Differences in the postexercise threshold for cutaneous active vasodilation between men and women

Glen P. Kenny,1 Jane E. Murrin,1 W. Shane Journeay,2 and Francis D. Reardon1

1Laboratory of Human Bioenergetics and Environmental Physiology, School of Human Kinetics, Faculty of Health Sciences, University of Ottawa, Ottawa, Ontario, Canada; and 2Toxicology Program and Department of Veterinary Biomedical Sciences, Western College of Veterinary Medicine, University of Saskatchewan, Saskatoon, Saskatchewan, Canada

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Differences in the postexercise threshold for cutaneous active vasodilation between men and women. Am J Physiol Regul Integr Comp Physiol 290: R172–R179, 2006. First published August 25, 2005; doi:10.1152/ajpregu.00428.2005.—The purpose of this study was to evaluate the possible differences in the postexercise cutaneous vasodilatory response between men and women. Fourteen subjects (7 men and 7 women) of similar age, body composition, and fitness status remained seated resting for 15 min or cycled for 15 min at 70% of peak oxygen consumption followed by 15 min of seated recovery. Subjects then donned a liquid-conditioned suit. Mean skin temperature was clamped at 34°C for 15 min. Mean skin temperature was then increased at a rate of 4.3 ± 0.8°C/h while local skin temperature was clamped at 34°C. Skin blood flow was measured continuously at two forearm skin sites, one with (UT) and without (BT) (treated with bretylum tosylate) intact α-adrenergic vasconstrictor activity. The exercise threshold for cutaneous vasodilation in women (37.51 ± 0.08°C and 37.58 ± 0.04°C for UT and BT, respectively) was greater than that measured in men (37.33 ± 0.06°C and 37.35 ± 0.06°C for UT and BT, respectively) (P < 0.05). A difference in the magnitude of the thresholds was noted between male and female subjects in the MAP postexercise were noted in women (P < 0.05). No differences in core temperature, HR, and MAP were measured in the no-exercise trial. The postexercise threshold for cutaneous vasodilation measured at the UT and BT sites for men (37.15 ± 0.03°C and 37.16 ± 0.04°C, respectively) and women (37.36 ± 0.05°C and 37.42 ± 0.03°C, respectively) were elevated above no exercise (36.94 ± 0.07°C and 36.97 ± 0.05°C for men and 36.99 ± 0.09°C and 37.03 ± 0.11°C for women for the UT and BT sites, respectively) (P < 0.05). A difference in the magnitude of the thresholds was measured between women and men (P < 0.05). We conclude that women have a greater postexercise onset threshold for cutaneous vasodilation than do men and that the primary mechanism influencing the difference between men and women in postexercise skin blood flow is likely the result of an altered active vasodilatory response and not an increase in adrenergic vasconstrictor tone.

For cutaneous circulation is particularly important when one considers that there is increasing evidence demonstrating gender differences in the postexercise threshold for cutaneous blood flow (25). Furthermore, changes in hemodynamic response induced by the application of lower body positive pressure in the upright position postexercise, such as an increase in stroke volume and mean arterial pressure, were shown to reverse the postexercise increase in the onset threshold for cutaneous vasodilation (16). The studies examining postexercise control of cutaneous circulation have primarily been conducted in men. To date, it remains to be determined how possible differences in the postexercise cardiovascular response between men and women may influence control of the cutaneous circulation. The study of differences between men and women in the control of cutaneous circulation is particularly important when one considers that there is increasing evidence demonstrating gender differences in the postexercise threshold for cutaneous vasodilation (25). Furthermore, changes in hemodynamic response induced by the application of lower body positive pressure in the upright position postexercise, such as an increase in stroke volume and mean arterial pressure, were shown to reverse the postexercise increase in the onset threshold for cutaneous vasodilation (16).

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differences in postexercise blood pressure regulation in the upright seated position. Carter et al. (2) measured a greater reduction in mean arterial pressure in women compared with men at 5 min after a 3-min exercise bout. Fisher et al. (8) demonstrated a gender difference in postexercise mean arterial pressure measured over the course of a 90-min recovery after 35 min of cycling at 60% peak oxygen consumption (V\text{\textsubscript{O2 peak}}). In addition, Sentik et al. (40) showed a gender difference in the postexercise mean arterial pressure response in the upright position in sedentary individuals following cycling at 60% V\text{\textsubscript{O2 peak}} for 60 min. These differences may possibly be explained by the observations that women have a reduced tolerance to orthostatic challenges at rest (6, 12, 13, 41, 45) and/or an attenuated responsiveness in mechanisms that regulate arterial pressure (6, 13). The aforementioned studies show a difference in the blood pressure response postexercise between men and women (2, 8, 40) as well as a relationship between postexercise blood pressure and thermal response thresholds in men (16, 25). Therefore, it is a reasonable postulate that women will show a greater increase in the cutaneous vasodilatory threshold in association with a greater reduction in postexercise mean arterial pressure than men.

Thus the purpose of this study was to examine possible differences in the threshold for active cutaneous vasodilatation between men and women using an exercise paradigm, which has previously been shown to elicit postexercise hypotension in men (24, 25). Specifically, we tested the hypothesis that women would demonstrate a greater reduction in mean arterial pressure and concurrently a greater increase in the postexercise core temperature at which the onset of cutaneous vasodilatation occurred. Furthermore, we hypothesized that an increase in the onset threshold for cutaneous vasodilatation would likely manifest as altered active vasodilator activity rather than activation of adrenergic vasoconstrictor tone.

METHODS

Subjects. Fourteen healthy and physically active subjects (7 men and 7 women) volunteered and gave written consent to participate in this study, previously approved by the Research Ethics Board of the University of Ottawa. The female subjects were eumenorrheic, with regular, ∼28-day-long menstrual cycles. To control for hormonal effects, the female subjects were tested during the early follicular phase (1–5 days after the onset of menstruation) of their menstrual cycle.

Five to seven days before the experiments, body adiposity and V\text{\textsubscript{O2 peak}} were estimated with total body densitometry and a progressive cycling protocol performed on a Monark cycle ergometer, respectively. The V\text{\textsubscript{O2 peak}} value was used to select the submaximal workload (∼70% V\text{\textsubscript{O2 peak}}) for the experimental exercise phase of the study. V\text{\textsubscript{O2 peak}} (expressed per kilogram of fat-free mass) was calculated for all subjects. The physical characteristics of the subjects are presented in Table 1. The subjects were matched for age, body composition, and physical fitness (based on V\text{\textsubscript{O2 peak}}, expressed per kilogram of fat-free mass).

Instrumentation. Esophageal temperature was monitored with a Mon-a-therm esophageal thermocouple (Mallinckrodt Medical, St. Louis, MO) inserted through a nostril and positioned at the level of the heart, at a depth equivalent to about one-fourth of the individual’s standing height by placing the tip of the thermocouple at the level of the left atrium (35). Skin temperature was monitored at 12 sites by heat-flow sensors with integrated skin temperature sensors (model FR-025-TH44018–6; Concept Engineering, Old Saybrook, CT). The area-weighted mean skin temperature was calculated by assigning the following regional percentages: head 6%, chest 9.5%, upper back 9.5%, upper arm 9%, forearm 6%, abdomen 9.5%, lower back 9.5%, anterior thigh 10%, posterior thigh 10%, anterior calf 9.5%, posterior calf 9.5%, and finger 2%.

Oxygen consumption was measured using an automated metabolic analyzer (MedGraphics, St. Paul, MN). Mean arterial pressure was calculated from the electrical integration of the pulsatile blood pressure signal obtained noninvasively, from the middle digit of the left hand (Finapres 2300; Ohmeda) referenced at the third intercostal space. The Finapres system is based on the volume clamp method first introduced by Penaz (37). These blood pressure data were recorded (with the Finapres servo control on) and stored continuously at 5-s intervals. Heart rate was monitored using a Polar coded transmitter, recorded continuously and stored with a Polar Advantage interface and Polar Precision Performance software (Polar Electro Oy).

Skin blood flow was measured by laser-Doppler velocimetry (Peri-Flux System 5000, main control unit; PF5010 LDPM, Function unit; Perimed, Stockholm, Sweden) from the left midanterior forearm. The laser-Doppler flow probes (PR 401 Angled Probe; Perimed) were taped to cleaned skin on the ventral aspect of the forearm, in an area that was not overly vascular to visual inspection and from where consistent readings were noted. Cutaneous vascular conductance was calculated throughout the experimental protocol using the ratio of 30-s averages of laser-Doppler flux and mean arterial pressure. To minimize the effects of movement artifacts during the experimental trials, the arm was held in a constant position using an adjustable arm support.

To determine the effect of exercise on cutaneous active vasodilator activity postexercise, the vasoconstrictor activity effect was abolished by iontophoresic application of bretylium tosylate (21) to 1.0 cm² of skin on the ventral side of the left forearm. Bretylium tosylate blocks the presynaptic release of neurotransmitters from sympathetic adrenergic nerve endings within the area of application. Thus neurally mediated adrenergic vasoconstriction is selectively blocked without modification of active vasodilatation (21). Periostel micropharmacology system PF480–1 (Perimed) was used in all experimental trials for the application of bretylium tosylate by iontophoresis. The system uses a disposable drug delivery electrode (PF 481–1) in which a 10 mM solution of bretylium tosylate in propylene glycol is delivered. The protocol consisted of a 10-min application period at a current density of 400 μA/cm² (21). In all experiments, skin blood flow was measured simultaneously at both an untreated and bretylium-treated site. At each site, a servo heater-controlled laser-Doppler flow probe was mounted on the skin. Local skin temperature at the probe holder was maintained at 34°C throughout the experimental protocol except during the cooling maneuver to evaluate the effectiveness of the presynaptic adrenergic blockade described below.

The effectiveness of the presynaptic adrenergic blockade was tested before and after the experimental protocol by cooling the entire skin surface (except feet, hands, face, and the local skin site on the forearm) using a liquid conditioned suit (Med-Eng, Ottawa, ON, Canada) and recording the skin blood flow and mean arterial pressure (39).

Table 1. Physical characteristics of the subjects

<table>
<thead>
<tr>
<th></th>
<th>Men</th>
<th>Women</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, yr</td>
<td>24±1.1</td>
<td>24±0.9</td>
</tr>
<tr>
<td>Height, cm</td>
<td>176.9±2.0</td>
<td>165.4±1.8*</td>
</tr>
<tr>
<td>Weight, kg</td>
<td>82.1±1.7</td>
<td>62.4±2.7*</td>
</tr>
<tr>
<td>BSA/mass</td>
<td>0.0242±0.001</td>
<td>0.026±0.001</td>
</tr>
<tr>
<td>Body fat,%</td>
<td>18.4±0.9</td>
<td>20.7±1.2</td>
</tr>
<tr>
<td>V\text{\textsubscript{O2 peak}}, ml·min⁻¹·kg⁻¹</td>
<td>52.0±1.7</td>
<td>47.9±2.4</td>
</tr>
<tr>
<td>V\text{\textsubscript{O2 peak}}, ml·min⁻¹·kg FFМ⁻¹</td>
<td>63.6±2.19</td>
<td>60.5±2.6</td>
</tr>
</tbody>
</table>

Values are means ± SE. BSA, body surface area; FFM, fat-free mass. *Significant difference from men (P < 0.05).
Core and skin temperature and skin blood flow were recorded (Hewlett Packard, data-acquisition module, model 3497A), stored (model PC-312, 9000; Hewlett Packard), and displayed in real time continuously at 10-s intervals.

Experimental protocol. Each subject performed a total of two experimental trials carried out in random order. Twelve (6 women and 6 men) of the 14 subjects performed their experimental trials between the months of February and early April. The experimental trials for the two remaining subjects were conducted in late May and early June. Experiments were conducted after a 48-h period without physical activity, and subjects were instructed to avoid excessive perambulation or other stresses during the period between awakening and experimentation, such as exposure to hot or cold temperatures and excessive physical activity during transit from home to the laboratory. Furthermore, they were asked to fast at least 4 h before experimentation but were permitted water ad libitum during this time.

Prewarming phase. On arrival to the laboratory, subjects clothed in shorts and athletic shoes were fitted with the appropriate instruments and donned the liquid-conditioned suit. Each of the two experimental trials commenced at ~0900. Subjects were initially habituated at an ambient temperature of 22°C, which was maintained for the duration of the experimental trial (Fig. 1). The bretylium tosylate was applied during this habituation period. The effectiveness of the presynaptic adrenergic blockade was verified after 90 min. Mean skin temperature was first held at ~34°C for ~15 min with the aid of the liquid-conditioned suit perfused with ~34°C water by use of a temperature-controlled circulation bath (Endocal, NESLAB, and model 200-00; Micropump, Vancouver, WA). The water perfusate was then rapidly changed to 2°C, and skin cooling continued for 3 min. The lack of change in cutaneous vascular conductance at both forearm skin sites was used to verify the effectiveness of presynaptic adrenergic blockade (33) before continuation of the experimental trial. After verification of blockade, the liquid-conditioned suit was removed.

The subjects were then required to either perform 15 min of cycling on a Monark cycle ergometer at ~70% of their predetermined $V_{O_2\, peak}$ (exercise; actual calculated work rate was equal to 72.4% of $V_{O_2\, peak}$) or remain resting (no exercise) for 15 min. For the no-exercise treatment, the subjects were instructed to remain resting in a seated upright position for 15 min. Immediately after these respective treatments, subjects either remained upright seated (no exercise) or were placed similarly seated (exercise) for 15-min resting recovery at an ambient temperature of 22°C.

Warming phase. Subjects then donned the liquid conditioned suit. Mean skin temperature was clamped at ~34°C for ~15 min. Mean skin temperature was then increased at a rate of 4.3 ± 0.8°C/h as the water circulating through the suit was progressively increased to 48°C. Whole body warming continued until the skin blood flow achieved a sustained elevated value (~40 min). Local skin temperature at the probe holder was maintained at 34°C during whole body warming.

At the end of each experiment, local skin temperature at the bretylium-treated and untreated forearm skin sites was raised to 43°C until peak cutaneous vascular conductance was measured (~30 min). Peak cutaneous vascular conductance was determined as a sustained elevated plateau in local skin blood flow (25, 33). Local warming was immediately followed by a second 3-min cold stress to verify the persistence of the presynaptic adrenergic blockade.

Data and statistical analysis. The onset threshold for cutaneous vasodilation was determined to be the esophageal temperature at which there was an increase in cutaneous vascular conductance measured on the ventral surface of the forearm, observed in three consecutive measurements (33). The thresholds were determined by a blinded investigator. Thermal sensitivity was defined as the slope of the linear portion of the cutaneous vascular conductance-esophageal temperature relationship as measured during whole body warming postexercise (33). The linear portion of this curve was selected by visual inspection, and the slopes were determined by least squares linear regression analysis. Changes in cutaneous vascular conductance during the 3-min cold stress at the treated and untreated forearm sites are reported as a percentage of peak cutaneous vascular conductance. The average response of the different physiological variables was compared for each condition by using ANOVA with repeated measures. In the event of statistical significance ($P < 0.05$), a Tukey’s test was used to identify significant differences. All values are presented as means ± SE.

RESULTS

Effectiveness of the presynaptic adrenergic blockade. The application of the cold stress before the experimental protocol (Fig. 1, see marker $V_1$) induced a significant reduction in cutaneous vascular conductance at the untreated control forearm skin site. Cutaneous vascular conductance decreased from 14.9 ± 1.5% of peak cutaneous vascular conductance at the start of whole body cooling to 9.2 ± 1.0% of peak cutaneous vascular conductance measured at the end of the 3-min cooling period (mean value for all trials, $P < 0.05$). A similar reduction was measured in the second cold stress conducted at the end of the experimental protocol (Fig. 1, see marker $V_2$) from 78.7 ± 2.7% to 63.0 ± 3.6% of peak cutaneous vascular conductance (mean value for all trials, $P < 0.05$). Iontophoresis of bretylium blocked the cold-induced reduction in cutaneous vascular conductance before and after exercise for all trials, demonstrating the effective and persistent sympathetic vasoconstrictor blockade at these forearm skin sites. Cutaneous vascular conductance during the cold stress application remained unchanged (36.0 ± 3.9% to 34.0 ± 3.6% of peak cutaneous vascular conductance). Similarly, no significant reduction in
cutaneous vascular conductance was noted during the second cold stress application performed at the end of the experimental protocol (87.5 ± 1.7% to 85.8 ± 2.4% of peak cutaneous vascular conductance).

Prewarming phase. Resting heart rate, mean arterial pressure, esophageal temperature, and mean skin temperature were similar for all conditions during baseline resting (Table 2).

Hemodynamic response. As shown in Table 2, the postexercise prewarming (i.e., measurement taken at 30 min posttreatment) mean arterial pressure for men (84 ± 3 mmHg) and women (79 ± 3 mmHg) remained significantly reduced relative to the baseline resting pressure reference value (93 ± 3 and 93 ± 4 mmHg for men and women, respectively) (P < 0.05). A greater decrease in the mean arterial pressure postexercise was noted in women (P < 0.05). Mean arterial pressure remained unchanged throughout the no-exercise trial.

End-exercise heart rates were 157 ± 4 and 161 ± 5 beats/min for men and women, respectively. For all exercise trials, heart rate remained significantly elevated (P < 0.05) above baseline rest values for the 15 min postexercise recovery period. Prewarming heart rates for men (77 ± 4 beats/min) and women (87 ± 6 beats/min), as measured at 30 min posttreatment, were significantly elevated above baseline resting (61 ± 3 and 68 ± 4 beats/min for men and women, respectively) (P < 0.05).

Table 3. Esophageal and mean skin temperature at the onset threshold for forearm cutaneous vasodilation during exercise at the bretylium-treated and untreated sites

<table>
<thead>
<tr>
<th>T_core, °C</th>
<th>Men</th>
<th>Women</th>
</tr>
</thead>
<tbody>
<tr>
<td>Untreated</td>
<td>37.33±0.06*</td>
<td>37.51±0.08</td>
</tr>
<tr>
<td>Bretylium-treated</td>
<td>37.35±0.06*</td>
<td>37.58±0.04</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>T_skin, °C</th>
<th>Men</th>
<th>Women</th>
</tr>
</thead>
<tbody>
<tr>
<td>Untreated</td>
<td>32.36±0.23</td>
<td>32.29±0.20</td>
</tr>
<tr>
<td>Bretylium-treated</td>
<td>32.51±0.22</td>
<td>32.30±0.19</td>
</tr>
</tbody>
</table>

Values are means ± SE. *Significant difference between men and women, P < 0.05.
Esophageal temperature and mean skin temperature for the no-exercise condition remained unchanged from baseline resting.

**Warming phase.** Mean skin temperature was increased at the same rate of 4.3 ± 0.8°C/h during the whole body warming maneuver for all subjects in all conditions.

**Cutaneous vasodilation.** The postexercise thresholds for cutaneous vasodilation measured at the untreated forearm sites for men (37.15 ± 0.03°C) and women (37.36 ± 0.05°C) were elevated above no-exercise levels (36.94 ± 0.07°C and 36.99 ± 0.09°C for men and women, respectively; P < 0.05) (Fig. 3). A similar response in the onset threshold for cutaneous vasodilation was measured for the bretylium-treated forearm for all trials (Table 4). A difference in the magnitude of the thresholds was measured between women and men at both the untreated and bretylium-treated forearm sites (P < 0.05). In contrast, no gender difference in the onset threshold for cutaneous vasodilation was measured for the no-exercise trial. These results, as well as those from no-exercise resting are presented in Table 4. In addition, Fig. 4 depicts the relative increase in the thresholds relative to baseline resting esophageal temperature.

Mean skin temperature at the onset threshold for cutaneous vasodilation was similar for all conditions (Table 4). The sensitivity of the thermal reflex was estimated from the slope of the linear relationship between cutaneous vascular conductance and esophageal temperature. The rate of rise of cutaneous vascular conductance per unit change in esophageal temperature was not significantly different between exercise and no-exercise control levels for either men or women.

**DISCUSSION**

The exercise (21, 25, 39) and postexercise (16, 23, 25) elevations in the threshold for cutaneous vasodilation are in agreement with previous findings. However, we observed a difference in the magnitude of the measured thresholds between men and women. Of note, the greater postexercise threshold measured in women was accompanied by a more pronounced decrease in postexercise mean arterial pressure relative to men. These observations support our hypothesis that

Table 4. Esophageal and mean skin temperature at the onset threshold for forearm cutaneous vasodilation for no-exercise and postexercise resting at the bretylium treated and untreated sites for all conditions

<table>
<thead>
<tr>
<th>Condition</th>
<th>Men (°C)</th>
<th>Women (°C)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>T\text{es}, °C</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Untreated</td>
<td>36.94±0.07</td>
<td>36.99±0.09</td>
</tr>
<tr>
<td>Bretylium-treated</td>
<td>36.97±0.05</td>
<td>37.03±0.11</td>
</tr>
<tr>
<td><strong>T\text{sk}, °C</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Untreated</td>
<td>36.14±0.10</td>
<td>36.24±0.09</td>
</tr>
<tr>
<td>Bretylium-treated</td>
<td>36.20±0.10</td>
<td>36.26±0.11</td>
</tr>
</tbody>
</table>

Values are means ± SE. *Significant difference between men and women, P < 0.05. †Significant increase from no-exercise group, P < 0.05.
women demonstrate a greater reduction in mean arterial pressure and concurrently a greater increase in the postexercise core temperature at which the onset of cutaneous vasodilation occurred compared with men. Furthermore, the similarity of the response of the postexercise resting threshold measured at the untreated and bretylium-treated sites in both men and women suggests that the primary mechanism for the difference likely results from altered active vasodilation and not from increased adrenergic vasoconstrictor tone.

**Threshold for vasodilation during exercise.** Our observation of an increase in the exercise threshold for cutaneous vasodilation, which was similar for both the untreated and bretylium-treated sites, is consistent with previous findings (25). However, we showed that the magnitude of the exercise increase in the threshold for cutaneous vasodilation was greater in women compared with men. This observation is consistent with the findings of Roberts et al. (39) who reported a higher threshold for skin blood flow in women compared with men as measured during exercise. Thus the difference in the magnitude of the threshold associated with an increase in exercise skin blood flow may be the result of differences in thermal sensitivity between men and women (7). In contrast, Kolka et al. (28) reported no differences between men and women in the threshold or thermal sensitivity during exercise performed in hot ambient conditions. Differences in the pattern of thermoregulatory control in skin and active muscle blood flow associated with the combined stress of exercise and hot ambient temperature may in part explain the discrepancy in the observed responses reported in these studies (1).

**Postexercise thermal response.** The observation of a postexercise elevation in core temperature measured in both male and female subjects, as recorded at 15-min postexercise, is noteworthy. This observation is consistent with the previous reports of a sustained elevation in core temperature postexercise in men (16, 22, 44).

The observed postexercise increase in the threshold for cutaneous vasodilation measured after a short recovery are similar to previous findings (16, 23, 25, 26). These previous studies, however, present a possible inherent bias in that they were either conducted on men exclusively or did not include enough women to determine possible sex-dependent differences. Although we report a postexercise increase in the onset threshold for the men and women, we observed that this postexercise elevation in the warm response threshold of cutaneous vasodilation is significantly greater in women compared with men.

Consistent with previous observations (16, 23, 25), we observed an increase in the postexercise threshold when passive whole body warming was initiated during the period of postexercise hypotension. Furthermore, a greater increase in the postexercise threshold recorded for the women was paralleled by a greater reduction in mean arterial pressure. Although it is possible that this relationship is merely fortuitous, recent evidence would suggest otherwise. For example, it has previously been shown that an increase in the postexercise hypotensive response, induced by exercise of increasing intensity, was shown to result in a relative increase in the onset threshold for cutaneous vasodilation in men (26). Studies have shown that different recovery modes (i.e., inactive, passive, or active recovery mode) impact the role of nonthermal (i.e., central command, baroreceptors, and muscle mechanoreceptors) influences on postexercise skin blood flow (3, 18, 47). Specifically, these studies show that skin blood flow during inactive recovery is influenced primarily by baroreceptors. Furthermore, restoration of mean arterial pressure through application of positive pressure to the lower limbs was shown to reverse the postexercise increase in the threshold for cutaneous vasodilation (16) and also increase whole body heat loss (17).

The postexercise hemodynamic response, as represented by heart rate and mean arterial pressure, is consistent with postexercise hypotension (5, 24, 31). There have been many reports of reduced tolerance in the face of an orthostatic challenge in women compared with men (6, 12, 13, 41, 45). This response is subsequent to a reduced mean arterial pressure and reduced compensatory vasoconstriction in women compared with men (2). Implicit in the evidence that women have reduced tolerance to orthostatic challenge is the observation that women regulate blood pressure via different mechanisms (2, 40). Moreover, it has been shown that women have less effective baroreflex buffering of arterial blood pressure compared with men (4). It has also been shown that women appear to have an attenuated responsiveness in the mechanisms that regulate arterial pressure (6, 13). It is plausible, therefore, given the orthostatic effect of the upright seated posture during exercise recovery employed in our study in combination with the decreased responsiveness of cardiovascular mechanisms to regulate arterial pressure in women, that this may have resulted in the greater postexercise hypotension. In parallel, it has been shown that acute reductions in central venous pressure, which can be associated with a hypotensive response, can delay or decrease the rise in skin blood flow (33, 34). This may explain our observation of a difference between men and women in the onset threshold for cutaneous vasodilation.

It should be noted that, although the study by Senitko et al. (40) did not show a postexercise hypotension in their endurance-trained individuals, they reported a difference in postexercise hypotension between men and women in their sedentary group in the upright posture. This difference was absent when subjects recovered in the supine posture, which may underscore the role of orthostasis in the observed differences between men and women. Although our subjects were considered fit, they did not meet the criteria of 20–60 miles/wk as outlined in their study. Of note, the maximal oxygen consumption values of the participants in the study by Senitko et al. (40) were not reported; thus we cannot make a comparison with our subjects. However, we estimate the fitness level of our subjects to be between that of the sedentary and endurance-trained participants, which may explain our observed difference in the postexercise hypotension response between men and women.

**Limitations of this investigation.** Factors such as aerobic fitness, heat acclimation, hydration status, and sex hormones may explain the differences in exercise and postexercise skin blood flow responses between men and women (10, 39). Furthermore, these differences between men and women may be attributed to 1) a larger ratio of body surface to body mass, 2) a greater subcutaneous fat content, and 3) a lower exercise capacity. However, it has been shown that these differences are reduced when men and women are matched for physical fitness (19, 29, 30, 42, 43, 48). In our study, the male and female subjects were matched for age, body composition, and physical fitness (based on VO2peak expressed per kilogram of fat-free mass). Furthermore, neither group was likely heat acclimated,
because 12 of the 14 subjects completed their experimental trials between the months of February and early April. The women were studied during the early follicular phase of their menstrual cycle to reduce the influence of hormone-mediated modulation of exercise skin blood flow response. It is possible that differences in hydration status may explain our observations of a gender difference in the postexercise onset threshold for cutaneous vasodilation. Studies have shown that hypohydration increases the threshold for cutaneous vasodilation (10) and that the magnitude of the response is dependent on the level of hypohydration. However, it has been shown that sweating rates are generally lower in women than in men (11). Furthermore, as noted by Senniko et al. (40), it is likely that the male subjects had a greater sweating-related fluid loss, subsequently losing more plasma volume and undergoing greater reductions in central venous pressure. Thus we would have expected a greater increase in the onset threshold for men compared with women. Furthermore, the hydration status of our subjects was not verified during the experiment because it was unlikely that any significant hypohydration occurred given the type and duration of the exercise. Montain and Coyle (36) demonstrated that 2 h of dynamic exercise at 65% of \( \dot{V}O_2 \text{peak} \) performed in a warm environment (33°C) with no water intake results in a maximum weight loss of 4.2%. Similarly, Mack and Nadel (32) noted that a 70-kg adult could potentially lose on the order of 2.5% of water content per hour of heavy exercise in the heat, owing primarily to water loss from sweating. In our study, the short duration of moderate-intensity exercise performed in a cooler environment with unrestricted pretrial water intake is unlikely to have caused more than a 0.5% weight loss. Under this condition, our subjects could be considered euhydrated (14).

In summary, our findings support the hypothesis that women would demonstrate a greater reduction in mean arterial pressure and concurrently a greater increase in the postexercise core temperature at which the onset of cutaneous vasodilation occurred compared with men. The greater onset threshold measured in women could be due to 1) an alteration of vasodilator outflow, due to baroreceptor unloading or 2) a baroreceptor-mediated increase in adrenergic vasoconstrictor tone. Our demonstration of a similar threshold for cutaneous vasodilation at the untreated and bretylium-treated forearm sites would suggest that the primary mechanism of control for the difference in postexercise skin blood flow between men and women is likely the result of an altered active vasodilatory response and not an increase in adrenergic vasoconstrictor tone.

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