Cutaneous active vasodilation in humans during passive heating postexercise

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Submitted 10 April 2003; accepted in final form 27 May 2003

Kenny, Glen P., Julien Périard, W. Shane Journeay, Ronald J. Sigal, and Francis D. Reardon. Cutaneous active vasodilation in humans during passive heating postexercise. J Appl Physiol 95: 1025–1031, 2003.—The hypothesis that exercise causes an increase in the postexercise esophageal temperature threshold for onset of cutaneous vasodilation through an alteration of active vasodilator activity was tested in nine subjects. Increases in forearm skin blood flow and arterial blood pressure were measured and used to calculate cutaneous vascular conductance at two superficial forearm sites: one with intact α-adrenergic vasoconstrictor activity (untreated) and one infused with bretylium tosylate (bretylium treated). Subjects remained seated resting for 15 min (no-exercise) or performed 15 min of treadmill running at either 55, 70, or 85% of peak oxygen consumption followed by 20 min of seated recovery. A liquid-conditioned suit was used to increase mean skin temperature (−4.0°C/h), while local forearm temperature was clamped at 34°C, until cutaneous vasodilation. No differences in the postexercise threshold for cutaneous vasodilation between untreated and bretylium-treated sites were observed for either the no-exercise or exercise trials. Exercise resulted in an increase in the postexercise threshold for cutaneous vasodilation of 0.19 ± 0.01, 0.39 ± 0.02, and 0.53 ± 0.02°C above those of the no-exercise resting values for the untreated site (P < 0.05). Similarly, there was an increase of 0.20 ± 0.01, 0.37 ± 0.02, and 0.53 ± 0.02°C for the treated site for the 55, 70, and 85% exercise trials, respectively (P < 0.05). It is concluded that reflex activity associated with the postexercise increase in the onset threshold for cutaneous vasodilation is more likely mediated through an alteration of active vasodilator activity rather than through adrenergic vasoconstrictor activity.

postexercise hypotension; skin blood flow; exercise intensity; baroreceptor; thermoregulation

Recent studies indicate that exercise induces a residual effect on thermal control, resulting in an increase of −0.3–0.4°C in the postexercise esophageal temperature threshold at which cutaneous vasodilation occurs (ThVD) (7, 15, 18). Although the mechanism(s) for thermoregulatory control of skin blood flow before and during exercise have been described, there remains a paucity of information on their nature and role during postexercise temperature regulation. It is well documented that the active vasodilator control of cutaneous circulation is the primary mechanism for increasing skin blood flow during heat stress. Activation of the active vasodilator system is responsible for 80–95% of the elevation in skin blood flow accompanying heat stress (28).

Recent studies clearly demonstrate that the cutaneous vasodilator system is subject to nonthermoregulatory baroreflex modulation (10, 20). Furthermore, dynamic exercise is known to result in postexercise hypotension in the upright position (1, 17, 19, 26). Thus, during postexercise hypotension, mean arterial pressure is reduced subsequent to both neural and vascular activity (4–6). This postexercise hypotension is thought to result in baroreceptor unloading as a result of decreased cardiac filling arising from venous pooling of blood in the previously active musculature (1, 25). As well, it has been shown that acute reductions in cardiac filling delay or decrease the rise in skin blood flow (20, 21). Elsewhere, it has been shown that the nonthermoregulatory baroreceptor response to this postexercise venous blood pooling significantly influences cutaneous vasomotor control during exercise recovery (7, 15). Thus the modification of postexercise venous pooling, either by head-down tilt (15) or by lower body positive pressure (7), results in a lowering of the resting postexercise elevation in ThVD. The effect of this baroreceptor response on cutaneous vascular tone is manifest either as an alteration of active vasodilator outflow (10) or as an activation of sympathetic vasoconstrictor activity.

To date and to our knowledge, there have been no studies that have examined the effect of exercise intensity on the postexercise skin blood flow response. It has been shown that exercise performed at increasing work intensities (i.e., work rate ≥70% maximal oxygen consumption) elicits a significant hypotension in the period immediately postexercise (19, 25). Furthermore, it has been shown that an increase in this postexercise...
hypotensive response, induced by exercise of increasing intensity, results in a relative decrease in the rate of heat loss and a concomitant increase in the postexercise core temperature recovery time (17). The attenuation of the disturbance in thermal balance postexercise during head-down tilt maneuvers (15) and upright lower body positive pressure application (7) underscores the significant impact of blood pooling on cutaneous vasodilation after a bout of moderate upright dynamic exercise. Whether an increase in the postexercise hypotensive response, and therefore baroreceptor unloading (as occurs with lower body negative pressure application (20)), will result in a baroreceptor-mediated increase in the ThVD, remains unclear.

Thus the following study was conducted to evaluate the mechanism of skin blood flow control during the postexercise period. Specifically, we evaluated the hypothesis that exercise causes a residual increase in the ThVD mediated by an alteration of active vasodilator activity. Skin blood flow monitored with laser-Doppler flowmetry and local application of bretylium tosylate were used to study the roles during the postexercise period of the cutaneous vasoconstrictor and vasodilator systems. In addition, we tested the hypothesis that an exercise-dependent decrease in postexercise mean arterial pressure, and thus a decrease in cardiac filling (baroreceptor unloading), results in a greater overall increase in the magnitude of the ThVD.

METHODS

Subjects

Nine healthy and physically active subjects (8 men and 1 woman) volunteered and gave written consent to participate in this study, previously approved by the Research Ethics Board of the University of Ottawa. The female subject was eumenorrheic with regular, ~28-day-long menstrual cycles. To control for hormonal effects, the female subject was tested during the follicular phase of her menstrual cycle.

Five to seven days before the experiments, body adiposity and peak oxygen consumption (V\textsubscript{O2 peak}) were estimated by using total body densitometry and a progressive treadmill running protocol, respectively. The V\textsubscript{O2 peak} value was used to select the submaximal workload for the experimental exercise phase of the study. Subjects were 22 ± 1 (SD) yr old, were 172 ± 2 cm tall, weighed 67.7 ± 2.3 kg, and had a percent body fat of 11.1 ± 2.0%. The average aerobic capacity was 62.4 ± 2.0 ml·kg\textsuperscript{-1}·min\textsuperscript{-1}.

Instrumentation

Central body temperature (esophageal temperature) was monitored continuously with a pediatric esophageal temperature probe (Mon-a-therm, Mallinckrodt Medical, St. Louis, MO) inserted through the nares to a depth one-fourth of the standing height of the subject, ostensibly placing the tip of the thermocouple at the level of the left atrium (22). Skin temperature was measured at 12 sites by using heat flow sensors (model FR-025-TH44018-6, Concept Engineering, Old Saybrook, CT), and 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 with 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. These blood pressure data were recorded (with the Finapres servo control on) and stored continuously at 5-s intervals. Heart rate was monitored by using a Polar coded transmitter, recorded continuously, and stored with a Polar Advantage interface and Polar Precision Performance software (Polar Electro Oy, Kempele, Finland).

Skin blood flow was measured by laser-Doppler velocimetry (PeriFlux 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 covered by vascular inspection and from where consistent readings were noted. Cutaneous vascular conductance (CVC) was calculated throughout the experimental protocol by using the ratio of 30-s averages of laser-Doppler flux and mean arterial pressure.

To determine the effect of exercise on cutaneous, active vasodilator activity postexercise, the vasoconstrictor activity effect was abolished by iontophoretic application of bretylium tosylate (9) to 1.0 cm\textsuperscript{2} 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 (3). Thus neurally mediated adrenergic vasoconstriction is selectively blocked without modification of active vasodilation (9). In all experimental trials, the application of bretylium tosylate was by iontophoresis with a Perilont micropharmacology system PF480-1 (Perimed). The Perilont system uses a disposable drug delivery electrode (PF 481-1) in which a 10 mM solution of bretylium tosylate in propylene glycol is absorbed. The protocol consisted of a 10-min application period at a current density of 400 μA/cm\textsuperscript{2} (9). In all experiments, skin blood flow was measured simultaneously at both an untreated and bretylium-treated site. At such site, the 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.

Effective α-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) by using a liquid-conditioned suit (Delta Temax, Pembroke, ON, Canada) and recording the skin blood flow and mean arterial pressure.

Core and skin temperature and skin blood flow were recorded (data-acquisition module, model 3497A, Hewlett Packard), 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 four experimental trials carried out in random order. Experiments were conducted after a 36-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

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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, the subjects, who were clothed in shorts and athletic shoes, were fitted with the appropriate instruments and donned the liquid-conditioned suit. Each of the four experimental trials commenced at ~9:00 AM. Subjects were initially acclimated in an environmentally controlled room at an ambient temperature of 22°C. The bretylium tosylate was applied during this acclimation period. The α-adrenergic blockade was verified after 90 min. Mean skin temperature was first held at ~33.5°C for ~15 min with the aid of the liquid-conditioned suit perfused with 33.5°C water. The water perfusate was then rapidly changed to 2°C, and skin cooling continued for 3 min. Changes in CVC at both forearm skin sites were used to verify the effectiveness of α-adrenergic blockade (20, 24) before the continuation of the experimental trial. After verification of blockade, the liquid-conditioned suit was removed.

Subjects were then either moved to a treadmill (exercise) or remained resting (no-exercise) for 15 min. For the exercise treatment, the subjects performed either 15 min of treadmill running at 55% (light), 70% (moderate), or 85% (intense) of their predetermined VO₂ peak. 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 seated upright (no exercise) or were placed similarly seated (exercise) for 20 min of resting recovery at an ambient temperature of 22°C.

**Warming phase.** Subjects donned the liquid-conditioned suit. Mean skin temperature was then clamped at ~33.5°C for ~15 min by using the liquid-conditioned suit perfused with 33.5°C water (the transition time required to suit the subject and start the perfusion of the liquid-conditioned suit was ~5 min). Mean skin temperature was then increased at a rate of 4.0°C/min to suit the subject. The ThVT was taken to be the esophageal temperature at which there was an increase in CVC measured on the ventral surface of the forearm, observed in three consecutive measurements (7, 20). Thermal sensitivity was defined as the slope of the linear portion of the CVC-esophageal temperature relationship. The linear portion of this curve was selected by visual inspection, and the slopes were determined by least squares linear regression analysis. 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 honestly significant difference test was used to identify significant differences. All values are presented as means ± SE.

**RESULTS**

**α-Adrenergic Blockade.**

Application of cold stress induced a significant reduction in CVC at the untreated skin site before (28.12 ± 5.04 to 14.63 ± 3.08% of peak CVC) and after (86.04 ± 1.51 to 58.12 ± 1.25% of peak CVC) the experimental protocol for all trials (mean value for all trials). Bretylium tosylate application blocked the cold-induced reduction in CVC before (30.78 ± 2.50 to 31.50 ± 2.06% of peak CVC) and after (87.27 ± 2.41 to 85.39 ± 2.70% of peak CVC) exercise for all trials demonstrating effective and persistent sympathetic vasoconstrictor blockade at these forearm skin sites.

**Prewarming Phase.**

Resting heart rate, mean arterial pressure, esophageal temperature, and mean skin temperature were similar for all conditions during baseline resting (Table 1).

**Hemodynamic response.** As shown in Table 1, the postexercise prewarming mean arterial pressure remained significantly reduced relative to the baseline.

### Table 1. Hemodynamic and esophageal and mean skin temperature values during baseline resting and postexercise for all conditions

<table>
<thead>
<tr>
<th></th>
<th>No-Exercise Treatment</th>
<th>Postexercise</th>
<th>55% VO₂ peak</th>
<th>70% VO₂ peak</th>
<th>85% VO₂ peak</th>
</tr>
</thead>
<tbody>
<tr>
<td>MAP, mmHg</td>
<td></td>
<td></td>
<td>92 ± 2</td>
<td>91 ± 2</td>
<td>92 ± 2</td>
</tr>
<tr>
<td>Baseline resting</td>
<td>93 ± 1</td>
<td></td>
<td>87 ± 1†</td>
<td>84 ± 1†</td>
<td>80 ± 1†</td>
</tr>
<tr>
<td>Postexercise</td>
<td></td>
<td></td>
<td>72 ± 3</td>
<td>70 ± 2</td>
<td>73 ± 5</td>
</tr>
<tr>
<td>HR, beats/min</td>
<td>74 ± 3</td>
<td></td>
<td>88 ± 2†</td>
<td>97 ± 4†</td>
<td>109 ± 3†</td>
</tr>
<tr>
<td>T es, °C</td>
<td></td>
<td></td>
<td>36.73 ± 0.03</td>
<td>36.78 ± 0.07</td>
<td>36.72 ± 0.06</td>
</tr>
<tr>
<td>Baseline resting</td>
<td>36.74 ± 0.04</td>
<td></td>
<td>37.06 ± 0.07†</td>
<td>37.22 ± 0.06‡</td>
<td>37.30 ± 0.07‡</td>
</tr>
<tr>
<td>Postexercise</td>
<td></td>
<td></td>
<td>33.65 ± 0.15</td>
<td>33.67 ± 0.10</td>
<td>33.67 ± 0.14</td>
</tr>
<tr>
<td>T sk, °C</td>
<td></td>
<td></td>
<td>33.75 ± 0.18</td>
<td>33.87 ± 0.19</td>
<td>33.93 ± 0.23</td>
</tr>
<tr>
<td>Baseline resting</td>
<td>33.75 ± 0.15</td>
<td></td>
<td>33.75 ± 0.15</td>
<td>33.87 ± 0.19</td>
<td>33.93 ± 0.23</td>
</tr>
</tbody>
</table>

Values are means ± SE. VO₂ peak, peak oxygen consumption; MAP, mean arterial pressure; HR, heart rate; T es, esophageal temperature; T sk, mean skin temperature. Note: postexercise data represent the values taken at 20-min postexercise before the subject donned the liquid-conditioned suit. †Significantly different from baseline resting, P < 0.05. ‡Significantly different from no-exercise, P < 0.05.
resting reference value after all exercise conditions \((P < 0.05)\). There was a greater overall reduction with increasing exercise intensity, that is, 4, 8, and 13 mmHg decrease from baseline resting for light, moderate, and intense exercise, respectively. Mean arterial pressure remained unchanged throughout the no-exercise trial.

End-exercise heart rate was \(157 \pm 5, 177 \pm 4,\) and \(190 \pm 2\) beats/min for light, moderate, and intense exercise, respectively. For all exercise trials, heart rate remained significantly elevated \((P < 0.05)\) above baseline rest values for the 20-min postexercise recovery period. Prewarming heart rate was elevated by \(18 \pm 1, 24 \pm 2,\) and \(37 \pm 2\) beats/min for light, moderate, and intense exercise, respectively \((P < 0.05)\).

**Thermal response.** The mean thresholds for cutaneous vasodilation during exercise are presented in Table 2. The \(T_{\text{THVD}}\) was significantly elevated \((P < 0.05)\) above no exercise for all exercise conditions for both the untreated and bretylium-treated sites (Fig. 1). There was an incremental increase in the magnitude of the threshold for cutaneous vasodilation with an increase in exercise intensity at both untreated and bretylium-treated sites \((P < 0.05)\).

Exercise resulted in a \(0.60 \pm 0.04, 0.96 \pm 0.05,\) and \(1.61 \pm 0.08\)°C increase in esophageal temperature above baseline resting for the light, moderate, and intense exercise, respectively \((P < 0.05)\). Esophageal temperature remained significantly elevated above baseline resting by \(0.28 \pm 0.03, 0.50 \pm 0.06,\) and \(0.65 \pm 0.07\)°C at the end of the 20-min resting recovery period for the light, moderate, and intense exercise, respectively \((P < 0.05)\), whereas mean skin temperature returned to baseline resting values (Table 1). In contrast, esophageal temperature and mean skin temperature for the no-exercise condition remained unchanged from baseline resting values.

**Warming Phase**

Mean skin temperature was increased at the same rate of \(-4.0 \pm 0.9\)°C/h for all subjects in all conditions.

Table 2. Esophageal and mean skin temperature equivalent at the onset threshold for forearm cutaneous vasodilation during light (55% of \(V_{\text{O}_2\text{peak}}\)), moderate (70% of \(V_{\text{O}_2\text{peak}}\)), and intense (85% of \(V_{\text{O}_2\text{peak}}\)) exercise

<table>
<thead>
<tr>
<th></th>
<th>Untreated</th>
<th>Bretylium Treated</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Light exercise</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(T_{\text{es}})</td>
<td>37.03 ± 0.05</td>
<td>37.06 ± 0.04</td>
</tr>
<tr>
<td>(T_{\text{sk}})</td>
<td>32.44 ± 0.18</td>
<td>32.46 ± 0.19</td>
</tr>
<tr>
<td><strong>Moderate exercise</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(T_{\text{es}})</td>
<td>37.47 ± 0.05(^a)</td>
<td>37.48 ± 0.05(^a)</td>
</tr>
<tr>
<td>(T_{\text{sk}})</td>
<td>32.52 ± 0.22</td>
<td>32.53 ± 0.18</td>
</tr>
<tr>
<td><strong>Intense exercise</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(T_{\text{es}})</td>
<td>38.01 ± 0.07(^\dagger)</td>
<td>38.02 ± 0.07(^\dagger)</td>
</tr>
<tr>
<td>(T_{\text{sk}})</td>
<td>32.64 ± 0.22</td>
<td>32.62 ± 0.25</td>
</tr>
</tbody>
</table>

Values are means ± SE given in °C. \(^a\)Significantly different from light exercise, \(P < 0.05\). \(^\dagger\)Significantly different from moderate exercise, \(P < 0.05\).

Cutaneous vasodilation. The postexercise \(T_{\text{THVD}}\) showed an incremental elevation with increasing exercise intensity. These results, as well as those from no-exercise resting, are presented in Table 3. Of note, the esophageal temperature at the initiation of vasodilation during exercise did not differ between the untreated and bretylium-treated sites (Fig. 2). Postexercise thresholds at the untreated forearm site increased by \(0.19 \pm 0.01, 0.39 \pm 0.02,\) and \(0.53 \pm 0.02\)°C above no exercise for the light, moderate, and intense exercise, respectively. Similar increases were measured for the bretylium-treated site (i.e., \(0.20 \pm 0.01, 0.37 \pm 0.02,\) and \(0.53 \pm 0.02\)°C for the light, moderate, and intense exercise, respectively). Of note, mean skin temperature at the onset threshold for cutaneous vasodilation were similar for all conditions (Table 4). The sensitivity of the thermal reflex was estimated from the slope of the linear relationship between CVC and esophageal temperature. The rate of rise of CVC per unit change in esophageal temperature was not significantly different between conditions (Table 4).

**DISCUSSION**

Our observations of a postexercise increase in \(T_{\text{THVD}}\) are similar to previous findings (7, 15, 18). However, this is the first time that it has been observed that the magnitude of the postexercise elevation in the threshold for cutaneous vasodilation is influenced by the intensity of exercise. Furthermore, the similar response of the postexercise resting \(T_{\text{THVD}}\) for the untreated and the bretylium-treated forearm sites suggests that the primary mechanism for the increase in this threshold is related to an altered active vasodilatory response.

**Cutaneous Vasodilation During Exercise**

Exercise is known to increase the threshold for cutaneous vasodilation compared with no-exercise rest-
ing (12, 29, 30). The magnitude of this increase is dependent on ambient temperature (14) and exercise intensity (29, 30). In the present study, we showed a comparable exercise intensity-dependent incremental shift in threshold for cutaneous vasodilation.

It has been well documented that exercise can modulate reflex control of the cutaneous active vasodilator system (20). This observation is based on the measurement of identical exercise-induced shifts in the esophageal temperature at which cutaneous vasodilation is initiated in skin locally treated with or without bretylium tosylate. Cutaneous vasoconstrictor nerves are known to release norepinephrine and act through α-adrenergic receptors. Bretylium is an antiadrenergic agent that blocks the release of norepinephrine without interfering with axon transmission (3). In the present study, we also showed a similarity in the ThyVD for the untreated and bretylium-treated forearm sites for the three exercise intensity trials. Our results therefore confirm that the primary mechanism of control for the incremental increase in the exercise ThyVD could be an altered active vasodilatory response.

Of note, however, Smolander et al. (29) concluded that cutaneous vasodilation was significantly delayed or absent at intensities beyond 80% maximal oxygen consumption. It has been shown that the attenuation in skin blood flow is manifested as an increase in vasoconstrictor tone at these higher exercise intensities (16, 29, 30). In the present study, we did not observe a similar response at our most intense work rate (i.e., 85% \( \dot{V}O_2 \text{peak} \)). All subjects demonstrated a measurable increase in skin blood flow, albeit at a significantly elevated esophageal temperature threshold \((P < 0.05)\). Furthermore, the onset thresholds for cutaneous vasodilation were similar for both the untreated (38.01 ± 0.07°C) and bretylium-treated (38.00 ± 0.07°C) sites. On the basis of our observations, it would seem that the delayed onset of cutaneous vasodilation is the result of a delay in the active vasodilatory response rather than an increase in vasoconstrictor activity previously noted at the more intense exercise intensities. It is plausible that the absence of a delay may be more of an indication of training status. Previous studies have shown that trained subjects have a higher skin blood flow at a given core temperature, the result of a lower core temperature threshold for cutaneous vasodilation (2, 27).

### Postexercise Core Temperature Response

It has been shown that an increase in the postexercise hypotensive response, induced by exercise of increasing intensity, results in a relative decrease in the rate of heat loss and a concomitant increase in the postexercise core temperature recovery time (17). Interestingly, changes in hemodynamic response induced by the application of lower body positive pressure (+50 mmHg) in the upright position, such as an increase in mean arterial pressure [and concomitant increase in cardiac filling (23)], were shown to be sufficient to result in a relative increase in whole body heat loss and...
a concomitant increase in the rate of core temperature decay (7). Thus, in conjunction with our observation of a postexercise attenuation of cutaneous vasomotor response discussed below, it is plausible that the postexercise core temperature response is, to a large extent, influenced by nonthermoregulatory mechanisms such as baroreceptors. Further studies are required to examine the kinetics of tissue heat exchange and its relation to blood pressure/pooling and control of skin blood flow.

Postexercise Control of Skin Blood Flow

As with previous studies, we observed that exercise resulted in a postexercise increase in the threshold for cutaneous vasodilation (7, 15, 18). Additionally, we noted that the postexercise resting threshold for cutaneous vasodilation increased when exercise was performed at progressively higher intensities. Moreover, there were no differences in the esophageal temperature at which cutaneous vasodilation was initiated at either the untreated or bretylium-treated forearm sites. This observation is consistent with previous findings of a similarly mediated reflex control over the cutaneous active vasodilator system during exercise (11, 12).

Dynamic exercise is known to result in postexercise hypotension in the upright posture (1, 17, 19, 26). Research indicates that venous pooling contributes to the postexercise hypotension in an upright-seated position (26). In parallel, it has been shown that acute reductions in central venous pressure delay or decrease the rise in skin blood flow (20, 21) during heat stress and possibly results in sustained postexercise elevation in core temperature (17). A reasonable postulate is that the postexercise increase in the threshold for cutaneous vasodilation is a consequence of baroreceptor unloading via lower body venous blood pooling.

Head-down tilt and lower body positive pressure application were shown to significantly influence cutaneous vasomotor control during exercise recovery (7, 15). Specifically, baroreceptor loading during postexercise resting reversed the postexercise increase in the esophageal temperature at which cutaneous vasodilation occurs.

The blood pressure and heart rate data obtained in the present study are consistent with a postexercise hypotensive response (i.e., decrease in mean arterial pressure and a concomitant increase in heart rate) (19, 26). On the basis of our previous observations, it is plausible that our finding of a postexercise increase in the threshold for cutaneous vasodilation is consistent with a baroreceptor-mediated attenuation of postexercise skin blood flow, subsequent to lower body venous pooling. As previously indicated, acute reductions in central venous pressure have been shown to delay or decrease the rise in skin blood flow during heat stress (20, 21). The suppressive reflex on skin blood flow may reflect an alteration of vasodilator outflow due to baroreceptor unloading (10). Conversely, a decrease in skin blood flow may indicate a baroreceptor-mediated increase in 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 postexercise increase is an altered active vasodilatory response.

Summary

The present study provides additional insight into the competition between thermoregulatory and cardiovascular reflexes and the integrated response postexercise. These results demonstrate that the cutaneous vasomotor response is significantly modified during upright exercise recovery and that the effect is relatively greater during recovery after exercise of greater intensities. It is apparent that the active cutaneous vasodilator system is directly affected by the nonthermal reflex (i.e., baroreceptor reflex) adjustments postexercise. These postexercise reflex adjustments are

Table 4. Mean skin temperature at the onset threshold and sensitivity of the thermal reflex for cutaneous vasodilation

<table>
<thead>
<tr>
<th>Condition</th>
<th>Untreated</th>
<th>Bretylium Treated</th>
</tr>
</thead>
<tbody>
<tr>
<td>No-exercise</td>
<td></td>
<td></td>
</tr>
<tr>
<td>T&lt;sub&gt;sk&lt;/sub&gt;, °C</td>
<td>36.08 ± 0.07</td>
<td>36.08 ± 0.08</td>
</tr>
<tr>
<td>Slope CVC-T&lt;sub&gt;sk&lt;/sub&gt;, %/°C</td>
<td>89 ± 5</td>
<td>88 ± 5</td>
</tr>
<tr>
<td>Postexercise: 55% VO&lt;sub&gt;2&lt;/sub&gt;peak</td>
<td></td>
<td></td>
</tr>
<tr>
<td>T&lt;sub&gt;sk&lt;/sub&gt;, °C</td>
<td>36.10 ± 0.10</td>
<td>36.13 ± 0.07</td>
</tr>
<tr>
<td>Slope CVC-T&lt;sub&gt;sk&lt;/sub&gt;, %/°C</td>
<td>91 ± 5</td>
<td>90 ± 4</td>
</tr>
<tr>
<td>Postexercise: 70% VO&lt;sub&gt;2&lt;/sub&gt;peak</td>
<td></td>
<td></td>
</tr>
<tr>
<td>T&lt;sub&gt;sk&lt;/sub&gt;, °C</td>
<td>36.22 ± 0.13</td>
<td>36.23 ± 0.12</td>
</tr>
<tr>
<td>Slope CVC-T&lt;sub&gt;sk&lt;/sub&gt;, %/°C</td>
<td>86 ± 6</td>
<td>91 ± 6</td>
</tr>
<tr>
<td>Postexercise: 85% VO&lt;sub&gt;2&lt;/sub&gt;peak</td>
<td></td>
<td></td>
</tr>
<tr>
<td>T&lt;sub&gt;sk&lt;/sub&gt;, °C</td>
<td>36.26 ± 0.09</td>
<td>36.26 ± 0.12</td>
</tr>
<tr>
<td>Slope CVC-T&lt;sub&gt;sk&lt;/sub&gt;, %/°C</td>
<td>91 ± 3</td>
<td>93 ± 4</td>
</tr>
</tbody>
</table>

Values are means ± SE. CVC, cutaneous vascular conductance.
shown to result in an increase in the $\text{Th}_{VD}$ and, more specifically, in the activation of the active vasodilatory system. The actual mechanism by which this occurs remains to be elucidated. Moreover, the magnitude of the postexercise increase in the threshold for cutaneous vasodilation seems to be influenced by the intensity of exercise. We have shown an incremental alteration of the cutaneous active vasodilator response. In the present study, this was not due to an increase in adrenergic vasoconstrictor tone. Further studies are required to examine the possible role of other neurotransmitters that are known to influence the full expression of cutaneous active vasodilation during heat stress (8, 13).

We thank the subjects for assistance and participation in this study. We also thank Patience E. M. Sutton and Nicole Drost for assistance with the collection of pilot data.

DISCLOSURES

This research project was funded by the Natural Science and Engineering Research Council of Canada (grant held by G. P. Kenny).

REFERENCES


