Effect of exercise intensity on the postexercise sweating threshold

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Submitted 23 June 2003; accepted in final form 11 August 2003

Recent studies indicate that exercise induces a residual effect on thermal control, resulting in an increase in the postexercise esophageal temperature at which sweating occurs (11, 13). Although the mechanism(s) for thermoregulatory control of sweating before and during exercise has been evaluated, there remains a lack of information on its nature and role during postexercise temperature regulation. Various studies have shown that the sweating response during exercise not only involves changes in thermal factors, such as core and skin temperatures (4, 23, 24), but also nonthermal factors, including central command, baroreceptors, mechanoreceptors, and metaboreceptors (15, 24, 30). This is in contrast to passive heating at rest, in which the primary stimuli for sweating is thought to be a factor of thermal origin (25).

More recently, it has been shown that the postexercise sweating response is to some degree influenced by nonthermal baroreflex control (10). Specifically, the modification of postexercise venous pooling by lower body positive pressure results in a lowering of the resting postexercise elevation in the onset threshold for sweating. However, the mechanism of control is still unknown. Dynamic exercise is known to result in postexercise hypotension, mean arterial pressure (MAP) is reduced subsequent to the primary stimuli for sweating is thought to be a factor of thermal origin (25).

To date and to our knowledge, there have been no studies that have examined the effect of exercise intensity on the postexercise sweating 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 (18, 26) and persists for an extended period (17). Whether an increase in the postexercise hypotensive response, and therefore baroreceptor unloading similar to that with lower body negative pressure application (20), will result in a baroreceptor-mediated increase in the onset threshold for sweating remains unclear. Thus the following study was conducted to evaluate the effect of increasing exercise intensity on sweating response during the postexercise period.
METHODS

Subjects

Eight healthy and physically active male subjects volunteered and gave written consent to participate in this study, previously approved by the Research Ethics Board of the University of Ottawa.

Five to seven days before the experiments, body adiposity and peak oxygen consumption (\(\dot{V}O_2\) peak) were estimated by using total body densitometry and a progressive treadmill running protocol, respectively. The \(\dot{V}O_2\) peak value was used to select the submaximal workload for the experimental exercise phase of the study. Subjects were 22 ± 1 \(\text{SD}\) yr old, 174 ± 3 cm tall, weighed 68.5 ± 2.2 kg, and had a percent body fat of 10.9 ± 2.1%. The average aerobic capacity was 63.3 ± 2.1 \(\text{ml} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}\).

Instrumentation

Central body temperature (esophageal temperature) was monitored continuously by using 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 Engineer- 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% (9).

Oxygen consumption was measured by using an automated metabolic analyzer (MedGraphics, St. Paul, MN). MAP was calculated from the electrical integration of the pulsatile blood pressure signal obtained noninvasively, from the middle digit of the left hand (Ohmeda, Finapres 2300) referenced at the third intercostal space. These blood pressure data were recorded with the Finapres servo control on and stored continuously at 5-s intervals. Heart rate (HR) 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).

Sweat rate was estimated from a 5.0-cm² capsule placed on the upper back and ventilated with anhydrous compressed air. The relative humidity of the effluent air was measured at known barometric pressure by using the readings from an Omega HX93 humidity and temperature sensor (Omega Engineering, Stamford, CT). Sweat rate was calculated from the product of the difference in water content between effluent and influent air and the flow rate. This value was normalized for the skin surface area under the capsule (expressed in \(\text{mg} \cdot \text{min}^{-1} \cdot \text{cm}^{-2}\)).

Core temperature, skin temperature, and sweat rate 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 balanced 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 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. 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 for a period no less than 90 min. A schematic representation of the experimental timeline is presented in Fig. 1.

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 \(\dot{V}O_2\) 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 upright seated (no exercise) or were placed similarly seated (exercise) for a 20-min resting recovery at an ambient temperature of 22°C.

Subjects then donned a liquid-conditioned suit (Med-Eng Systems, Ottawa, ON, Canada) covering the torso, arms, and head. A 20°C water perfusion was started through the liquid-conditioned suit by using a temperature-controlled circulation bath (Endocal, Neslab; and model 200-00, Micropump, Vancouver, WA). This procedure was performed to control and stabilize skin and core temperature to baseline resting values before whole body warming (~10 min).

Warming phase. Mean skin temperature was then increased at a rate of ~4.0 ± 0.8°C/h as the water circulating through the suit was progressively increased to 48°C (~40 min). Whole body warming continued until sweating achieved a sustained elevated value. To ensure that the subjects were euhydrated during the experimental trial, they were required to ingest 0.125 liter of water every 30 min for the duration of the experimental trial.

| Baseline resting, 90 min upright seated rest, T<sub>amb</sub> of 22°C. | No-Exercise 15 min rest | Exercise 15 min @ 55, 70 or 85% of \(\dot{V}O_2\) peak | 20 min upright seated recovery, T<sub>amb</sub> of 22°C. | LCS perfused with 20°C water, Duration: ~10 min. | LCS perfused with 48°C water and continued until onset of sweating, Duration: ~40 min. |

Fig. 1. Experimental protocol timeline. T<sub>amb</sub>, ambient temperature; \(\dot{V}O_2\) peak, peak oxygen consumption; LCS, liquid-conditioned suit.
POSTEXERCISE SUDOMOTOR CONTROL

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

<table>
<thead>
<tr>
<th>Condition</th>
<th>No Exercise</th>
<th>Light (55% (\dot{V}O_2_{peak}))</th>
<th>Moderate (70% (\dot{V}O_2_{peak}))</th>
<th>Intense (85% (\dot{V}O_2_{peak}))</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>MAP, mmHg</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Baseline resting</td>
<td>93 ± 1</td>
<td>92 ± 1</td>
<td>92 ± 2</td>
<td>93 ± 2</td>
</tr>
<tr>
<td>End of 20-min postexercise recovery</td>
<td>93 ± 1</td>
<td>88 ± 1*†</td>
<td>85 ± 1*†</td>
<td>83 ± 2*†</td>
</tr>
<tr>
<td><strong>HR, beats/min</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Baseline resting</td>
<td>72 ± 2</td>
<td>70 ± 2</td>
<td>73 ± 2</td>
<td>72 ± 2</td>
</tr>
<tr>
<td>End of 20-min postexercise recovery</td>
<td>74 ± 2</td>
<td>88 ± 2*†</td>
<td>97 ± 2*†</td>
<td>109 ± 3*†</td>
</tr>
<tr>
<td><strong>(T_{es}), °C</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Baseline resting</td>
<td>36.68 ± 0.03</td>
<td>36.68 ± 0.07</td>
<td>36.69 ± 0.05</td>
<td>36.70 ± 0.06</td>
</tr>
<tr>
<td>End of 20-min postexercise recovery</td>
<td>36.66 ± 0.04</td>
<td>37.00 ± 0.06*†</td>
<td>37.20 ± 0.06*†</td>
<td>37.30 ± 0.08*†</td>
</tr>
<tr>
<td><strong>(T_{sk}), °C</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Baseline resting</td>
<td>33.64 ± 0.17</td>
<td>33.80 ± 0.18</td>
<td>33.77 ± 0.15</td>
<td>33.69 ± 0.11</td>
</tr>
<tr>
<td>End of 20-min postexercise recovery</td>
<td>33.78 ± 0.20*†</td>
<td>33.94 ± 0.32*†</td>
<td>33.98 ± 0.28*†</td>
<td>33.95 ± 0.23*†</td>
</tr>
</tbody>
</table>

Values are means ± SE. Note: postexercise data represent the values taken at 20 min postexercise before the subject donned the liquid-conditioned suit. MAP, mean arterial pressure; HR, heart rate; \(T_{sk}\), mean skin temperature; \(T_{es}\), esophageal temperature; \(\dot{V}O_2_{peak}\), peak \(\dot{V}O_2\) consumption. *Significantly different from baseline resting, \(P < 0.05\). †Significantly different from no exercise, \(P < 0.05\).

Data and Statistical Analysis

The esophageal temperature threshold for sweating was attained when a rapid increase in sweat rate was observed in at least three consecutive measurements (10, 16). 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

Resting HR, MAP, and esophageal and mean skin temperatures were similar for all conditions during baseline resting (Table 1).

Prewarming Phase

End-exercise HR was 152 ± 2, 173 ± 2, and 187 ± 3 beats/min for light, moderate, and intense exercise, respectively. HR remained significantly elevated \((P < 0.05)\) above baseline rest values at the end of the 20-min postexercise recovery period by 18 ± 2, 24 ± 2 and 37 ± 2 beats/min for the light, moderate, and intense exercise, respectively \((P < 0.05)\). In contrast, for all exercise conditions, MAP remained significantly reduced relative to the baseline resting value \((P < 0.05)\), although there was a greater overall reduction with increasing exercise intensity. No differences were noted during the no-exercise trial.

Exercise resulted in 0.64 ± 0.03, 1.00 ± 0.05, and 1.58 ± 0.07°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 0.24 ± 0.03, 0.43 ± 0.05, and 0.60 ± 0.06°C at the end of the 20-min 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 and mean skin temperatures for the no-exercise condition remained unchanged from baseline resting values.

Warming Phase

Core and mean skin temperatures were similar to baseline values before the start of the whole body warming maneuver. Mean skin temperature was increased at the same rate of ~4.0 ± 0.8°C/h for all subjects in all conditions.

Sweating response. The postexercise threshold for sweating for all exercise conditions was significantly elevated above the no-exercise condition \((P < 0.05)\). The effect was relatively greater during recovery after exercise of greater intensities. These values were 0.11 ± 0.02, 0.23 ± 0.01, and 0.33 ± 0.02°C above no-exercise for the light, moderate, and intense exercise, respectively \((P < 0.05; \text{Fig. 2})\). The

![Fig. 2. Esophageal temperature at onset for sweating for no-exercise and postexercise resting protocols as measured after 15 min of exercise performed at 55 (light), 70 (moderate), and 85% (intense) of \(\dot{V}O_2_{peak}\). Values are means ± SE. Note: onset of sweating was measured a minimum of 30 min posttreatment (i.e., during the whole body warming phase). Exercise resulted in a significant increase in the threshold for sweating above no-exercise resting (*\(P < 0.05\)). †Significant difference from light exercise, \(P < 0.05\).](http://jap.physiology.org/content/95/6/2357)
relative increase in esophageal temperature above baseline resting at sweating onset for all conditions is presented in Fig. 3. Mean skin temperatures at the onset threshold for sweating were similar for all conditions (Table 2).

**DISCUSSION**

The major finding of the present study was that an increase in exercise intensity resulted in a residual increase in the postexercise esophageal temperature at which sweating occurred. We did note that there was a greater hypotension as a function of exercise intensity as measured at the end of the 20-min exercise recovery. Thus it is plausible that the increase in the postexercise threshold for sweating may be related to postexercise hypotension. MAP remained significantly below baseline resting for the duration of the 20-min postexercise resting period for all exercise conditions, with the intense exercise showing a greater overall reduction.

Exercise caused an increase in postexercise threshold for sweating consistent with previous reports. Kenny et al. (11, 13) previously demonstrated a ~0.2°C increase in the onset threshold for sweating after moderate-intensity exercise (60–65% maximal oxygen consumption). Lopez et al. (16) reported no change in the postexercise sweating threshold compared with preexercise values. However, their subjects had been infused with 3–5 liters of fluid over a 2.5-h period. It is likely that the similarities in pre- and postexercise sweating threshold in the study of Lopez et al. are the result of opposing effects of hyperhydration and exercise on sudomotor activity. Moderate hyperhydration (i.e., 1.2% body wt increase) has been shown to lower the onset threshold for sweating (5).

Studies have shown that hypohydration increases the onset threshold for sweating. It possible therefore that the measured increase in the onset threshold for sweating may be subsequent to a change in the hydration status of our subjects. Although the hydration status was not verified, it is unlikely that any significant hypohydration occurred. First, our subjects were permitted water ad libitum before arriving at the laboratory, and, second, to ensure that the subjects were euhydrated during the experimental trial, they were required to ingest 0.125 liter of water every 30 min for the duration of the experimental trial. In this study, the short duration of exercise (only 15 min) performed in a cooler environment (22°C) with unrestricted water intake is unlikely to have caused significant water loss even under the intense exercise condition.

Studies have shown that the sweating response during exercise involves not only changes in core and skin temperature response (4, 24) but also nonthermal factors such as central command, baroreceptors, mechanoceptors, and metaboreceptors (15, 24, 30). This is in contrast to passive heating at rest, in which the primary stimuli for sweating have generally been thought to be thermal in origin (25). Recently, Carter et al. (2) suggested that the postexercise sweating response may also be modulated by nonthermoregulatory mechanisms. Specifically, they observed that the postexercise decrease in sweat rate during the initial 5 min of recovery was attenuated when exercise was followed by active recovery. However, given that the increase in sweat rate during active vs. inactive recovery was not paralleled by a difference in arterial blood pressure, it is unlikely that the increase in sweat rate was due to differences in arterial baroreceptor unloading. The authors hypothesized that the differences may be due to greater central command associated with unloaded cycling during active recovery. In contrast, Jackson and Kenny (10) showed that baroreceptor loading during postexercise resting attenuates the postexercise esophageal temperature at which sweating occurs. They concluded that the postexercise increase in the threshold for sweating is consistent with a baroreceptor mediation of postexercise sudomotor activity, subsequent to lower body venous pooling. Their results are consistent with previous observations of a similar decrease in sweat rate with baroreceptor unloading during exercise (20, 21). It is important to note, however, that the study by Carter et al. (2) examined the postexercise sweating response during the first 5 min of exercise recovery, whereas Jackson and Kenny (10) measured the sweating response ~65 min postexercise. Thus it is plausible that, whereas sweat rate is influenced by the nonthermoregulatory influence of

<table>
<thead>
<tr>
<th>No Exercise</th>
<th>Light</th>
<th>Moderate</th>
<th>Intense</th>
</tr>
</thead>
<tbody>
<tr>
<td>T_s, °C</td>
<td>36.80 ± 0.03*</td>
<td>36.90 ± 0.03*</td>
<td>36.60 ± 0.03*</td>
</tr>
<tr>
<td>T_e, °C</td>
<td>36.16 ± 0.03*</td>
<td>36.14 ± 0.03*</td>
<td>36.24 ± 0.03*</td>
</tr>
</tbody>
</table>

Values are means ± SE. *Significantly different from no exercise, $P < 0.05$. †Significantly different from moderate exercise, $P < 0.05$.

Fig. 3. Increase in esophageal temperature above baseline resting at which sweating occurred for no-exercise and postexercise resting protocols as measured after 15 min of exercise performed at 55% (light), 70% (moderate), and 85% (intense) of $V_{O2\text{peak}}$. Values are means ± SE. Note: onset of sweating was measured a minimum of 30 min posttreatment (i.e., during the whole body warming phase). Values were significantly elevated above baseline resting (*$P < 0.05$). †Significant difference from light exercise, $P < 0.05$.  

J Appl Physiol • VOL 95 • DECEMBER 2003 • www.jap.org
central command during the initial stages of exercise recovery, baroreceptor modulation of sweating may predominate later in exercise recovery.

It is well documented that dynamic exercise results in postexercise hypotension in the upright posture (3, 6, 7, 14, 18, 26, 27). A number of studies have shown that the duration of the postexercise hypotension in normotensive individuals may vary between 1 and 2 h after exercise (17) and that the magnitude of the response is influenced by the intensity of exercise (12). The blood pressure and HR data are consistent with a postexercise hypotensive response. One contributing factor in postexercise hypotension is that a significant quantity of blood remains pooled in the lower limbs during recovery (3, 14, 26). Venous pooling reduces central blood volume, which in turn decreases cardiac filling pressure and lowers systemic arterial blood pressure. It is plausible, therefore, that the increase in the postexercise threshold for sweating is a consequence of baroreceptor unloading via lower body venous blood pooling similar to that response previously reported during exercise (20). The observed increase in the postexercise threshold for sweating, which was preceded by a postexercise hypotension (measured at the end of the 20-min recovery), supports the hypothesis of baroreceptor-mediated modulation of postexercise sudomotor activity.

Bini and coworkers (1) suggested that changes in blood pressure may modulate sweat gland activity. Their observation was based on the fact that skin sympathetic nerve recordings from sudomotor fibers showed cardiac rhythmicity. Macefield and Wallin (19) also found that sudomotor neuronal discharge is modulated by baroreflexes. Mack et al. (20, 21) showed a greater increase in the esophageal temperature required to elicit sweating during exercise with baroreceptor unloading even though they did not measure skin sympathetic nerve activity. Others studies, however, have shown that skin sympathetic nerve activity and sweat rate are not modulated by arterial baroreflexes (28, 29). Although we did not directly measure skin sympathetic nerve activity, there is a potential for confounding effects of non-baroreflex-mediated origin on the postexercise thermal response to sweating yet to be studied.

In summary, these results demonstrate that the sweating response during upright recovery, measured during the warming phase 30 min postexercise, is significantly modified by exercise intensity. Given the measured reduction in blood pressure postexercise, it is possible that sudomotor activity is influenced by nonthermal baroreceptor reflex adjustments postexercise.

We thank the subjects for assistance and participation in this study.

DISCLOSURES

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

REFERENCES