Control of cutaneous vascular conductance and sweating during recovery from dynamic exercise in humans

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Submitted 11 November 2003; accepted in final form 4 February 2004

Changes in SkBF and sweating can be initiated by thermoregulatory reflexes, but they are also subject to nonthermoregulatory reflexes such as those of central command, baroreflexes, and muscle mechanoreceptors and metaboreceptors (10, 11, 25, 30). At the cessation of exercise, however, dramatic changes in MAP occur secondary to the removal of central command and to metaboreceptor and mechanoreceptor feedback. Thus SkBF and sweating may still be subject to baroreceptor control during exercise recovery (3). This is plausible given that previous evidence exists for baroreceptor modulation of SkBF and sweating (4, 12, 13, 20).

Recent work has shown that recovery mode can influence the responses of MAP, SkBF, and sweating. Carter et al. (2) first demonstrated the relative roles of the skeletal muscle pump and central command in the control of cardiovascular responses by using active, passive, and inactive recovery modes after exercise that was 3 min in duration. The exercise time was chosen to avoid initiating any significant thermoregulatory responses. Subsequently, Carter et al. (3) examined the effect of active and inactive recovery from 15 min of dynamic exercise on SkBF and sweating responses. They observed that SkBF and sweating are preserved during 5 min of active recovery compared with inactive recovery. Both studies utilized a 5-min recovery period. The results of these investigations give rise to a number of questions concerning exercise recovery. First, many cardiovascular and thermoregulatory adjustments occur after 5 min into recovery. For example, Kilgour et al. (16) and Brown et al. (1) have reported observations on seated exercise recovery for 60 min postexercise, whereas Thoden et al. (33) have reported that SkBF and sweating have returned to preexercise levels 20 min after exercise and that this response is accompanied by postexercise hypotension when exercise is performed at a sufficient intensity. The effect of recovery mode on cardiovascular and thermoregulatory responses during a longer recovery period is currently unknown. Second, Carter et al. (3) demonstrated that active recovery can preserve SkBF and sweating responses. During active recovery, both the skeletal muscle pump and central command are activated. A passive recovery mode is thought to include the skeletal muscle pump without the involvement of central command (2). Thus the relative effect of passive and active recovery modes, and by extension the role of central command, in the control of SkBF and sweating has not been evaluated during exercise recovery.

Therefore, we chose to test the effect of passive recovery in addition to active and inactive recovery on cardiovascular and...

Recovery from dynamic exercise is associated with significant cardiovascular and thermoregulatory adjustments (16). During sustained dynamic exercise, metabolic heat production tends to increase body heat content and results in the activation of the thermoregulatory responses of skin blood flow (SkBF) and sweating. Sweat rate can increase to as much as 2,000 g/h (34) while elevated cutaneous circulation may result in increased heat dissipation (22). During exercise mean arterial pressure (MAP) is elevated secondary to increased central command activity, metaboreflexes, mechanoreflexes, baroreflexes, and the skeletal muscle pump (17, 18, 27–29).
thermoregulatory responses during extended recovery from 15 min of dynamic exercise. We hypothesized that the active recovery mode would be most effective in attenuating the fall in SkBF, sweating, and MAP compared with the passive and inactive recovery modes.

METHODS

Subjects. Six healthy, physically active men volunteered and gave written consent to participate in this study. The study was approved by the Research Ethics Board at the University of Ottawa. Subjects were 23 ± 1 (SE) yr old and 174 ± 2 cm tall, weighed 77 ± 3 kg, and had a mean VO_{2} \text{peak} of 48.4 ± 2.2 ml·kg⁻¹·min⁻¹.

Measurements. 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). MAP was estimated from the integration of a noninvasive recording of blood pressure at the middle digit of the left hand (Finapres 2300, Ohmeda, Madison, WI) fixed at heart level (the third intercostal space). The Finapres system is based on the Penaz volume clamp method (dynamic unloaded arterial wall principle). MAP was verified periodically throughout the protocol by auscultation.

Pulmonary oxygen consumption was estimated by using a metabolic cart (model CPX/D, Medgraphics, St. Paul, MN) during VO_{2} assessment preceding the experimental trials. Cardiac output (CO) was estimated by using the CPX/D computerized version of the carbon dioxide-rebreathing technique of Defaere (5). It has been shown that Doppler-derived aortic blood flow (CO) measurements correlate well with the indirect carbon dioxide-rebreathing method (6). The Defaere method has also been shown to work well in “un-steady-state” testing (8). Stroke volume (SV) was calculated as CO/HR. Total peripheral resistance (TPR) was calculated as MAP/CO.

SkBF was estimated by using laser-Doppler velocimetry (PeriFlux System 5000, Main control unit: PF5010 LDPM, Function unit; Perimed, Stockholm, Sweden) at the right midanterior forearm and over the right superior portion of the trapezius. The laser-Doppler flow probes (PR 401 angled probe, Perimed) were taped to cleaned skin, in an area that did not appear by visual inspection to be overly vascular and from which consistent readings were noted (19). Cutaneous vascular conductance (CVC) was calculated as the ratio of laser-Doppler flow to MAP. At the end of the experiment, local skin temperature at the skin sites were raised to 42°C until peak CVC was measured (~30 min) (32). A heating element (PF 5020 temperature unit, Perimed) housed the laser-Doppler flow probe, and it was then activated to elevate local skin temperature to 42°C. Peak CVC was determined as a sustained elevated plateau in local SkBF. CVC data are presented as a percentage of maximal CVC as determined by local heating. All SkBF measures were taken in the period preceding rebreathing to avoid causing fluctuations in SkBF data at each time point. SkBF measures were recorded from the right midanterior forearm such that the arm was level with the heart.

Sweat rate was measured by using a 5.0-cm² ventilated capsule placed over the medial inferior aspect of the trapezius muscle. Anhydrous compressed air was passed through the capsule and over the skin surface (Brooks 5850, mass flow controller, Emerson Electric, Hefield, PA). The vapor density of the effluent air was calculated from the relative humidity and temperature measured by using the Omega HX93 humidity and temperature sensor (Omega Engineering, Stanford, CT). Sweat rate was defined as the product of the difference in water content between effluent and influent and the flow rate. The flow rate through the capsule was 1.0 l/min. The sweat rate value was adjusted for skin surface area under the capsule (expressed in mg·min⁻¹·cm⁻²).

Central body temperature [esophageal temperature (T_{es})] 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, whereby the tip of the thermocouple is estimated to be at the level of the left atrium (21). Skin temperature was recorded at 11 sites (model FR-025-TH44018-6, Concept Engineering, Old Saybrook, CT). The area-weighted mean skin temperature was estimated by calculating the weighted mean value, by using 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% (7).

Data analysis. For the continuous measures of temperature, SkBF, MAP and sweat rate a 30-s average value was determined at each of the following time points: baseline resting, end exercise, and 2, 5, 8, 12, and 15 min postexercise. CO, SV, HR, and TPR were also recorded at baseline resting, end exercise, 2, 5, 8, 12, and 15 min postexercise. As previously indicated, all SkBF measures were taken in the period preceding rebreathing to avoid causing fluctuations in SkBF data at each time point. All values represent the means ± SE for six subjects. A two-way ANOVA with repeated measures was used to...
compare the values with the two main factors of the ANOVA being time and recovery mode. When significant main effects were observed, a Tukey's post hoc test was performed. Differences were considered significant when \( P < 0.05 \).

RESULTS

Back and forearm CVC results are presented graphically in Fig. 1. Exercise resulted in similar increases in CVC across conditions for each site. In the active and passive recovery modes, CVC was higher than during the inactive mode at both sites (\( P < 0.05 \); Fig. 1). Sweat rate results are presented in Fig. 2. Sweat rate was higher in the active and passive recovery modes than during the inactive mode throughout recovery (\( P < 0.05 \); Fig. 2). After 8 min of recovery, sweat rates for all conditions were significantly different from each other (\( P < 0.05 \); Fig. 2).

MAP reached similar levels under all conditions during exercise. MAP values were greater during active and passive recovery modes than during inactive recovery (\( P < 0.05 \); Table 1). TPR during active recovery was significantly lower than both passive and inactive recovery modes (\( P < 0.05 \); Table 1). Values for TPR were not significantly different between the passive and inactive modes. CO was significantly lower during inactive recovery than both the passive and active modes (\( P < 0.05 \); Table 1). At the 2-min mark only into recovery were CO values different among all recovery modes (\( P < 0.05 \); Table 1). SV was higher throughout the active and passive recovery modes than the inactive mode (\( P < 0.05 \); Table 1). HR remained significantly higher in the active mode than passive between 2 and 12 min into recovery (\( P < 0.05 \); Table 1), whereas the HR in the active mode remained higher than inactive only at 2 and 5 min of recovery.

Temperature data are presented in Table 2. No differences in \( T_{es} \) and skin temperature were observed among recovery modes.

DISCUSSION

The major finding of this investigation is that both active and passive exercise recovery modes are effective in attenuating the fall in CVC, sweat rate, and MAP compared with an inactive recovery mode. Given that passive recovery served as a control for central command involvement, the results suggest that attenuating the fall in MAP through action of the skeletal muscle pump is the main determinant in preserving CVC, although the role of mechanoreceptors cannot be excluded. The different sweat rate responses among all three recovery modes suggest that factors such as central command and mechanoreceptors influence sweat rate in addition to those influences associated with MAP. Although the precise mechanism explaining the different CVC and sweat rate responses among recovery modes is unknown, these data suggest that attenuating

Fig. 1. Effect of active (○), passive (●), and inactive (▲) recovery after 15 min of moderate-intensity cycle ergometry on cutaneous vascular conductance (CVC) from upper back (A) and forearm (B) skin. Values are means ± SE for 6 subjects. *Significant difference between passive recovery, \( P < 0.05 \). †Significant difference from active recovery, \( P < 0.05 \).

Fig. 2. Effect of active (○), passive (●), and inactive (▲) recovery after 15 min of moderate-intensity cycle ergometry on sweating. Values are means ± SE for 6 subjects. *Significant difference between passive recovery, \( P < 0.05 \). †Significant difference from active recovery, \( P < 0.05 \).
the fall in MAP, and thus baroreceptor unloading, may be a factor in addition to mechanoreceptor activation during active and passive recovery modes.

The results of the study are not totally consistent with our original hypothesis. That is, whereas active recovery attenuated the fall in CVC more than inactive, it was not more effective than passive recovery and was thus not the most effective in attenuating the fall in CVC. With regard to sweat rate, active recovery was the most effective in attenuating the fall in CVC only beyond 8 min postexercise.

Carter et al. (3) postulated that an active recovery may sustain CVC above inactive recovery either due to 1) less arterial and/or cardiopulmonary baroreceptor unloading and/or 2) an effect of central command to sustain CVC and sweat rate during active recovery. It is well known that thermoregulatory effectors of SkBF and sweating are modulated by thermoregulatory controls, but it has also been reported that such responses are subject to nonthermoregulatory baroreceptor control (10, 11, 25). Our MAP data suggest that arterial baroreceptor unloading was much greater in the inactive recovery than the passive and active modes. This is in contrast to Carter et al., who observed no separation of MAP responses between active and inactive recovery modes during a 5-min recovery period. Second, SV in the passive and active recovery modes was much higher than inactive recovery, suggesting greater central blood volume, as confirmed by Carter et al. (2, 3) using thoracic electrical impedance techniques, and thus less cardiopulmonary baroreceptor unloading. Therefore, the greater reduction in CVC observed with inactive recovery may be due to greater cardiopulmonary and/or arterial baroreceptor unloading. During active recovery, it is thought that both central command and the skeletal muscle pump are engaged (2), and thus the effect of central command on CVC cannot be determined. Our study demonstrates that, with a reduced influence of descending signals from the motor cortex during a passive recovery mode, CVC is maintained to the same degree as during active recovery. This observation suggests that attenuating the fall in MAP during exercise recovery through action of the skeletal muscle pump is the major contributor to the observed elevation in CVC and that central command contribution is minimal. A recent study by Wilson et al. (36) supports this hypothesis. In their study, they performed active and inactive recovery modes in the supine position, thus removing the baroreceptor unloading effect of being upright on the cycle ergometer. They observed no difference in CVC between recovery modes. However, afferent feedback from changes in joint angle and muscle stretch may also be influencing CVC.

Carter et al. (3) showed that with active recovery CVC did not change appreciably from exercise values, suggesting that central command does not play a critical role in CVC because central command input is much less during active recovery than during peak exercise. CVC in our study was not maintained at exercise levels during the first 5 min of active or passive recovery, and the values continued to fall throughout recovery. Given that the skeletal muscle pump and the similar SV and MAP values are common threads in our active and passive recovery modes.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Recovery Mode</th>
<th>Baseline</th>
<th>Exercise</th>
</tr>
</thead>
<tbody>
<tr>
<td>CO, l/min</td>
<td>Active</td>
<td>6.0 ± 0.2</td>
<td>20.6 ± 0.9</td>
</tr>
<tr>
<td></td>
<td>Passive</td>
<td>6.0 ± 0.1</td>
<td>20.9 ± 0.5</td>
</tr>
<tr>
<td>SV, ml</td>
<td>Active</td>
<td>85.7 ± 5</td>
<td>130.6 ± 4</td>
</tr>
<tr>
<td></td>
<td>Passive</td>
<td>88.5 ± 3</td>
<td>131.9 ± 4</td>
</tr>
<tr>
<td>HR, beats/min</td>
<td>Active</td>
<td>94.0 ± 2.4</td>
<td>132.1 ± 2.2</td>
</tr>
<tr>
<td></td>
<td>Passive</td>
<td>91.5 ± 2.4</td>
<td>131.9 ± 2.4</td>
</tr>
<tr>
<td>MAP, mmHg</td>
<td>Active</td>
<td>92.4 ± 0.7</td>
<td>114.2 ± 2.2</td>
</tr>
<tr>
<td></td>
<td>Passive</td>
<td>92.4 ± 0.7</td>
<td>113.3 ± 1.9</td>
</tr>
<tr>
<td>TPR, mmHg(1)/l/min</td>
<td>Active</td>
<td>15.4 ± 0.5</td>
<td>5.5 ± 0.2</td>
</tr>
<tr>
<td></td>
<td>Passive</td>
<td>15.4 ± 0.2</td>
<td>5.4 ± 0.1</td>
</tr>
</tbody>
</table>

Values are means ± SE for 6 subjects. CO, cardiac output; SV, stroke volume; HR, heart rate; MAP, mean arterial pressure; TPR, total peripheral resistance. *Different from other 2 recovery modes, P < 0.05. †Different from passive mode, P < 0.05.

Table 2. Thermal responses to recovery modes

<table>
<thead>
<tr>
<th>Measure</th>
<th>Recovery Mode</th>
<th>Baseline</th>
<th>Exercise</th>
</tr>
</thead>
<tbody>
<tr>
<td>(T_{es}, ^\circ C)</td>
<td>Active</td>
<td>36.95 ± 0.11</td>
<td>37.68 ± 0.14</td>
</tr>
<tr>
<td></td>
<td>Passive</td>
<td>36.87 ± 0.07</td>
<td>37.63 ± 0.17</td>
</tr>
<tr>
<td></td>
<td>Inactive</td>
<td>36.94 ± 0.08</td>
<td>37.64 ± 0.17</td>
</tr>
<tr>
<td>(T_{as}, ^\circ C)</td>
<td>Active</td>
<td>34.32 ± 0.18</td>
<td>35.41 ± 0.12</td>
</tr>
<tr>
<td></td>
<td>Passive</td>
<td>34.50 ± 0.18</td>
<td>35.48 ± 0.11</td>
</tr>
<tr>
<td></td>
<td>Inactive</td>
<td>34.09 ± 0.25</td>
<td>35.12 ± 0.20</td>
</tr>
</tbody>
</table>

Values are means ± SE for 6 subjects. \(T_{es}\), esophageal temperature; \(T_{as}\), mean skin temperature.
passive recovery modes suggests that attenuating the baroreceptor unloading effect associated with exercise cessation is a major factor in sustaining CVC. However, it cannot be ignored that mechanoreceptor activation plays a role in the response. It has been noted that passive and loadless cycling may activate mechanoreceptors (24).

Evidence also exists for nonthermoregulatory modulation of sweating (30). There is a paucity of information regarding baroreceptor control of sweating during recovery from exercise. Jackson and Kenny (9) have demonstrated a reduction in the postexercise resting threshold for sweating with the application of lower body positive pressure. More recently, Kenny et al. (14) have demonstrated that the postexercise sweating threshold is increased in parallel with the magnitude of post-exercise hypotension observed. That is, with a greater postexercise hypotensive response the sweating threshold is delayed. At rest, however, some researchers did not observe decreases in sweating with baroreceptor unloading (35, 37), whereas others have observed a reduction in the sweat rate-Tes slope with baroreceptor unloading at rest (31) and during exercise (20). As with CVC, the higher MAP and SV values observed in the active and passive modes would suggest that both arterial and cardiopulmonary baroreceptors may have influenced the observed differences. Another possible factor in our observed differences in sweat rate is the effect of central command. In the first 5 min of recovery, both active and passive sweat rates were higher than inactive. At 8 min, however, sweat rates were different among all recovery modes. This observation could indicate a differential control of sweating during recovery such that, in active recovery, central command and factors associated with the skeletal muscle pump contribute to sweat rate, whereas passive recovery includes only the skeletal muscle pump component. In passive recovery, our SV and MAP data suggest that cardiopulmonary and arterial baroreceptor stimulation would be similar but without the descending central command input sweat rate falls below the active mode value but still remains elevated above inactive. In support of our observations, results from a recent study by Wilson et al. (36) suggest that factors such as mechanoreceptor stimulation and central command may affect sweat rate during recovery, whereas baroreceptor influence would be minimal. However, on the basis of our findings, we cannot rule out the possibility of arterial baroreceptor influence given the separation in our MAP responses.

Although our study suggests that nonthermoregulatory influences on CVC and sweating are at play during recovery from dynamic exercise, there remains the possible confounding effect of differential thermal stimuli between recovery modes. One might suggest that blood temperature at the level of the hypothalamus may influence the responses of CVC and sweating. Although this may be true, our Tes temperature data, which are representative of arterial blood temperature at the level of the heart, indicate that the stimuli did not differ between recovery modes. Additionally the contribution of skin temperature to eliciting centrally mediated responses appeared similar among recovery modes.

Differences in muscle metabolic heat production and thus differences in intramuscular temperature between recovery modes must also be considered. To the best of our knowledge, only Kenny et al. (15) have characterized intramuscular temperatures during inactive recovery from dynamic exercise. Although it is likely that some metabolic heat production occurs during active recovery and possibly during passive recovery, it is unlikely that significant muscle temperature changes occur with passive recovery mode. At present, it is unknown what role, if any, thermoreceptive afferents in the muscle play in CVC and sweating responses (26).

We did not compare oxygen consumption between recovery modes in this study. As suggested by Wilson et al. (36), even if heat production was higher in the active modes, it was not great enough to produce changes in hypothalamic or mean skin temperature sufficient to modify CVC or sweating. If heat production had been significantly greater, an increase in CVC and sweating may have resulted during the 15-min recovery mode.

Our results show that augmenting venous return through active and passive recovery modes attenuates the fall in MAP and CVC over the inactive mode during recovery from dynamic exercise. The fall in sweat rate during exercise recovery was dependent on each recovery mode. Taken together with recent results from Wilson et al. (36), our data suggest that attenuating the baroreceptor unloading effect of inactive recovery by activation of the skeletal muscle pump preserves CVC and not central command. The sweat rate data cannot exclude the role of baroreceptors given MAP differences between recovery conditions. However, this study’s data suggest that both central command and mechanoreceptors can modulate postexercise sweat rate responses. These results have potential application for those involved in occupations or tasks in hot environments. Specifically, passive recovery could be used to maintain blood pressure and heat loss responses in a hyperthermic individual who may not be capable of completing an active recovery.

ACKNOWLEDGMENTS

The authors thank Jane Murrin for assistance in the laboratory. Preliminary results from this study were presented in an oral presentation at the annual meeting of the Canadian Society for Exercise Physiology, Niagara-on-the-Lake, Ontario, Canada, 2003.

GRANTS

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

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