

Effects of mode of exercise recovery on thermoregulatory and cardiovascular responses

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Carter, Robert III, Thad E. Wilson, Donald E. Watenpaugh, Michael L. Smith, and Craig G. Crandall. Effects of mode of exercise recovery on thermoregulatory and cardiovascular responses. *J Appl Physiol* 93: 1918–1924, 2002. First published August 16, 2002; 10.1152/jappphysiol.00056.2002.—To identify the effects of exercise recovery mode on cutaneous vascular conductance (CVC) and sweat rate, eight healthy adults performed two 15-min bouts of upright cycle ergometry at 60% of maximal heart rate followed by either inactive or active (loadless pedaling) recovery. An index of CVC was calculated from the ratio of laser-Doppler flux to mean arterial pressure. CVC was then expressed as a percentage of maximum (%max) as determined from local heating. At 3 min postexercise, CVC was greater during active recovery (chest: 40 ± 3 , forearm: $48 \pm 3\%$ max) compared with during inactive recovery (chest: 21 ± 2 , forearm: $25 \pm 4\%$ max); all $P < 0.05$. Moreover, at the same time point sweat rate was greater during active recovery (chest: 0.47 ± 0.10 , forearm: $0.46 \pm 0.10 \text{ mg} \cdot \text{cm}^{-2} \cdot \text{min}^{-1}$) compared with during inactive recovery (chest: 0.28 ± 0.10 , forearm: $0.14 \pm 0.20 \text{ mg} \cdot \text{cm}^{-2} \cdot \text{min}^{-1}$); all $P < 0.05$. Mean arterial blood pressure, esophageal temperature, and skin temperature were not different between recovery modes. These data suggest that skin blood flow and sweat rate during recovery from exercise may be modulated by nonthermoregulatory mechanisms and that sustained elevations in skin blood flow and sweat rate during mild active recovery may be important for postexercise heat dissipation.

skin blood flow; sweat rate; blood pressure; central command

DURING DYNAMIC EXERCISE, INCREASES in metabolism lead to an elevation in internal temperature and subsequent pronounced increases in skin blood flow and sweating. These latter responses are necessary for humans to appropriately thermoregulate, especially in warm environmental conditions. As the individual continues to exercise, the initial rapid elevation in skin blood flow and sweating gives way to more moderate increases in these variables. Nevertheless, depending on a number of factors, including environmental conditions, workload, and hydration status, sweat rate can

increase upward to 3.7 l/h (2), while skin blood flow also has the capacity to increase substantially (16, 27). Despite these well-characterized responses to dynamic exercise, relatively little is known about skin blood flow and sweating responses during the recovery from exercise.

In addition to the aforementioned thermoregulatory responses during exercise, skin blood flow and sweating may also be modulated by nonthermoregulatory responses associated with exercise such as baroreceptor loading status, central command, and muscle metaboreceptor and mechanoreceptor stimulation (14, 28). On cessation of dynamic exercise, and subsequent removal of central command and metaboreceptor and mechanoreceptor stimulation, cutaneous vasodilation and sweating responses may still be modulated by baroreceptors. Moreover, our group previously reported that the muscle pump was beneficial in preserving stroke volume and blood pressure during exercise recovery independent of central command (5). This latter point, together with evidence suggesting that skin blood flow and sweating responses may be modulated by baroreceptor unloading (7, 15, 18, 22), suggests that cutaneous vascular and sweating responses during exercise recovery may be preserved after exercise if baroreceptor unloading is attenuated via engagement of the skeletal muscle pump. Preserved cutaneous vasodilation and sweating would be beneficial during the recovery period in facilitating the removal of the added heat strain associated with dynamic exercise.

Given the lack of information regarding the effects of recovery from dynamic exercise on cutaneous vascular and sweating responses, the purpose of this project was to identify the influence of exercise recovery mode by testing the hypothesis that active recovery (unloaded cycling) would preserve the elevation in skin blood flow and sweating after dynamic exercise. This hypothesis was tested by assessing postexercise cutaneous vascular and sweating responses during active and inactive recovery.

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METHODS

Subjects. Eight men between 25 and 37 yr of age were studied. Only men were studied in this experimental paradigm because of potential differences in postexercise hypotension and exercise recovery responses between genders (4). Their demographic composition was as follows: one African-American, one Hispanic-American, two Asians, and four Anglo-Americans. Subjects were moderately fit, with maximal oxygen consumption values ranging from 40 to 60 ml·kg⁻¹·min⁻¹, and of average height (173 ± 2 cm) and weight (75 ± 2 kg). All subjects were free of any known cardiovascular diseases. Subjects were asked to refrain from exercise and stimulants such as caffeine for 24 h before testing. All experimental procedures and protocols were approved by the University of North Texas Health Science Center and the University Of Texas Southwestern Medical Center Institutional Review Boards. Each subject provided written, informed consent to participate in the study.

Experimental design. Subjects were instrumented for the measurement of hemodynamic and thermoregulatory variables as described below. After instrumentation, 5 min of baseline data were collected with the subject seated upright on a magnetically braked cycle ergometer (Lode, Groningen, North Los Angeles, CA). Each subject then performed an exercise bout began with a 1-min warm-up period in which the subject pedaled with no resistance (unloaded cycling). After this period, exercise intensity increased within 10 s to a workload that elicited ~60% of the individual's predicted maximal heart rate (HR) while pedaling at a rate of 65 rpm. Subjects exercised at this workload for 15 min. After the exercise bout, each subject either rested on the cycle ergometer (inactive recovery) or performed loadless pedaling (active recovery) for 5 min also at a rate of 65 rpm. Thus the following exercise bouts were performed in random order and on separate days: 1) 15 min of exercise followed by active recovery with unloaded cycling at a pedaling rate of 65 rpm, and 2) 15 min of exercise followed by inactive recovery. We hypothesized that inactive recovery would not engage the skeletal muscle pump and possibly central command, whereas the skeletal muscle pump and central command would be engaged during loadless pedaling. Any effects of circadian variation were accounted for by conducting each session at the same time of day for a given subject. Environmental conditions of the laboratory were controlled between experimental days for each subject. Ambient temperature during the studies was 24.7 ± 0.3°C.

Thermal measurements. Each subject was instrumented for the measurement of esophageal temperature with a thermistor (YSI, Yellow Springs, OH) placed at 25% of the subject's standing height (24) and the measurement of mean skin temperature from the electrical average of six thermocouples placed on the skin (32). Sweat rate was measured by using capacitance hygrometry (Viasala, Woburn, MA) by perfusing 100% nitrogen at a flow rate of 500 ml/min through a ventilated capsule attached to forearm and chest skin.

Hemodynamic measurements. Tetrapolar impedance cardiography (model 304A, Surcom, Minneapolis, MN) was used to measure changes in thoracic impedance. This measurement involved placing four Mylar band electrodes circumferentially around the neck and thorax. Decreases in thoracic blood volume are associated with increases in basal thoracic impedance (13). Therefore, we used changes in thoracic impedance as an indirect measure of changes in central blood volume. Stroke volume was estimated via thoracic impedance (25) with COP 4.0 cardiac output software (MicroTronics, Chapel Hill, NC). Resistivity of blood was assumed con-

stant between rest and during exercise, considering the short duration of exercise and the moderate intensity. The COP software contains automatic artifact-rejection and error-detection algorithms. At the end of each ensemble-averaging period, the accumulated data and ensemble-averaged impedance signals were automatically analyzed, and cardiac indexes were computed using the widely recognized Kubicek equation (19–21). This cardiac output program has been shown to accurately measure stroke volume at rest (13, 34), during lower body negative pressure (10, 13) and during exercise (25, 26, 34). Prior studies suggest changes in skin temperature and hydration status do not have a significant effect on bioimpedance measurements using this method (6). HR was obtained from the electrocardiogram (SpaceLabs, Redmond, WA) interfaced with a cardiometer (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 a finger (Finapres blood pressure monitor, Ohmeda). These measurements were periodically verified by auscultation of the brachial artery (Critikon, Tampa, FL). Mean arterial blood pressure (MAP) was obtained from the integrated arterial blood pressure waveform. Cardiac output was calculated as stroke volume × HR, and total peripheral resistance was calculated as MAP divided by cardiac output. Chest and forearm skin blood flows were monitored from integrative laser-Doppler flowmetry probes (Perimed, North Raylton, OH). After the exercise protocol and recovery periods, a 3-cm-diameter heater element, which housed the laser-Doppler flow probe, was used to elevate local skin temperature to 42°C. Local temperature was held at this level for 30 min to elicit maximal cutaneous vasodilation (32). An index of cutaneous vascular conductance (CVC) was calculated from the ratio of laser-Doppler flux to MAP. CVC was then expressed as a percentage of maximal cutaneous vasodilation as determined from local heating.

Data analyses. Data were continuously acquired throughout each protocol (Biopac, Santa Barbara, CA). The following data were analyzed: the minute immediately before exercise, the last minute of exercise, and 1-min averages during the 5-min recovery period. Comparison of responses during both exercise recovery modes was performed by using a two-way ANOVA. The two main factors of the ANOVA were exercise recovery mode and time. When significant main effects were observed, post hoc analyses were performed by using Tukey's multiple-comparison tests. Statistical significance was set at an α level of 0.05. All data are presented as means ± SE.

RESULTS

Esophageal temperature increased significantly during exercise and remained elevated during both recovery conditions. No differences of this variable were observed between recovery conditions (Table 1). Relative to baseline values, skin temperature was also significantly elevated during 15 min of exercise and throughout both modes of recovery (Table 1). However, no significant differences in skin temperature were found between recovery modes (Table 1).

Forearm and chest CVC are illustrated during rest, exercise, and postexercise recovery in Fig. 1. Fifteen minutes of exercise significantly increased forearm (50 ± 8% of maximum) and chest (40 ± 5% of maximum) CVC. After exercise, CVC at both sites decreased less during active recovery than during inactive recov-

Table 1. *Effect of mode of exercise recovery on temperature responses*

Variable	Recovery Mode	Baseline	Exercise	Recovery				
				Minute 1	Minute 2	Minute 3	Minute 4	Minute 5
T_{es} , °C	Active	36.7 ± 0.1	37.4 ± 0.2	37.4 ± 0.2	37.3 ± 0.2	37.3 ± 0.1	37.2 ± 0.2	37.2 ± 0.2
	Inactive	36.7 ± 0.1	37.2 ± 0.2	37.1 ± 0.2	37.2 ± 0.2	37.1 ± 0.1	37.1 ± 0.2	37.0 ± 0.1
T_{sk} , °C	Active	31.9 ± 0.3	32.4 ± 0.4	32.4 ± 0.4	32.4 ± 0.3	32.5 ± 0.3	32.4 ± 0.3	32.3 ± 0.3
	Inactive	31.7 ± 0.3	32.4 ± 0.4	32.5 ± 0.4	32.6 ± 0.3	32.6 ± 0.3	32.5 ± 0.3	32.4 ± 0.3

Values are means \pm SE. T_{es} , esophageal 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 baseline, exercise, or recovery periods for either variable.

ery. Sweat rate also increased significantly during exercise relative to baseline values (Fig. 2). The decrease in sweat rate during inactive recovery at both sites was significantly greater than the decrease in sweat rate during active recovery. Nevertheless, during both recovery conditions, sweat rate remained significantly above preexercise values (see Fig. 2).

Exercise significantly increased MAP, and, immediately after exercise, MAP decreased similarly during both inactive and active recovery conditions (see Fig. 3). Calculated total peripheral resistance decreased similarly during both exercise bouts (Fig. 4). However, throughout the recovery period, total peripheral resistance was significantly less during active recovery com-

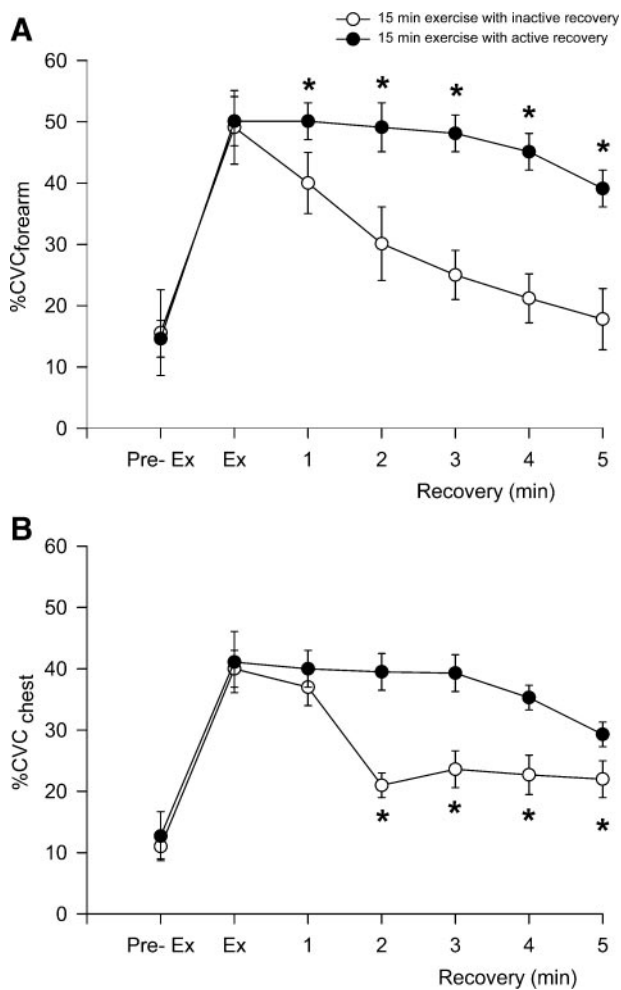


Fig. 1. Forearm (A) and chest (B) cutaneous vascular conductance (CVC) during 15 min of submaximal cycling exercise and 5 min of recovery from exercise. Values are means \pm SE; $n = 8$ subjects. Data are expressed as %maximum CVC. Pre-Ex, 1 min before beginning exercise; Ex, last minute of exercise. CVC during active recovery remained significantly elevated during the recovery period relative to inactive recovery. *Significant differences between inactive and active recovery modes, $P < 0.05$.

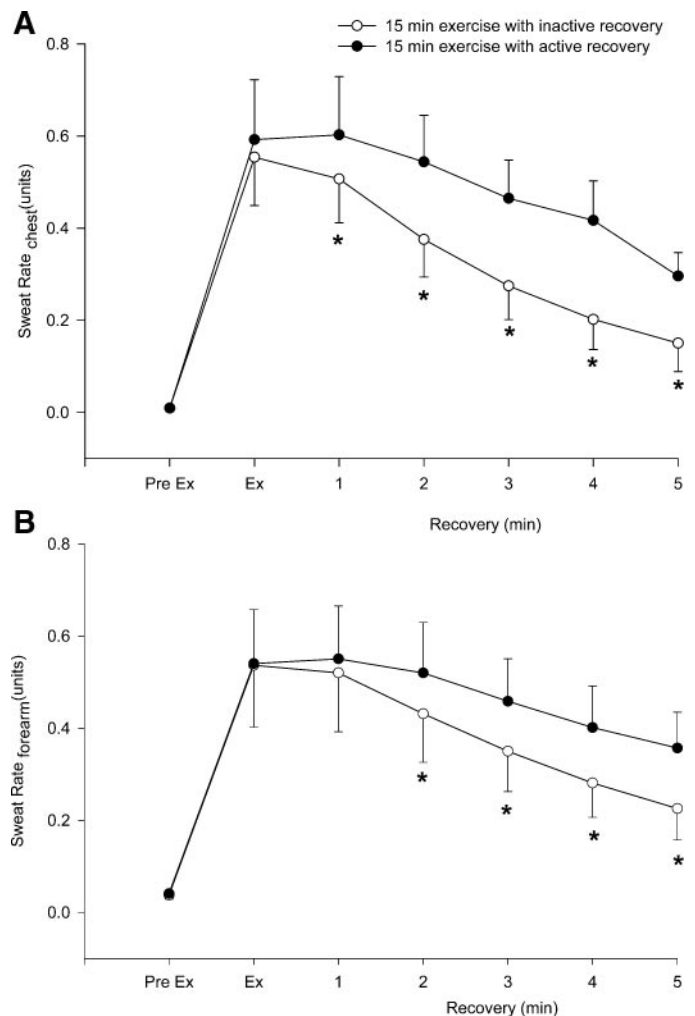


Fig. 2. Forearm (A) and chest (B) sweat rate during 15 min of submaximal cycling exercise and 5 min of recovery from exercise. Values are means \pm SE; $n = 8$ subjects. Units of sweat rate are $\text{mg} \cdot \text{cm}^{-2} \cdot \text{min}^{-1}$. Sweat rate during active recovery remained significantly elevated during the recovery period relative to inactive recovery. *Significant differences between inactive and active recovery, $P < 0.05$.

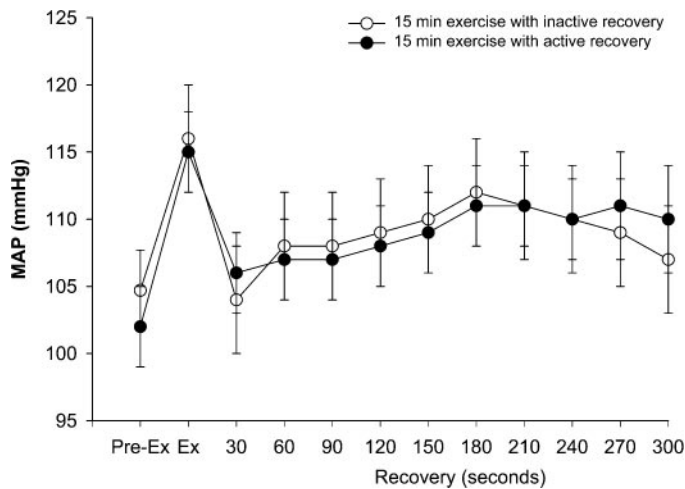


Fig. 3. Mean arterial blood pressure (MAP) during 15 min of submaximal cycling exercise and 5 min of recovery from exercise. Values are means \pm SE; $n = 8$ subjects. MAP was not different between modes of recovery for any period during or after exercise.

pared with inactive recovery. Total peripheral resistance increased to values near preexercise levels at the end of the inactive recovery period, whereas this variable remained near end-exercise values at the end of the active recovery period (see Fig. 4).

Exercise resulted in significant and similar increases in cardiac output, HR, stroke volume, and thoracic impedance (index of central blood volume; see Table 2). During active recovery, cardiac output, stroke volume, and central blood volume index were significantly elevated relative to during inactive recovery. In contrast, the return of HR toward preexercise levels was similar between recovery modes ($P = 0.25$).

DISCUSSION

The major findings of the present study were that active recovery from 15 min of dynamic exercise compared with inactive recovery 1) attenuated the fall in CVC and sweat rate and 2) was effective in maintaining stroke volume and cardiac output. In addition, total peripheral resistance was maintained near exercising values during active recovery but increased toward preexercise values during inactive recovery. Similar decreases in MAP were observed between inactive and active exercise recovery modes. The maintenance of stroke volume and cardiac output during active recovery is probably due to the activation of the skeletal muscle pump, whereas the mechanism(s) explaining differences between recovery modes with respect to CVC and sweat rate remain speculative.

The exercise bout caused expected increases in MAP, cardiac output, stroke volume, and HR, whereas it lowered total peripheral resistance. Exercise also increased CVC and sweat rate, thus indicating the activation of heat dissipation mechanisms. Esophageal and skin temperatures increased during exercise and remained significantly higher than baseline throughout both recovery periods. Moreover, there were no differences in skin or esophageal temperatures be-

tween recovery modes. This indicates that thermal stimuli from exercise were similar during exercise as well as during both recovery periods.

Results of the present study support our hypothesis that, after dynamic exercise, active recovery attenuates the decrease in CVC and sweat rate compared with inactive recovery. During active recovery, CVC was maintained at exercise levels despite a significant decrease in exercise workload (loadless pedaling). These differences in responses during recovery from exercise are likely due to one of two mechanisms: 1) greater arterial and/or cardiopulmonary baroreceptor unloading during inactive recovery and/or 2) an effect of central command to sustain CVC and sweat rate during active recovery.

In addition to thermoregulatory modulation, skin blood flow is also modulated by nonthermoregulatory responses, including baroreceptors during resting (7, 18) and exercise (22, 23) conditions. This mechanism is presumably in place to maintain arterial blood pressure during a hypotensive challenge, even at the expense of heat dissipation. In the present study, CVC during inactive recovery was significantly lower compared with the active recovery period. During inactive recovery the index of central blood volume (i.e., thoracic impedance) was significantly less, suggesting that cardiopulmonary baroreceptor unloading was greater in this condition. Thus, despite the maintenance of MAP during exercise recovery, greater unloading of the cardiopulmonary baroreceptors may contribute to the reduction in CVC during inactive recovery.

The role of baroreceptor unloading in modulating sweat rate is less clear than that in CVC. During rest (31) and exercise (22), some researchers have observed decreases in sweat rate with baroreceptor unloading. However, Wilson et al. (35) and Vissing et al. (33) did

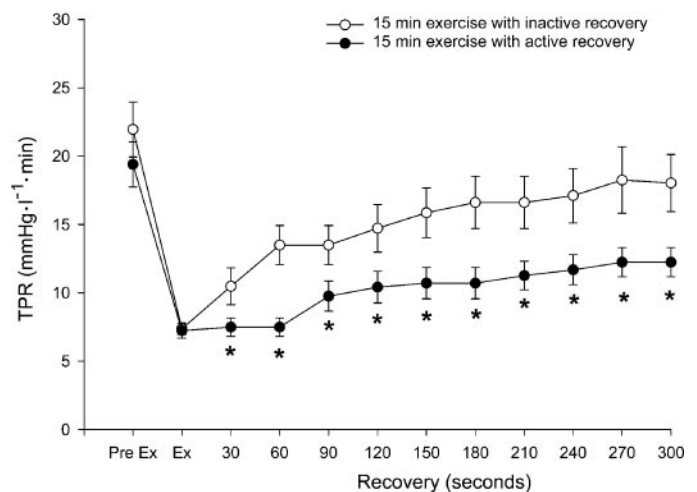


Fig. 4. Calculated total peripheral resistance (TPR) during 15 min of submaximal cycling exercise and 5 min of recovery from exercise. Values are means \pm SE; $n = 8$ subjects. TPR during active recovery was significantly lower throughout the recovery period relative to inactive recovery. *Significant differences between inactive and active recovery, $P < 0.05$.

Table 2. *Effect of mode of exercise recovery on cardiovascular responses*

Variable	Recovery Mode	Baseline	Exercise	Recovery				
				Minute 1	Minute 2	Minute 3	Minute 4	Minute 5
Q̇, l/min	Active	5.4 ± 0.6	16.5 ± 1.6	12.2 ± 1.3*	10.8 ± 1.1*	10.2 ± 0.9*	9.6 ± 0.2*	9.0 ± 0.8*
	Inactive	5.0 ± 0.5	16.1 ± 1.4	8.7 ± 1.1	7.7 ± 1.1	7.4 ± 1.1	6.9 ± 1.0	6.16 ± 0.6
SV, ml/beat	Active	76 ± 10	129 ± 10	119 ± 12*	114 ± 10*	113 ± 10*	109 ± 8*	105 ± 10*
	Inactive	71 ± 7	127 ± 10	92 ± 11	86 ± 11	86 ± 10	82 ± 10	75 ± 7
HR, beats/min	Active	68 ± 5	128 ± 5	102 ± 5	93 ± 6	88 ± 4	88 ± 3	86 ± 4
	Inactive	72 ± 4	127 ± 5	96 ± 7	89 ± 4	85 ± 3	84 ± 3	83 ± 3
TI, Ω	Active	22.2 ± 0.2	21.7 ± 0.2	21.6 ± 0.1*	21.7 ± 0.3*	21.8 ± 0.1*	21.7 ± 0.1*	21.8 ± 0.2*
	Inactive	22.1 ± 0.2	21.8 ± 0.3	22.1 ± 0.1	22.2 ± 0.3	22.3 ± 0.2	22.2 ± 0.2	22.3 ± 0.2

Values are means ± SE. Q̇, cardiac output, SV, stroke volume, HR, heart rate, TI, thoracic impedance; exercise, last minute of the 15-min exercise bout. *significant difference between mode of recovery (i.e., active vs. inactive), $P < 0.05$.

not observe decreases in sweating or changes in skin sympathetic nerve activity with baroreceptor unloading. Despite these apparently conflicting reports, data from the present experiment clearly show differences in sweating responses between modes of recovery. We cannot exclude the possibility that these differences in sweating responses were due to differences in cardiopulmonary baroreceptor unloading. However, the absence of differences in arterial blood pressure between modes of exercise recovery suggests that the disparity in sweating and CVC responses between exercise recovery modes was not due to differences in arterial baroreceptor unloading.

An alternate hypothesis explaining differences in both CVC and sweat rate between inactive and active recoveries may be due to greater central command associated with unloaded cycling during active recovery. There has yet to be a definitive study on the effects of central command on the control of CVC and sweating. Nevertheless, a number of studies suggest that central command may have a role in governing CVC and sweat rate during exercise (8, 9, 30). Thus it is possible that factors associated with central command contribute to differences in CVC and sweating between recovery modes. However, the fact that CVC did not appreciably change from peak exercise throughout the active recovery period, in which workload and central command were significantly reduced, suggests that central command does not play a critical in the CVC response during recovery from exercise.

Results of the present study support our hypothesis that engaging the skeletal muscle pump during active recovery after exercise, compared with inactive recovery, attenuated the fall in cardiac output and stroke volume. This muscle pump presumably increased venous return (preload) and, therefore, maintained cardiac output and stroke volume in the active recovery condition. In contrast to cardiac output and stroke volume, the decreases in MAP were similar between inactive and active recovery modes. This result was different than expected because we hypothesized that the fall in MAP during inactive recovery after 15 min of exercise would be greater relative to the fall in MAP during active recovery. Moreover, this finding is in contrast to our laboratory's prior findings in which, after 3 min of exercise, the decrease in MAP was

greater during inactive recovery relative to during active recovery (4, 5). It is unclear why MAP responses during exercise recoveries are apparently different between 3 and 15 min of exercise, except that with 15 min of exercise the individual is exposed to a greater thermal load.

In the present study, total peripheral resistance remained significantly less during active recovery compared with during inactive recovery (see Fig. 4). A possible mechanism leading to this response may be due to sustained local vasodilator substances secondary to metabolism associated with active recovery. Such a response will cause the vasculature of the leg to remain in a relatively vasodilated state compared with that during inactive recovery. In support of this hypothesis, others have shown that leg blood flow increases 1–3 l/min during the transition from rest to unloaded (or very low loads; 10 W) exercise without proportional increases in blood pressure (1, 29). Thus lower total peripheral resistance during unloaded cycling may be due to reduced leg vascular resistance relative to that during inactive recovery. Alternatively, or in addition, because cardiopulmonary baroreceptors are likely unloaded to a greater extent during inactive recovery, coupled with findings that cardiopulmonary baroreceptor unloading will increase total peripheral resistance, differences in total peripheral resistance between active and inactive recovery conditions could be secondary to cardiopulmonary baroreceptor unloading. Finally, differences in CVC (and thus cutaneous vascular resistance) between recovery modes (see Fig. 1) likely contribute to the observed differences in total peripheral resistance.

Previously, our laboratory identified a separation in HR responses during mode of exercise recovery after 3 min of exercise (5). In the present study, no differences were observed in HR between recovery modes as HR remained elevated after 15 min of exercise under both conditions. This elevation in HR may be associated with the elevation in body temperature, as elevated temperature increases HR independent of autonomic control (11, 12, 17). A lack of difference in HR between modes of recovery may suggest that central command is similar during these conditions. However, such a hypothesis ignores the aforementioned impact of elevated internal temperature in increasing HR. Thus

central command could still be greater during active recovery despite no difference in HR, if HR during both modes of recovery is primarily mediated by elevated internal temperature. Such a hypothesis is supported by our laboratory's prior investigation in which, after brief exercise (3 min) that did not increase internal temperature, HR was significantly elevated during active recovery, whereas HR during inactive recovery returned to preexercise baseline before the end of the 5-min recovery period (5).

Limitations to the interpretation of the data. Measurement of skin blood flow with laser-Doppler flowmetry is sensitive to motion artifact. Thus it may be concluded that differences in CVC between recovery modes were due to motion artifact associated with unloaded cycling relative to a lack of motion artifact during inactive recovery. To address this concern, we statistically compared CVC at baseline with CVC during the first minute of loadless pedaling before increasing workload (data not shown). CVC was not different between these conditions for either the forearm or the chest. Thus we conclude that motion artifact associated with loadless cycling was not responsible for the large difference in CVC observed between recovery modes. Moreover, differences in sweating responses between recovery modes would be immune to motion artifact.

In the present protocol we did not measure oxygen consumption during exercise or exercise recovery, nor did we measure intramuscular temperature. Previous studies have shown that whole body and muscle oxygen consumption are elevated during active recovery compared with inactive recovery (3). Accordingly, we recognize that metabolism, and thus muscle heat production, will be higher during active recovery relative to inactive recovery. However, despite the probability that muscle temperature was slightly greater during active recovery, we did not observe any differences in esophageal temperature between recovery modes. This apparent contradiction limits the interpretation of the esophageal temperature data as it relates to muscle temperature. Nevertheless, we are unaware of data supporting the possibility that subtle differences in muscle temperature during unloaded cycling relative to passive recovery can cause such large differences in CVC and sweat rate between recovery modes (see Figs. 1 and 2) either through thermosensitive afferents in the muscle or through small differences in blood temperature as a result of muscle that is slightly warmer during active recovery.

CONCLUSIONS

These data demonstrate that postexercise recovery mode can affect CVC and sweat rate. Hence, mode of recovery modifies both cardiovascular as well as thermoregulatory responses after sustained dynamic exercise sufficient to cause thermal stress. Furthermore, persistent cutaneous vasodilation and sweating during mild active exercise recovery may be important for postexertional heat dissipation, although the mecha-

nism(s) for these altered responses during active recovery remains unknown.

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