Mean non-evaporative heat loss remained significantly greater than preexercise resting values for 70 min of recovery for both males and females (Fig. 4B). Percentage of maximum forearm CVC was influenced by both sexes $(F_{1,10} = 7.6, P = 0.020)$ and postexercise recovery time $(F_{2,3,22.9} = 388.7, P < 0.001)$, with significantly greater changes from baseline observed for males for between 5 and 20 min postexercise (Fig. 5A). No significant differences were found between males and females for changes from preexercise rest of triceps MbF (Fig. 5B). Sweat rate was significantly different between sex $(F_{1,10} = 6.0, P = 0.034)$ and became less with recovery time $(F_{4,4,44.3} = 330.5, P < 0.001)$, with males showing a greater sweat rate at end-exercise and between 30 and 50 min postexercise recovery (Fig. 5C).

**DISCUSSION**

The major findings of this investigation were that females demonstrated significantly greater and more prolonged elevations in postexercise $T_{es}$ and deep-to-superficial active and inactive muscle temperatures relative to males. In parallel, females also exhibited a significantly greater and more prolonged decrease in postexercise MAP, as well as significantly greater decreases in forearm CVC and upper back sweating.

**End-exercise response.** Consistent with previous reports (25, 33), the elevation in $T_{es}$ at the end of exercise was similar between sexes. Furthermore, a significantly greater elevation from preexercise rest was observed for females at end exercise in the superficial, intermediate, and deep muscle temperature measurement sites of the inactive triceps brachii. This was in contrast to the active muscle temperature measurements taken in the vastus medialis where no significant differences were observed between sexes at the end of exercise. The origin of these apparent differences in end-exercise inactive muscle temperatures between sexes may be regional variance in 1) deep and peripheral convective blood flow, and/or, 2) conductive heat exchange between adjacent tissue (7). Sex-related differences in end-exercise MbF of the inactive triceps brachii
were not observed in the present study; however, in line with previous literature (25), a significantly greater forearm skin blood flow did occur in males at the end of exercise. This potentially suggests a greater convective heat transfer from deeper tissues to the periphery in males leading to smaller elevations in inactive muscle temperature. Even though elevations in active muscle temperature tended to be greater in females at the end of exercise (Fig. 1B), no significant differences were found between the sexes, possibly suggesting a less prominent sex difference in active MbF. This interpretation, however, must be considered with the reservation that there are several sources of variation in triceps MbF (as evidenced by the large standard deviations in Fig. 5B) due to the measurement method employed. These include 1) variable baseline MbF values, which influence the relative changes expressed in terms of percentage change; 2) MbF standardization could not be performed; and 3) although the investigators used an arm sling to immobilize the arm, movement artifacts are possible.

Postexercise response. During the postexercise recovery period, forearm CVC and sweating of the upper back approached preexercise resting levels within the early stages of recovery, despite a prolonged elevation in esophageal and active and inactive muscle tissue temperatures for both sexes. As such, these findings are consistent with a disturbance in core temperature regulation following the cessation of exercise (21, 22, 24, 26, 42). Other studies have provided evidence indicating that the magnitude of heat loss responses and therefore, tissue temperature responses, appears to be correlated to the marked reduction in MAP that occurs after dynamic exercise (19–21). This notion is further supported presently by the observation of a marked reduction in MAP in parallel to the postexercise disturbance in core temperature homeostasis. A particularly novel finding, however, is that a significantly greater and more prolonged elevation in esophageal, active vastus medialis and inactive triceps brachii temperatures occurs in females relative to males. This is also paralleled by a greater reduction in MAP and heat loss responses of forearm CVC and upper back sweating in females.

In the limited number of studies comparing sex differences in postexercise arterial blood pressure, there are contrasting findings. Some studies report a more pronounced depression in blood pressure in females relative to males following moderate intensity exercise (40–70% VO2 peak) ranging in duration from 5 to 35 min duration (2, 8, 25). Alternatively, Senitko et al. (37) reported no difference in postexercise MAP between sexes following 60 min of moderate intensity (60% VO2 peak) upright seated cycling. However, in that particular study, the majority

![Fig. 4. Skin temperature (A) and mean nonevaporative heat flux (B) at preexercise rest and throughout 90 min of postexercise recovery. Data separated according to males (○) and females (□). Values are means ± SE. *Significant difference between sexes at an alpha level of 0.05.](http://ajpregu.physiology.org/)

![Fig. 5. Changes from preexercise rest in forearm cutaneous vascular conductance (CVC) (A), triceps brachii muscle blood flow (MbF) for n = 6 (B), and upper back sweat rate (C). Data are separated according to males (○) and females (□). Values are means ± SE. *Significant difference between sexes at an alpha level of 0.05.](http://ajpregu.physiology.org/)
of the recovery period was spent in the supine position with participants only in a head-up recovery position for a 5-min period, after ~45 min of recovery. It is possible that differences in exercise intensity, duration, and/or mode of exercise may explain the differences in postexercise MAP. However, the apparent effect of recovery posture upon the sex-related difference in postexercise hypotension does underscore the role of a lower orthostatic tolerance in females upon postexercise hemodynamics. The resultant effect may be manifested as a decrease in the rate of tissue temperature decay in females compared with males as shown in the present study.

Although it is possible that the greater thermal strain coupled with a greater postexercise hypotension in females could simply be fortuitous, recent evidence would suggest otherwise. Studies have shown that dynamic exercise elicits a persistent reduction in MAP lasting nearly 2 h (15, 16, 31). Postexercise hypotension is associated with a persistent rise in systemic vascular conductance that is not completely offset by an increase in $Q$ (13, 16, 37). The combination of upright seated posture and removal of the skeletal muscle pump is thought to promote venous and muscle blood pooling (36), which reduces cardiac filling and unloads cardiopulmonary baroreceptors (13, 28). Halliwill et al. (15) reported the baroreflex is reset to defend a lower blood pressure following exercise, and sympathetic vasoconstrictor outflow is consequently reduced. Further, vascular responsiveness to sympathetic vasoconstrictor outflow is impaired so that vascular resistance is attenuated for a given level of sympathetic nerve stimuli (14). Other factors contributing to the postexercise hyperemia include a sustained histamine-receptor dependent vasodilation (30, 34). The present observations of a difference in vascular conductance and arterial pressure between males and females following exercise suggest that the integration of the various mechanisms and pathways modulating the pattern of postexercise hemodynamics is sex dependent.

The greater and more prolonged postexercise reduction in vascular resistance observed for females could, in part, be explained by a temperature-induced modulation of the H1- and H2-receptor-mediated vasodilation. Muscle temperature in both the active and inactive muscles was greater in females. It has been suggested that physical stimuli, such as heat, can cause histamine release from mast cells (1). Histamine can induce vasodilation by binding to H1 receptors located on vascular endothelial cells or to H2 receptors located on vascular smooth muscle cells. While speculative, our data support this possibility.

Senitko et al. (37) stated that forearm and calf vascular resistances are decreased in parallel with systemic vascular resistance; thus the vasodilation that underlies postexercise hypotension is not restricted to active skeletal muscles but also involves inactive muscle regions in the supine recovery position. However, recovery in the present study was in the upright seated posture, which would tend to exacerbate venous and muscle pooling of warm blood (11). This response would be more pronounced for females, as indicated by the greater and more prolonged decrease in TPR. In parallel, we show that the active muscle temperature of the vastus medialis remains elevated above $T_{es}$ for a prolonged period postexercise in both males and females. However, the magnitude of the elevation is greater in females. In conjunction with a rapid reduction in skin perfusion, sweating, and whole body heat loss, and a decrease in circulatory convective heat transfer following the cessation of exercise, a time-dependent transfer of heat from previously active muscle to the central core region, likely contributes to the prolonged elevation of $T_{es}$.

The reduction in postexercise MAP has been associated with a marked reduction in skin blood flow and sweating and a decrease in the rate of core (24, 26, 42) and muscle (24) temperature recovery. This is further supported by studies showing that manipulation of postexercise hemodynamics using either lower body positive pressure (+45 mmHg) (20) or head-down tilt (35) attenuates the fall of MAP, forearm CVC, and sweat rate and elicits a shorter $T_{es}$ recovery time. Similarly, attenuating the reduction in MAP using active (loadless pedaling) or passive (assisted pedaling) recovery modes increases forearm CVC and sweating relative to inactive (motionless) recovery (2, 3, 21, 38, 43). Collectively, these observations, based primarily on work conducted in males, underscores a baroreceptor-mediated attenuation in heat loss response most likely manifested via a redistribution of blood from the lower extremities to the central circulation. Given the relationship between CVC and sweating and baroreceptor unloading, our present findings suggest that the heat loss responses are attenuated secondary to the greater hypotension observed. While a reduction in CVC might lead to greater central blood volume and a concomitant increase in MAP, there are many autonomic and humoral differences in cardiovascular control between males and females that could be at play postexercise and therefore may contribute to the larger MAP reduction in females (5, 10, 12). Furthermore, Wilkins et al. (42) reported that CVC as measured at four skin sites (chest, forearm, thigh, and leg) does not contribute to postexercise hypotension. Therefore, it is difficult to assume that a reduction in forearm CVC should contribute to the greater MAP in females.

Given that the circulatory system is a significant avenue of heat transfer within the body, the observed difference in esophageal and muscle tissue temperature response between males and females may likely be due to differences in the rate of convective heat transfer between muscle and blood and between blood and the central core region (7), as previously discussed. This effect would likely be exacerbated by the known baroreceptor-mediated attenuation of the postexercise heat loss responses of skin vasodilation and sweating (19–22, 35). The present findings show that the postexercise attenuation of vasomotor and sudomotor activity was greater in females compared with males. Inoue et al. (18) reported that heat loss for females depends more on skin perfusion than on sweating, irrespective of the phase of the menstrual cycle, in response to passive heating. In the present study, forearm CVC remained significantly lower in females during the early stages of recovery (first 20 min of recovery), whereas a significantly lower sweating rate in females was only recorded between 30 and 45 min of recovery. Concurrently, a significant difference between sexes was observed for esophageal and both active and inactive muscle temperatures within the initial 10 min of exercise recovery. This suggests that skin blood flow had a greater contribution than sweat rate to the greater and more prolonged elevation in tissue temperatures in females during the early stages of recovery following exercise. A study by Wilson et al. (43) concluded that factors other than thermal or...
baroreceptor loading status contribute to sweat rate during exercise recovery; however, their observations were limited to 5 min postexercise in the supine posture. We first noted a significant difference in upper back sweating in the later stages of recovery. Thus, it is possible that a role for baroreceptors in the modulation of postexercise sweat rate might be masked in the initial stages of recovery and become more apparent later in recovery (35).

Factors such as exercise capacity (41), sex hormones (18), and body composition (i.e., body surface to body mass ratio, subcutaneous fat content) (17) may, in part, explain the sex differences in postexercise core temperature regulation between males and females. These differences are reduced when participants are matched across sexes for physical fitness (23, 40). The male and female participants in the present study were matched for body composition and physical fitness (based on $\text{VO}_2\text{peak}$ expressed per kilogram of fat-free mass). Further, neither group was likely heat acclimated, as all trials were conducted between the months of November and March. We also ensured that the female participants were studied during the early follicular phase of their menstrual cycle to reduce the known influence of hormone-mediated modulation of vasomotor (29) and sudomotor (9) responses. We cannot exclude, however, the possibility that sex differences in neuromuscular activation patterns or alterations in synergistic muscle recruitment may have resulted in differences in the pattern of response of postexercise recovery tissue temperature and hemodynamics (4).

**Practical consideration.** Dynamic exercise appears to result in altered hemodynamics that persist for a prolonged period postexercise in the upright supported recovery position. Such changes are thought to be the root cause of postexercise syncope and orthostatic intolerance. In the context of our observations supporting a possible link between hemodynamic and heat loss response postexercise, this may lead to a compromised thermal function following exercise, which we show to be more pronounced in females. Regardless of the actual underlying mechanism, a compromised thermoregulatory response following physical exertion is of considerable concern within a working environment due to the associated increased risk of postexertional heat-related illnesses and collapse often reported in industrial workers, military personnel, and athletes. The further elucidation of the sex-related difference in the nature, time course, and mechanisms of the postexercise disturbance in thermoregulation is therefore required.

In conclusion, the present study demonstrates a greater and more prolonged elevation in $T_a$ and active and inactive muscle temperatures in females following dynamic exercise, as measured during the early follicular phase. This was paralleled by a greater and more prolonged decrease in postexercise MAP in females. These findings provide unique evidence that further support the association between postexercise hypotension, heat loss responses, and tissue temperature recovery. Further research is required to examine the influence of menstrual cycle phase on recovery hemodynamic and core temperature regulation.

**ACKNOWLEDGMENTS**

We sincerely thank the subjects involved in this study for their participation. We would also like to thank Drs. Witek Zaleski, Mark Reardon, and Frank Reardon for technical assistance.

**REFERENCES**

22. Journet WS, Reardon FD, McNiss NH, Kenny GP. Nonthermoregulatory control of cutaneous vascular conductance and sweating during...


