Nonthermoregulatory control of cutaneous vascular conductance and sweating during recovery from dynamic exercise in women

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Journeay, W. Shane, Francis D. Reardon, Natalie H. McInnis, and Glen P. Kenny. Nonthermoregulatory control of cutaneous vascular conductance and sweating during recovery from dynamic exercise in women. J Appl Physiol 99: 1816–1821, 2005. First published July 21, 2005; doi:10.1152/japplphysiol.00497.2005.—The purpose of the study was to examine the effect of 1) active (loadless pedaling), 2) passive (assisted pedaling), and 3) inactive (motionless) recovery modes on mean arterial pressure (MAP), cutaneous vascular conductance (CVC), and sweat rate during recovery after 15 min of dynamic exercise in women. It was hypothesized that an active recovery mode would be most effective in attenuating the fall in MAP, CVC, and sweating during exercise recovery. Ten female subjects performed 15 min of cycle ergometer exercise at 70% of their predetermined peak oxygen consumption followed by 20 min of 1) active, 2) passive, or 3) inactive recovery. Mean skin temperature (Tsk), esophageal temperature (Tes), skin blood flow, sweating, cardiac output (CO), stroke volume (SV), heart rate (HR), total peripheral resistance (TPR), and MAP were recorded at baseline, end exercise, and 2, 5, 8, 12, 15, and 20 min postexercise. Cutaneous vascular conductance (CVC) was calculated as the ratio of laser-Doppler blood flow to MAP. In the active recovery mode, CVC, sweat rate, MAP, CO, and SV remained elevated over inactive values (P < 0.05). The passive mode was equally as effective as the active mode in maintaining MAP. Sweat rate was different among all modes after 12 min of recovery (P < 0.05). TPR during active recovery remained significantly lower than during recovery in the inactive mode (P < 0.05). No differences in either Tes or Tsk were observed among conditions. The results indicate that CVC can be modulated by central command and possibly cardiopulmonary baroreceptors in women. However, differences in sweat rate may be influenced by factors such as central command, mechanoreceptor stimulation, or cardiopulmonary baroreceptors.

skin blood flow, sweat rate, gender, mechanoreceptors, central command

CONTROL OF THE HEAT LOSS RESPONSES of sweating and skin blood flow (SkBF) has been studied extensively during exercise. Although it is well known that during exercise SkBF and sweating can be initiated via hypothalamic activity, there is evidence that they are also subject to nonthermoregulatory influences such as central command, baroreflexes, and muscle mechanoreceptors and metaboreceptors (13, 14, 25, 26). Through efferent and afferent feedback, these mechanisms also stimulate the cardiorespiratory centers of the brain stem and therefore also influence the control of mean arterial pressure (MAP). At the cessation of exercise, there is a reduction in the nonthermoregulatory activity of central command and in muscle mechanoreceptor and metaboreceptor feedback. Marked reductions in these inputs are also accompanied by a drop in MAP from exercise values. Thus, immediately postexercise, SkBF and sweating may still be under baroreceptor influence during an inactive recovery (4).

A number of studies have attempted to differentiate the relative roles of central command, mechanoreceptors, and baroreceptors in modulating SkBF and sweating during exercise recovery as a function of recovery mode. Cardiovascular responses to active, passive, and inactive recovery modes were first studied by Carter et al. (3). Subsequently, these recovery modes have been used to study the nonthermal influences on heat-loss responses. The mechanisms of the different recovery modalities are the following: 1) during active recovery (loadless pedaling), skeletal muscle pump/mechanoreceptors and central command are activated; 2) during passive cycling, mechanoreceptors are stimulated without the involvement of central command (3, 23, 24, 33); and 3) during inactive recovery, baroreceptors are primarily involved. Although baroreceptors are implicated in each recovery mode, it is believed they are the primary influence on SKBF and sweating in the inactive mode (4, 15, 17). Thus the relative effects of active, passive, and inactive modes, and by extension the roles of central command, skeletal muscle pump/mechanoreceptors, and baroreceptors can be evaluated. The recovery mode studies (4, 16, 27, 34) similarly suggested that SKBF is at least partially modulated by baroreceptors and mechanoreceptors. Control of sweating is influenced by such other factors as central command, as well as mechanoreceptors and baroreceptors. However, there is a possible inherent bias in these studies in that they were either conducted on male subjects exclusively or did not include enough female subjects to determine possible gender differences.

There have been many reports of reduced tolerance of orthostatic challenge in women compared with men (5, 7, 8, 29, 31). Carter et al. (2) suggested that women may be more susceptible to postexercise orthostatic hypotension and that active recovery should reduce that risk. They observed a greater reduction in MAP and less compensatory vasconstriction in women than in men. In their study, however, the participants performed only 3 min of low-intensity exercise. It remains to be determined whether the same response occurs to longer, more strenuous exercise. Presently, it is unknown whether these observed cardiovascular gender differences will...
affect the relative roles of nonthermal influences on SkBF and sweating responses.

To date, little is known about the cardiovascular effects of exercise recovery in women in general, and in particular no studies have investigated the mechanisms by which nonthermal factors influence the heat-loss responses. Additionally, the hemodynamic response to passive recovery, and by extension the role of the skeletal muscle pump/mechanoreceptors, in women has not been examined. Given that previous studies have shown that upright active recovery can attenuate the fall in MAP, SkBF, and sweat rate in men (4, 16), and that Carter et al. (2) have shown that active recovery may help maintain MAP in women, we hypothesised that active recovery would be the most effective mode in attenuating the fall in MAP, SkBF, and sweat rate in women.

METHODS

Subjects. Ten healthy, physically active women volunteered and gave written consent to participate in this study. The study was approved by the Research Ethics Board at the University of Ottawa. Five to 7 days before the experiments, peak oxygen consumption (VO2 peak) was measured during a progressive cycle ergometer protocol. The VO2 peak data were used to select the submaximal workload for the experimental exercise phase of the study. Subjects were (mean ± SE) 24 ± 2 yr old and 167 ± 2 cm tall, weighed 67 ± 2 kg, and had a mean VO2 peak of 49.8 ± 2.0 ml·kg−1·min−1.

Measurements. Heart rate (HR) was monitored using a Polar coded transmitter, recorded continuously, and stored with a Polar Advantage interface and Polar Precision Performance software (Polar Electro Oy, 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 using a metabolic cart (model CPX/D, Medgraphics, St. Paul, MN) during VO2 peak assessment preceding the experimental trials. Cardiac output (CO) was estimated using the CPX/D computerized version of the carbon dioxide-rebreathing technique of Defares (6). It has been shown that Doppler-derived aortic blood flow measurements correlate well with the indirect carbon dioxide-rebreathing method (9). The Defares method has also been shown to work well in “unsteady-state” testing (11). Each measure took ~20–25 s to perform. Subjects performed one rebreathing protocol per designated time point. Stroke volume (SV) was calculated as CO/HR. Total peripheral resistance (TPR) was calculated as MAP/CO.

SkBF was estimated using laser-Doppler velocimetry (PeriFlux System 5000, main control unit; PF5010 LDPM, function unit; Perimed, Stockholm, Sweden) at the right midanterior forearm. The laser-Doppler flow probe (PR 401 angled probe, Perimed) was 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 (20). 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 site was raised to 42°C until peak CVC was measured (~30 min) (30). 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 CVC. 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 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 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 air 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 (Tes)] was monitored continuously 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 (22). Skin temperature was recorded at 11 sites (model FR-025-TTH4108-6, Concept Engineering, Old Saybrook, CT). The area-weighted mean skin temperature (Tsk) was estimated by calculating the weighted mean value, 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% (10). Temperature data were collected and digitized (data acquisition module model 3497A, Hewlett-Packard) at 5-s intervals, displayed graphically in real time, and stored on hard disk (model PC-312, 9000, Hewlett-Packard).

Experimental protocol. Each subject performed a total of three experimental trials carried out in random order. Experiments were separated by a minimum of 48 h, during which subjects were instructed to avoid physical activity and excessive stressors such as exposure to hot or cold temperatures, particularly during the period between awakening and experimentation and during transit from home to the laboratory. Trials were performed at the same time of day for each subject to avoid circadian variation in skin temperature and Tes. Subjects were in the follicular phase of the menstrual cycle. Furthermore, they were asked to fast at least 4 h before experimentation, and water ingestion was permitted ad libitum during this time. On arrival at the laboratory, subjects, who were clothed in shorts and athletic shoes, were fitted with the appropriate instruments. All experimental trials were performed at an ambient temperature of 24.0 ± 0.5°C and a relative humidity of 45%.

After preparation of the subjects, a 15-min baseline was recorded. Subjects then completed 15 min of exercise on the cycle ergometer at 70% of their VO2 peak. Immediately after the cessation of exercise, the subjects began one of three 20-min recovery conditions: 1) active recovery, consisting of loadless pedaling at 60 rpm; 2) inactive recovery, during which the subjects remained seated and motionless on the cycle ergometer; and 3) passive recovery, during which the subjects remained seated while a second person pedaled a tandem Twin-Rider bike (model 60261) such that the subject was driven through the full range of the pedaling motion at 60 rpm in a passive manner. These recovery modes are based on the work of Carter et al. (3), who selected passive pedaling as a recovery mode to examine the role of the skeletal muscle pump without the concomitant participation of central command. A study by Nobrega and Araujo (23) indicated that during passive cycling, no electromyographic activity was observed, indicating the absence of central command. A subsequent study by Nobrega et al. (24) reported some electromyographic activity during passive cycling, although it was markedly less than during active cycling. Thus one mode involves no skeletal muscle pump/mechanoreceptors and no central command (inactive), another mode involves skeletal muscle pump/mechanoreceptors and central command (active, loadless pedaling), and the other mode includes the
skeletal muscle pump/mechanoreceptors and reduced central command (passive). Therefore the passive pedaling recovery mode serves as a control for the central command effect (3).

At the end of the experiment, peak CVC was determined using a local heating protocol as described above.

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, 15, and 20 min postexercise. CO, SV, HR, and TPR were also recorded at baseline resting, end exercise, and 2, 5, 8, 12, 15, and 20 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 10 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

Exercise caused significant changes in all parameters compared with resting values (effect of time \( P < 0.05 \)); however, there were no differences in the values of each parameter at rest or at end exercise (effect of recovery mode).

Forearm CVC results are displayed graphically in Fig. 1. In the active recovery mode, CVC was greater than the inactive recovery at 5 min \( P < 0.05 \); Fig. 1). Between 8 and 20 min of recovery, CVC in the active recovery mode was greater than both the passive and inactive conditions \( P < 0.05 \); Fig. 1). There was no difference in CVC between the inactive and passive recovery modes. Sweat rate results are presented in Fig. 2. Sweat rate during the active recovery mode was higher than the passive and inactive modes between 5 and 20 min of recovery \( P < 0.05 \); Fig. 2). After 12 min of recovery, sweat rates for all conditions were significantly different from each other \( P < 0.05 \); Fig. 2).

MAP values were higher in the active mode than inactive mode throughout recovery \( P < 0.05 \), Table 1). Between 12 and 20 min of recovery, MAP in the passive mode was greater than in the inactive mode \( P < 0.05 \); Table 1). Values for MAP did not differ between the active and passive modes. TPR values in the active mode were lower than in the inactive mode throughout recovery \( P < 0.05 \); Table 1). After 8 min, TPR in the active mode was significantly lower than the passive mode also \( P < 0.05 \); Table 1). Values for TPR did not differ between the inactive and passive modes except at 2 min. CO was different between all conditions after 5 min \( P < 0.05 \); Table 1). SV in the active and passive modes was significantly different from inactive at 2 and 5 min \( P < 0.05 \); Table 1). SV was significantly different between all conditions after 8 min. \( P < 0.05 \); Table 1). HR during active recovery was higher than in the inactive mode at 15 and 20 min of recovery \( P < 0.05 \); Fig. 2).

Temperature data are presented in Table 2. No differences in \( T_{es} \) or \( T_{sk} \) were observed among the recovery modes.

DISCUSSION

The most striking conclusion from this study is that, on the basis of process of elimination using recovery mode as a variable, that central command modulates CVC in women. This is in contrast to previous studies conducted on men. The observations from the present study are consistent with our hypothesis that active recovery would be the most effective in attenuating the fall in MAP, CVC, and sweat rate. However, the nonthermal physiological control of sweating is consistent with previously reported data on male subjects (4, 16, 27, 34). Furthermore, we report female subjects’ hemodynamic responses to a passive recovery mode. We observed that passive recovery is as effective in maintaining MAP relative to the active mode. Nonetheless, the MAP values are far less than those observed previously in men (16).

Prior studies on male subjects have shown that the responses of CVC and sweating are subject to nonthermal influences postexercise (4, 16, 27, 34). Taken together, these studies suggest that attenuating the baroreceptor unloading effect associated with inactive recovery (through an active or passive recovery mode), and not central command, preserves CVC.
Postexercise measurement of sweat rate responses indicate that sweating can be modulated by central command and mechanoreceptors. One of the studies (16) showed a difference in the MAP responses between recovery modes. This finding cannot exclude the role of arterial baroreceptors in the modulation of sweat rate. The application of lower body positive pressure postexercise has been shown to increase MAP, SKBF, and sweating responses and subsequently increase the rate of core temperature decline (15). This supports a nonthermal baroreceptor influence on the postexercise thermal response in men. In the present study, the data indicate that CVC is influenced by central command and possibly cardiopulmonary baroreceptors. This is supported by the observation that active recovery attenuated the fall in CVC more so than passive and inactive, yet there was no difference in MAP responses between active and passive modes. The absence of a difference in MAP between the active and passive recovery modes suggest that different afferent information from arterial baroreceptors did not contribute to differences between modes. SV was different among all recovery modes, suggesting differences in central blood volume as confirmed by Carter et al. (3, 4) using thoracic electrical impedance techniques. Therefore, the role of cardiopulmonary baroreceptors cannot be excluded. Another noteworthy observation is that CVC in the inactive mode returned to preexercise values at ~20 min postexercise despite a persistent elevation in core temperature. This response is in agreement with our previously reported recovery mode data on men (16) and is also consistent with a “resetting” of the postexercise skin blood flow-core temperature relationship reported by Kenny et al. (12, 18, 19) and confirmed by Wilkins et al. (32) in the context of postexercise hypotension.

Nonthermal influences on sweat rate during recovery from exercise have also been documented in men. Specifically, sweat rate is modulated by both mechanoreceptors, central command (16, 27), and some evidence exists for a baroreceptor influence on postexercise sweat rate (15, 16). The present data on women demonstrate that both central command and mechanoreceptors can modulate sweat rate. Central command appears to influence sweat rate throughout recovery because sweat rate remained higher during active recovery than both passive and inactive modes. However, at 12 min postexercise, sweat rate was different among all modes, suggesting mechanoreceptors and central command play a role in the recovery period. In support of this observation, the MAP data indicate that arterial baroreceptor unloading was similar between active and passive conditions. Without the involvement of central command in the passive mode, sweat rate is reduced but remains above inactive when mechanoreceptors are activated. We cannot rule out the possibility that cardiopulmonary baroreceptors influence sweat rate because SV values and thus central blood volume were different among all recovery modes.

Our observations of hemodynamic responses supplement the work of Carter et al. (2) in that our data extend to 20 min in women. In addition, we examined the effect of a passive recovery mode on the responses. During inactive recovery,

<table>
<thead>
<tr>
<th>Variable</th>
<th>Recovery Mode</th>
<th>Baseline</th>
<th>Exercise</th>
<th>Recovery Time</th>
</tr>
</thead>
<tbody>
<tr>
<td>CO, l/min</td>
<td>Active</td>
<td>6.1±0.4</td>
<td>17.9±0.5</td>
<td>12.2±0.4†</td>
</tr>
<tr>
<td></td>
<td>Passive</td>
<td>5.8±0.5</td>
<td>17.9±0.3</td>
<td>12.1±0.6</td>
</tr>
<tr>
<td></td>
<td>Inactive</td>
<td>5.9±0.3</td>
<td>17.4±0.3</td>
<td>9.7±0.5*</td>
</tr>
<tr>
<td>SV, ml</td>
<td>Active</td>
<td>89.7±1.6</td>
<td>104.3±2.8</td>
<td>102.1±4.1</td>
</tr>
<tr>
<td></td>
<td>Passive</td>
<td>86.7±2.4</td>
<td>104.6±2.1</td>
<td>104.3±6.0</td>
</tr>
<tr>
<td></td>
<td>Inactive</td>
<td>88.6±2.4</td>
<td>101.7±2.7</td>
<td>83.8±5.5*</td>
</tr>
<tr>
<td>HR, beats/min</td>
<td>Active</td>
<td>68±2</td>
<td>172±2</td>
<td>120±2</td>
</tr>
<tr>
<td></td>
<td>Passive</td>
<td>67±2</td>
<td>171±2</td>
<td>117±3</td>
</tr>
<tr>
<td></td>
<td>Inactive</td>
<td>67±2</td>
<td>172±2</td>
<td>117±3</td>
</tr>
<tr>
<td>MAP, mmHg</td>
<td>Active</td>
<td>89.5±1.7</td>
<td>111.4±1.5</td>
<td>97.8±1.8†</td>
</tr>
<tr>
<td></td>
<td>Passive</td>
<td>91.7±1.2</td>
<td>113.1±1.3</td>
<td>94.1±2.9</td>
</tr>
<tr>
<td></td>
<td>Inactive</td>
<td>91.6±1.5</td>
<td>112.0±1.2</td>
<td>90.8±3.2</td>
</tr>
<tr>
<td>TPR, mmHg·l⁻¹·min⁻¹</td>
<td>Active</td>
<td>14.9±0.6</td>
<td>63.0±2.0</td>
<td>8.2±0.4</td>
</tr>
<tr>
<td></td>
<td>Passive</td>
<td>16.0±0.6</td>
<td>62.0±1.1</td>
<td>8.1±0.6</td>
</tr>
<tr>
<td></td>
<td>Inactive</td>
<td>15.5±0.3</td>
<td>65.5±0.1</td>
<td>9.6±0.6*</td>
</tr>
</tbody>
</table>

Values are means ± SE for 10 subjects. CO, cardiac output; SV, stroke volume; HR, heart rate; MAP, mean arterial pressure; TPR, total peripheral resistance. *Different from passive mode, P < 0.05. †Different from inactive recovery, 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>
<th>Recovery Time</th>
</tr>
</thead>
<tbody>
<tr>
<td>T_core, °C</td>
<td>Active</td>
<td>36.8±0.1</td>
<td>38.1±0.1</td>
<td>38.0±0.1</td>
</tr>
<tr>
<td></td>
<td>Passive</td>
<td>36.7±0.1</td>
<td>38.0±0.1</td>
<td>37.9±0.1</td>
</tr>
<tr>
<td></td>
<td>Inactive</td>
<td>36.8±0.1</td>
<td>38.0±0.1</td>
<td>37.8±0.1</td>
</tr>
<tr>
<td>T_ab, °C</td>
<td>Active</td>
<td>31.4±0.2</td>
<td>32.7±0.2</td>
<td>32.6±0.2</td>
</tr>
<tr>
<td></td>
<td>Passive</td>
<td>31.6±0.1</td>
<td>32.8±0.2</td>
<td>32.7±0.2</td>
</tr>
<tr>
<td></td>
<td>Inactive</td>
<td>31.5±0.1</td>
<td>32.6±0.1</td>
<td>32.5±0.1</td>
</tr>
</tbody>
</table>

Values are means ± SE for 10 subjects. T_core, esophageal temperature; T_ab, skin temperature.
female subjects showed a decrease in MAP below preexercise levels. Even with active recovery, MAP still decreased to about preexercise levels. This value was similar to MAP in men under inactive recovery conditions (16). Earlier work by Carter and colleagues (3) demonstrated that both active and passive recovery modes were effective in attenuating the fall in MAP during the first 5 min of recovery in male subjects. We have shown that passive and active recovery modes were equally effective in attenuating the fall in MAP relative to inactive recovery using a greater intensity of exercise and a longer duration recovery period (16). The present data from female subjects demonstrate that in the active mode, where both central command and the skeletal muscle pump/mechanoreceptors are activated, MAP remains at or slightly above preexercise levels. In contrast, during inactive recovery, MAP falls to levels that are hypotensive relative to preexercise values by 8 min ($P < 0.05$). Our present inactive recovery data in the first 5 min after cessation of exercise are consistent with that of Carter et al. (2).

Passive and active modes have been shown to have a greater effect in maintaining central blood volume compared with inactive recovery in men in the upright posture (3). SV values were altered by all recovery modes in the data from women in the present study, suggesting different levels of central blood volume as confirmed in other studies using thoracic electrical impedance (3, 4). This differential effect of recovery mode on central blood volume would tend to alter cardiopulmonary baroreceptor loading. The modality of CO was different between all recovery modes. Specifically, we see the difference is attributed to changes in SV because HR was not different between modes except at 15 and 20 min between active and inactive modes.

The thermal response during exercise recovery is the result of thermoregulatory and nonthermoregulatory inputs. Thus, using the present study design, one can only determine the nonthermal factors when assuming similar thermoregulatory influences between modes. The primary mechanism for stimulating heat loss responses of CVC and sweating is that of the hypothalamic feedback circuit, which receives an integrated signal from core and skin temperature (21). $T_s$ data, which are representative of arterial blood temperature at the level of the heart (28), indicate no differences between recovery modes. Moreover, the contribution of skin temperature to CVC and sweating responses appeared similar between recovery modes. Given no differences in temperature, we conclude that the observed effects on CVC and sweating are due to nonthermally influences modulated by recovery mode.

Oxygen consumption between recovery modes was not compared in this study. Mild elevations in oxygen consumption have been observed during active (1) and passive (27) recovery modes; however, as Wilson et al. (34) have suggested, even if heat production had been higher in the active modes, it was not enough to produce changes in hypothalamic or mean skin temperature sufficient to modify CVC or sweating. This is important because core and skin temperature are known to be the primary influences on CVC and sweating responses. Furthermore, if heat production had been significantly greater during active recovery, we probably would have observed an increase in CVC and sweating given the 20-min duration of our recovery.

Active recovery is the most effective in attenuating the fall in CVC and sweat rate. By extension and contrary to previous conclusions with men, central command can modulate CVC in women. Furthermore, the role of cardiopulmonary baroreceptors cannot be ruled out as a possible influence. Control of sweat rate appears to be similar to men in that both central command and mechanoreceptors can influence the response. As with CVC, the role of the cardiopulmonary baroreceptors also cannot be excluded. Finally, although passive recovery may help maintain MAP in women, it does not attenuate the fall in CVC as observed in men.

GRANTS

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REFERENCES


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