

Active recovery attenuates the fall in sweat rate but not cutaneous vascular conductance after supine exercise

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Wilson, Thad E., Robert Carter III, Michael J. Cutler, Jian Cui, Michael L. Smith, and Craig G. Crandall. Active recovery attenuates the fall in sweat rate but not cutaneous vascular conductance after supine exercise. *J Appl Physiol* 96: 668–673, 2004. First published September 23, 2003; 10.1152/jappphysiol.00522.2003.—The purpose of this study was to identify whether baroreceptor unloading was responsible for less efficient heat loss responses (i.e., skin blood flow and sweat rate) previously reported during inactive compared with active recovery after upright cycle exercise (Carter R III, Wilson TE, Watenpaugh DE, Smith ML, and Crandall CG. *J Appl Physiol* 93: 1918–1929, 2002). Eight healthy adults performed two 15-min bouts of supine cycle exercise followed by inactive or active (no-load pedaling) supine recovery. Core temperature (T_{core}), mean skin temperature (T_{sk}), heart rate, mean arterial blood pressure (MAP), thoracic impedance, central venous pressure ($n = 4$), cutaneous vascular conductance (CVC; laser-Doppler flux/MAP expressed as percentage of maximal vasodilation), and sweat rate were measured throughout exercise and during 5 min of recovery. Exercise bouts were similar in power output, heart rate, T_{core} , and T_{sk} . Baroreceptor loading and thermal status were similar during trials because MAP (90 ± 4 , 88 ± 4 mmHg), thoracic impedance (29 ± 1 , $28 \pm 2 \Omega$), central venous pressure (5 ± 1 , 4 ± 1 mmHg), T_{core} (37.5 ± 0.1 , $37.5 \pm 0.1^\circ\text{C}$), and T_{sk} (34.1 ± 0.3 , $34.2 \pm 0.2^\circ\text{C}$) were not significantly different at 3 min of recovery between active and inactive recoveries, respectively; all $P > 0.05$. At 3 min of recovery, chest CVC was not significantly different between active ($25 \pm 6\%$ of maximum) and inactive ($28 \pm 6\%$ of maximum; $P > 0.05$) recovery. In contrast, at this time point, chest sweat rate was higher during active ($0.45 \pm 0.16 \text{ mg}\cdot\text{cm}^{-2}\cdot\text{min}^{-1}$) compared with inactive ($0.34 \pm 0.19 \text{ mg}\cdot\text{cm}^{-2}\cdot\text{min}^{-1}$; $P < 0.05$) recovery. After exercise CVC and sweat rate are differentially controlled, with CVC being primarily influenced by baroreceptor loading status while sweat rate is influenced by other factors.

skin blood flow; baroreceptors; central command

DISSIPATING HEAT DURING AND AFTER physical activity is imperative to prevent heat-related injuries. The primary method of human heat dissipation is accomplished by increasing skin blood flow and evaporative cooling. Most athletic events and occupational tasks are composed of intermittent activity and recovery periods. Thus to understand thermoregulation under these conditions it is essential to understand thermoregulatory effector responses, especially in terms of skin blood flow and sweating, during activity and subsequent recovery from activity.

Despite well-characterized responses to exercise, less is understood about thermoregulation and thermoregulatory ef-

factor responses during recovery from physical activity. Noteworthy work has been completed by Kenny and colleagues (20–22) addressing postexercise thermoregulation; however, those studies did not address the effects of different modes of recovery on thermoregulation. Previously Carter et al. (4), identified improved thermoregulatory effector responses during active recovery (i.e., loadless pedaling at the same cadence as performed during exercise) compared with inactive recovery (i.e., no pedaling) after dynamic cycling in the upright posture. Despite no significant differences in power output and temperatures (ambient, internal, and mean skin) between exercise bouts and conditions, active recovery attenuated the fall in skin blood flow and sweat rate compared with inactive recovery. There were no significant differences in mean arterial pressure between recovery modes; however, central blood volume (indexed via thoracic impedance) was significantly lower in the inactive recovery mode (4). Hence, sustained cutaneous vasodilation and sweating during active recovery after exercise were concluded to not be thermally mediated but possibly occurred via baroreceptors or other nonthermal factors associated with leg movements such as muscle mechanoreceptor stimulation and/or central command.

Thermal stimuli are the primary mechanism leading to elevated effector responses in humans during exercise (2, 12, 32, 33). In addition, nonthermal factors can modulate skin blood flow and sweat rate responses (9, 16, 19, 35). Examples of these nonthermal factors include exercise intensity, hydration status, age, baroreceptor loading status, muscle metabolic and/or mechanoreceptor stimulation, and central command. Given the ability of baroreceptors to modify skin blood flow and possibly sweat rate, we tested the hypothesis that the larger decrease in skin blood flow and sweat rate previously observed at the end of exercise during upright passive recovery, compared with these responses at the end of exercise during upright active recovery (4), was due primarily to baroreceptor unloading. This hypothesis was tested by assessing postexercise cutaneous vascular and sweating responses during active and inactive recovery while in the supine position. The supine recovery position was chosen to minimize the magnitude of baroreceptor unloading compared with recovery in the upright position. By minimizing the role of baroreceptors and then comparing these data with previous works (4), the role of baroreceptors in heat loss responses during exercise recovery could be identified.

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Table 1. Effect of mode of exercise recovery on cardiovascular responses

Variable	Recovery Mode	Preexercise	Exercise	Recovery Minute 1	Recovery Minute 2	Recovery Minute 3	Recovery Minute 4	Recovery Minute 5
HR, beats/min	Active	62±8	130±4	114±6*	102±6*	97±6*	94±7*	93±7*
	Inactive	56±7	127±6	102±8	86±8	82±9	78±9	76±8
MAP, mmHg	Active	95±4	102±5	92±4	89±4	90±4	90±4	91±4
	Inactive	90±4	99±3	87±4	86±4	88±4	87±4	88±3
SBP, mmHg	Active	136±5	166±6	149±6	135±6	138±7	138±6	138±6
	Inactive	130±6	164±7	143±8	132±7	133±7	132±7	132±7
DBP, mmHg	Active	74±4	70±4	64±3	66±3	66±3	66±3	67±3
	Inactive	71±4	67±3	59±4	64±3	66±3	65±3	65±3
CVP, mmHg	Active	9.1±1.9	7.8±1.2	6.5±0.7*	5.4±0.6	5.3±0.6	5.2±0.6	5.2±0.7
	Inactive	9.5±1.5	7.9±0.6	5.4±0.4	4.6±0.2	4.4±0.3	4.5±0.4	4.7±0.5
Z ₀ , Ω	Active	29.3±1.6	30.0±1.4	29.6±1.3	29.5±1.3	29.4±1.3	29.3±1.4	29.2±1.4
	Inactive	28.2±1.8	28.9±1.8	28.8±1.8	28.5±1.8	28.3±1.8	28.2±1.8	28.2±1.8

Values are means ± SE. HR, heart rate; MAP, mean arterial blood pressure; SBP, systolic blood pressure; DBP, diastolic blood pressure; CVP, central venous pressure; Z₀, thoracic impedance. Exercise, last minute of the 15-min exercise bout. *Significant difference between mode of recovery (i.e., active vs. inactive) at the $P < 0.05$ level.

METHODS

Subjects. Eight individuals (6 men and 2 women) with mean age of 31 ± 3 (range 22–47) yr, height of 175 ± 3 (range 157–185) cm, and weight of 71 ± 2 (range 65–82) kg underwent the study protocols. All subjects were healthy nonsmokers and free of any known cardiovascular and metabolic diseases. Subjects refrained from exercise, alcohol, and stimulants such as caffeine for 24 h before testing. Studies were conducted in the morning over the summer months in a historically warm climate (Dallas, TX). This study received Institutional Review Board approval from the University of Texas Southwestern Medical Center at Dallas and the Presbyterian Hospital of Dallas, and each subject provided written, informed consent to participate in this study.

Measurements. Internal temperature was indexed via esophageal temperature ($n = 6$) by means of a thermistor (YSI, Yellow Springs, OH) placed at the level of the atria, determined by 0.25-standing height (33). Two subjects were unable to correctly place or maintain the esophageal temperature probe. In all subjects, internal temperature was also obtained via an ingestible pill telemetry system (HTI Technologies, Palmetto, FL). Mean skin temperature was derived from the weighted electrical average of six thermocouples placed on the skin (37). Sweat rate was measured by use of capacitance hygrometry (Viasala, Woburn, MA) by perfusing 100% nitrogen at a flow rate of 500 ml/min through a ventilated capsule (surface area = 2.83 cm²) attached to dorsal forearm and chest skin. Chest and forearm skin blood flows were monitored from integrative laser-Doppler flowmetry probes (Perimed, North Rayalton, OH). After the exercise protocol and recovery periods, a 3-cm-diameter heating element, which housed the laser-Doppler flow probe, was activated to elevate local skin temperature to 42°C. Local temperature was held at this level for 30 min to elicit maximal cutaneous vasodilation (38). An index of cutaneous vascular conductance (CVC) was calculated from the ratio of laser-Doppler flux to mean arterial blood pressure. CVC was then expressed as a percent of maximal cutaneous vasodilation as determined from local heating. Heart rate was obtained from the electrocardiogram (SpaceLabs, Redmond, WA) interfaced with a cardiatachometer (CWE, Ardmore, PA). Measurements of systolic arterial pressure and diastolic arterial pressure were performed on a beat-by-beat basis noninvasively by using a photoplethysmographic cuff attached to the finger (Ohmeda, Louisville, CO). This measurement was periodically verified by electrophygmomanometry of the upper arm (Suntech, Raleigh, NC). Impedance cardiography (Biopac Systems, Santa Barbara, CA) was used to measure transthoracic impedance, which was used as an index of central blood volume (14, 15). Central venous pressure ($n = 4$) was measured from a catheter inserted in the subject's basilic vein and advanced to the superior vena cava.

Experimental protocol. After subject instrumentation, 5 min of baseline data were collected with the subject in the supine position on a supine cycle ergometer (Atomic Products, Shirley, NY). Each subject exercised for 15 min at a workload that elicited ~65% of the individual's predicted maximal heart rate at a pedaling cadence of 65 rpm. After the exercise bout, and with the subject remaining in the supine position, the subject stopped exercising (inactive recovery) or performed no-load pedaling at a cadence of 65 rpm (active recovery). Thus the following exercise bouts were performed in random order, with a 2-h rest period between exercise trials: 1) 15 min of exercise followed by 5 min of supine inactive recovery, and 2) 15 min of exercise followed by 5 min of supine active recovery. During the 2-h period between trials no probes were removed, and thus skin blood flow and sweat rate responses were measured from the same locations between trials. Subjects were permitted to drink 12 oz of water or noncaffeinated carbohydrate-free drink during the 2-h period. We hypothesized that inactive recovery would not engage the skeletal muscle pump or central command, whereas some degree of skeletal muscle pumping and central command would be engaged during no-load pedaling. Because both exercise and recovery from exercise were done in the supine position, baroreceptor unloading would be minimized between recovery conditions compared with exercise recovery in the upright position (4). Environmental temperature of the laboratory was controlled for all experimental bouts at $25 \pm 1^\circ\text{C}$.

Data analyses. Data were continuously acquired (Biopac) and averaged into the following 1-min stages for both protocols: preexercise, last minute of exercise, and recovery minutes 1–5. Telemetry pill temperature data were obtained at 10-s intervals and were averaged to the aforementioned stages. Movement associated with exercise can elicit motion artifact in the measurement of skin blood flow as indexed by laser-Doppler flowmetry. To account for this artifact, the initial increase (within the first minute of exercise) in the skin blood flow signal was offset from (i.e., subtracted from) the cycle ergometry data. Data were analyzed via a two-way repeated-measures ANOVA with main factors of recovery mode and stage of exercise or recovery. If significant differences were observed, Student-Newman-Keuls post hoc analysis was employed to identify time-point differences. Statistical significance was set at an α level of 0.05. All data are presented as means ± SE.

RESULTS

Power outputs were equivalent between the two exercise bouts (104 ± 14 W). Additionally, end-exercise heart rates were not different between exercise bouts (see Table 1). Heart rate was significantly greater throughout active recovery relative to inactive recovery. Baseline, exercise, and recovery

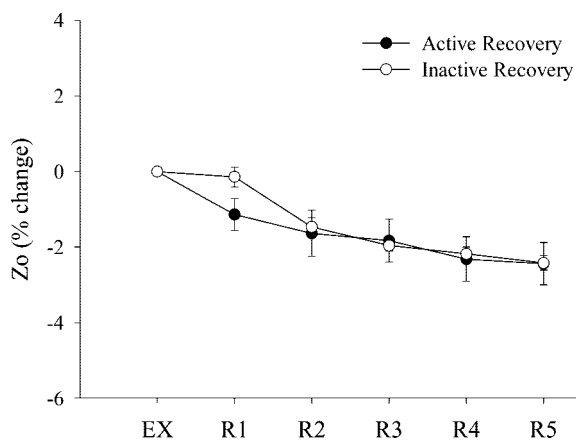


Fig. 1. Effect of mode of recovery after 15 min of cycle ergometry exercise (EX) on thoracic impedance (Z_0). Z_0 is expressed as a percent change from exercise value. No differences were identified between recovery modes. Data are expressed as means \pm SE. R1–R5, minutes 1 through 5 of recovery.

arterial pressures, mean central venous pressure, and thoracic impedance values are shown in Table 1. Mean arterial blood pressure and systolic and diastolic blood pressures decreased similarly during both recovery modes (all $P > 0.05$). Mean central venous pressure, however, was significantly ($P = 0.03$) lower during the first minute of exercise recovery for the inactive recovery mode but then was not significantly different between recovery modes for the remainder of the recovery period ($P > 0.05$). Whether expressed in absolute (see Table 1) or relative terms (see Fig. 1), there was no significant difference between recovery modes in thoracic impedance at the end of exercise or throughout the recovery periods.

Esophageal and telemetry pill temperatures increased significantly during exercise and remained elevated during both recovery conditions. The agreement of these two internal temperature measures was high throughout baseline, exercise, and recovery periods (average $r = 0.94$), although mean internal temperature was higher (0.44°C) with the telemetry pill. Similar to others (29), these data highlight the ability of the telemetry pill device to follow changes in internal temperature during exercise and recovery from exercise. Regardless of the mode of temperature monitoring, no differences in internal temperature were identified between active and inactive recoveries (see Table 2). Relative to preexercise values, mean skin temperature was also significantly elevated during 15 min of exercise and remained higher throughout both modes of recovery. No significant differences existed in mean skin temperature between the two recovery modes (see Table 2).

Sweat rate was not different before exercise between bouts, suggesting that during the 2-h period between exercise bouts sweat rate returned to baseline. At the last minute of exercise, forearm sweat rate was significantly ($P < 0.001$) elevated relative to baseline, although there were no differences between bouts ($0.49 \pm 0.19 \text{ mg}\cdot\text{cm}^{-2}\cdot\text{min}^{-1}$ before active recovery and $0.51 \pm 0.16 \text{ mg}\cdot\text{cm}^{-2}\cdot\text{min}^{-1}$ before inactive recovery). Similarly, exercise significantly increased chest sweat rate to 0.52 ± 0.23 and $0.57 \pm 0.17 \text{ mg}\cdot\text{cm}^{-2}\cdot\text{min}^{-1}$ ($P < 0.001$) before active and inactive recoveries, respectively, and these values were also not different between bouts. Throughout active recovery, chest and forearm sweat rate were significantly elevated relative to sweat rate during inactive recovery,

with the exception of forearm sweat rate at minute 5 of recovery (see Fig. 2).

Preexercise chest CVC was 14.2 ± 2.1 and $15.1 \pm 1.9\%$ of maximal CVC for the active and inactive treatments, respectively. Preexercise forearm CVC was 14.6 ± 5.1 and $10.0 \pm 2.5\%$ of maximal CVC for the active and inactive treatments, respectively. For both locations, there were no differences between these preexercise CVC values ($P > 0.05$). Just before the end of exercise, chest CVC (active recovery: $42.7 \pm 8.3\%$ of maximal CVC; inactive recovery: $40.1 \pm 7.1\%$ of maximal CVC) and forearm CVC (active recovery: $30.2 \pm 7.6\%$ of maximal CVC; inactive recovery: $32.8 \pm 9.4\%$ of maximal CVC) were significantly elevated ($P < 0.001$) relative to preexercise values. No differences in chest or forearm CVC were observed at the end of exercise between exercise bouts. In contrast to sweat rate responses, there were no differences in chest or forearm CVC between modes of recovery throughout the recovery periods (see Fig. 3).

DISCUSSION

Previously, our laboratory identified that CVC and sweat rate were significantly elevated during active recovery (i.e., loadless cycling) in the upright position compared with inactive recovery also in the upright position (4). In that study, arterial pressure was similar between recovery conditions; however, there were differences in an index of central blood volume (i.e., thoracic impedance) between recovery modes. Elevated thoracic impedance during inactive recovery suggests a greater degree of baroreceptor unloading, relative to the active recovery condition, despite similar arterial blood pressures. The present study was designed to minimize differences in central blood volume between recovery modes by having the subject recover from dynamic exercise in the supine position. The major finding of this study is that sweat rate remains significantly elevated during active compared with inactive recovery after dynamic supine exercise, whereas CVC responses are similar between recovery modes. These data indicate that when similar pressure stimuli are presented to the cardiovascular system, factors associated with unloaded pedaling modulate sweat rate but not skin blood flow during recovery from dynamic exercise. This increased sweating would potentially allow for greater heat loss during active recovery compared with inactive recovery.

Internal temperature (both esophageal and telemetry pill) was not different between active and inactive recovery. Other investigators (20, 21) have identified larger rates of change in postexercise esophageal temperature than the present study; however, in the cited studies exercise esophageal temperatures were also greater. This issue, along with different modes of exercise, intensity of exercise, and ambient conditions, might explain the different rate of postexercise temperature change between studies. In the present study, mean skin temperature was also not affected by recovery mode. Together, these data indicate that whole-body thermal factors were similar between recovery conditions.

Baroreceptors modify skin blood flow during normothermia (17, 39), passive heat stress (5, 18, 36), and exercise (13, 25, 26). In the present study, the two recovery conditions resulted in similar arterial blood pressures, central venous pressures (with the exclusion of the first minute of recovery), and

Table 2. Effect of mode of exercise recovery on temperature responses

Variable	Recovery Mode	Preexercise	Exercise	Recovery Minute 1	Recovery Minute 2	Recovery Minute 3	Recovery Minute 4	Recovery Minute 5
T_{es} , °C	Active	36.5±0.1	37.0±0.1	37.1±0.1	37.1±0.1	37.0±0.1	37.0±0.1	37.0±0.1
	Inactive	36.5±0.1	37.1±0.1	37.1±0.1	37.1±0.1	37.1±0.1	37.0±0.1	36.9±0.1
T_{pill} , °C	Active	36.9±0.1	37.5±0.1	37.5±0.1	37.5±0.1	37.5±0.1	37.5±0.2	37.5±0.2
	Inactive	37.0±0.1	37.4±0.2	37.4±0.1	37.4±0.1	37.5±0.1	37.4±0.1	37.4±0.1
T_{sk} , °C	Active	33.4±0.3	34.1±0.4	34.1±0.4	34.1±0.4	34.1±0.3	34.0±0.4	33.9±0.4
	Inactive	33.7±0.2	33.8±0.4	34.1±0.3	34.2±0.2	34.2±0.2	34.2±0.2	34.2±0.2

Values are means ± SE. T_{es} , esophageal temperature; T_{pill} , telemetry pill temperature; T_{sk} , mean skin temperature. Exercise, last minute of the 15-min exercise bout. No significant differences were observed between modes of recovery during preexercise, exercise, or recovery periods for any variable.

thoracic impedances (an index of central blood volume). Therefore, the supine exercise paradigm minimized the magnitude of baroreceptor unloading between recovery conditions. Supine exercise increases venous return and central venous pressure compared with upright exercise (32). Increasing venous return by means of fluid loading or supine exercise increases skin blood flow (6, 13, 28). Moreover, our laboratory

previously reported that, in the upright position, skin blood flow decreased to a greater extent during inactive recovery compared with active recovery (4). Data from the present study, combined with our laboratory's prior findings (4), confirm the hypothesis that larger reductions in CVC previously observed during upright inactive recovery were due to greater baroreceptor unloading compared with upright active recovery.

The role of baroreceptor unloading in modulating sweat rate is less clear relative to findings regarding baroreceptor control

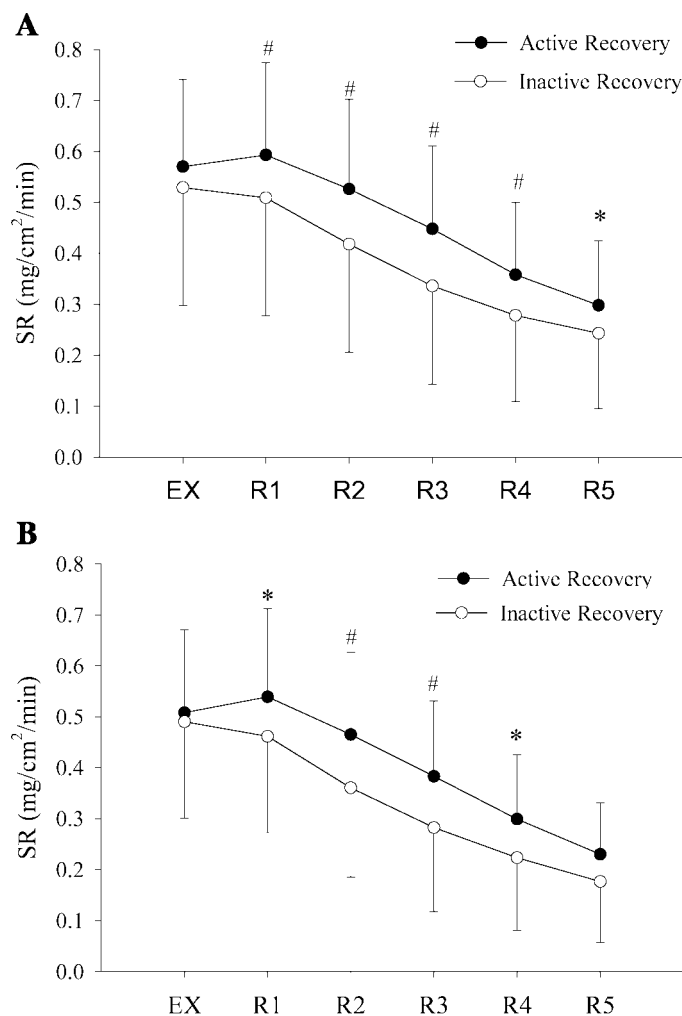


Fig. 2. Effect of mode of exercise recovery after 15 min of cycle ergometry exercise on sweat rate (SR) from chest (A) and forearm (B) skin. No differences were identified at the end of exercise; however, SR during active recovery was significantly greater relative to during inactive recovery. Data are expressed as means ± SE. # $P < 0.01$ and * $P < 0.05$ relative to inactive recovery.

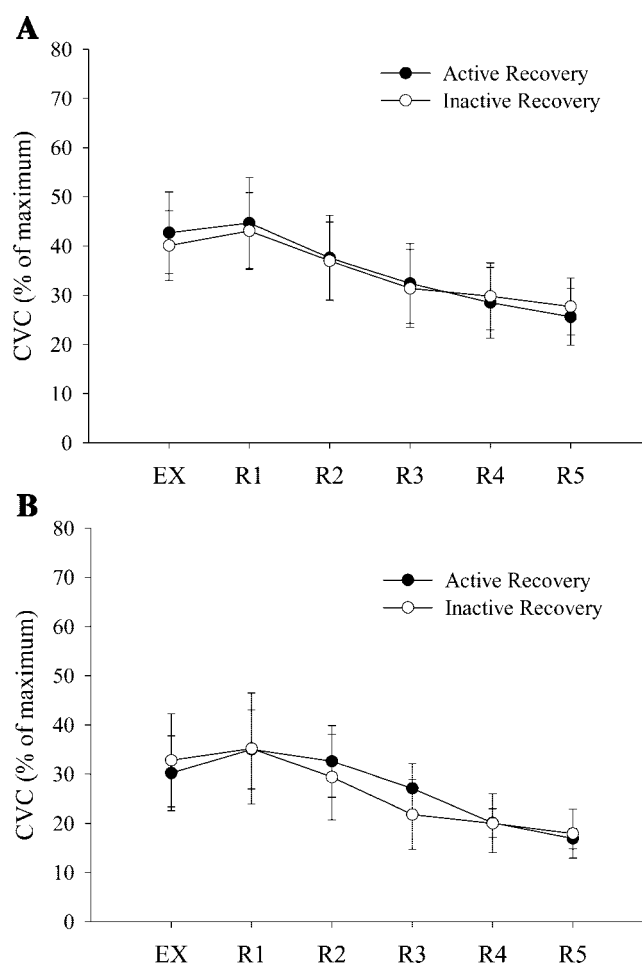


Fig. 3. Effect of mode of recovery after 15 min of cycle ergometry exercise on cutaneous vascular conductance (CVC) from chest (A) and forearm (B) skin. CVC is expressed as a percentage of maximal flow induced via local heating. No differences in CVC were identified at the end of exercise or between recovery modes. Data are expressed as means ± SE.

of skin blood flow. For example, some studies report reductions in sweating during baroreceptor unloading (8, 25, 36), whereas other studies find unaltered sweat rates during baroreceptor perturbations (42–44). Data from the present experiment clearly show blunted decreases in sweating during active recovery compared with inactive recovery (see Fig. 2). This result occurred despite minimal differences in arterial (indexed from systolic, diastolic, and mean arterial blood pressure) and cardiopulmonary (indexed from central venous pressure and thoracic impedance) baroreceptor loading status between recovery modes. Consistent with this observation, a previous investigation reported minimal differences in central venous pressure between the last minute of one-legged exercise (30 W) and inactive recovery (31). Therefore, these results support the notion that baroreceptors were not the mechanism responsible for the separation in sweat rate observed between recovery modes in the present and the prior study (4).

Possible mechanisms resulting in elevated sweat rate during active recovery include nonthermal factors (e.g., central command and muscle metabo/mechanoreceptor stimulation) previously shown or hypothesized to modulate sweat rate during exercise (7, 23, 34, 40, 45). Because the subjects moved their legs during active recovery, some degree of central command stimulation must have occurred, albeit to a lesser degree than the exercise bout. Heart rate during recovery from dynamic exercise has been observed to be higher during active recovery compared with either inactive or passive recovery (3). Differences in heart rate between recovery modes, coupled with a lack of difference in blood pressure, provide evidence for augmented central command during active recovery (see Table 1). Central command is capable of augmenting sweat rate and skin sympathetic nerve activity independent of muscle metabo/mechanoreceptor stimulation (34, 41). Thus it is possible that differences in sweating responses between modes of recovery may be due to factors associated with increased central command during active recovery.

Increases in sweating during exercise are due in part to stimulation of metaboreceptors within the exercising muscle (7, 23, 34). In the present experiment we did not measure any indexes of the release of metabolic byproducts during exercise or recovery. However, we do not expect that there would be an appreciable difference between the level of metaboreceptor stimulation between inactive and active recoveries because metaboreceptor stimulation increases blood pressure (1, 24), and blood pressure responses between recovery modes were not different (see Table 1). However, the present data do not exclude a possible contribution from mechanoreceptor stimulation in mediating some of the sweat rate response.

Limitations. In the present study we measured central venous pressure in only four subjects. Our rationale was that this invasive measurement was redundant with thoracic impedance, coupled with post hoc power analysis that revealed a total of 32 subjects would be needed to raise the power of the statistical test to an appropriate level to confirm that central venous pressure was not different between recovery modes. With this information, combined with our effort to minimize the risk-to-benefit ratio for the human participants in this study, we stopped central venous pressure measures after the fourth subject.

Even though there were no internal or mean skin temperature differences between recovery modes, these data do not

discount that muscle metabolism, and thus muscle heat production, was slightly higher during loadless active recovery relative to inactive recovery. Although intramuscular temperatures were not measured, on the basis of others' work (22) it is possible that intramuscular temperatures were higher during active recovery, the extent of which is unknown. Nevertheless, it is debatable whether muscles have thermoreceptors or what participation they have, if any, in an integrated thermoregulatory response (30). In the present study we did not measure oxygen uptake during exercise or exercise recovery; however, pilot work (unpublished observations) indicated minimal differences in oxygen uptake (0.115 ± 0.019 l/min) and metabolic heat production [20.6 W/m ± 0.036 ; calculated according to Gagge and Gonzalez (11)] between recovery modes. This increase in oxygen uptake is consistent with previous observations that have identified increases in oxygen uptake with increases in contraction frequency (10, 27). We contend that although there was slightly more heat produced in active compared with inactive recovery modes, it is doubtful that this heat production was sufficient to stimulate thermoreceptors (i.e., hypothalamus, skin, or possibly muscle) requisite to modify an efferent response (i.e., CVC and sweat rate). Moreover, if differences in sweat rate between recovery modes were strictly a result of thermally mediated responses, then similar differences in CVC should have also been observed, because internal and skin temperatures are the primary controllers of both CVC and sweat rate (2, 12, 32, 33).

In conclusion, sweat rate remained elevated during active recovery from dynamic supine exercise, whereas CVC responses were not different between recovery modes. There were no differences in either internal or mean skin temperatures between exercise bouts or between recovery modes. Furthermore, the supine exercise paradigm minimized both arterial and central venous pressure differences between recovery conditions. Taken together, elevated sweat rate during active recovery from dynamic exercise is likely due to factors unrelated to thermal or baroreceptor loading status. Possible mechanisms responsible for this response include central command and/or muscle mechanoreceptor stimulation.

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