

Muscle mechanoreceptor modulation of sweat rate during recovery from moderate exercise

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Shibasaki, Manabu, Mieko Sakai, Mayumi Oda, and Craig G. Crandall. Muscle mechanoreceptor modulation of sweat rate during recovery from moderate exercise. *J Appl Physiol* 96: 2115–2119, 2004. First published February 6, 2004; 10.1152/jappphysiol.01370.2003.—The objective of this study was to identify whether muscle mechanoreceptor stimulation is capable of modulating sweat rate. Seven healthy subjects performed two 20-min bouts of supine exercise on a tandem cycle ergometer (60 rpm at 65% of maximal heart rate). After one bout, the subject stopped exercising (i.e., no pedaling), whereas, after the other bout, the subject's legs were passively cycled (at 60 rpm) via a second person cycling the tandem ergometer. This allows for mechanical stimulation of muscle with minimal activation of central command. Esophageal temperature (T_{es}), mean skin temperature (T_{sk}), heart rate, mean arterial blood pressure, oxygen consumption, cutaneous vascular conductance (CVC), and sweat rate were not different during the two exercise bouts. Regardless of the mode of exercise recovery, there were no differences in T_{es} , T_{sk} , or CVC. In contrast, early in the recovery period, chest and forearm sweat rate were significantly greater in the passive cycling recovery mode relative to the no-pedaling condition (chest: 0.57 ± 0.13 vs. 0.39 ± 0.14 , forearm: 0.30 ± 0.05 vs. 0.12 ± 0.02 $\text{mg}\cdot\text{cm}^{-2}\cdot\text{min}^{-1}$; both $P < 0.05$). These results suggested that muscle mechanoreceptor stimulation to the previously activated muscle is capable of modulating sweat rate.

metaboreceptor; sweating response

ELEVATED INTERNAL AND SKIN temperatures lead to increases in sweat rate and skin blood flow. Independent of thermal control of these variables, sweat rate and skin blood flow can also be affected by nonthermal factors such as exercise, baroreceptor loading status, and body fluid status (10, 20, 23, 26). During exercise, heart rate and mean arterial pressure (MAP) are elevated via a combination of central command and muscle afferent (i.e., mechanoreceptor and metaboreceptor) stimulation (16). Similarly, our laboratory found that sweat rate can also be modulated by central command and muscle metaboreceptor stimulation during isometric exercise (22, 24). Kondo et al. (13) compared the increase in chest sweat rate during mechanoreceptor stimulation, via 2 min of passive limb movement, in mildly heated subjects with previously stable sweat rates. However, in that study, because the increase in sweat rate during passive limb movement was quite small, it would be difficult to identify whether this change in sweat rate was due to the mild heat stress or mechanoreceptor stimulation. There-

fore, the effects of mechanoreceptor stimulation in modulating sweat rate remain unclear.

A number of studies indicate that sweat rate and skin blood flow can be modulated by nonthermal factors during the postexercise period (4, 9, 11, 30). Previously Carter et al. (4) reported that, after moderate exercise in the upright position, sweat rate and skin blood flow during loadless cycling (i.e., 0 W) was elevated relative to when the subjects did not pedal during recovery. After that study, Wilson et al. (30) performed a similar protocol; however, subjects were in the supine position during exercise and recovery to minimize baroreceptor unloading associated with recovery in the upright position. They reported that, after exercise, sweat rate but not cutaneous vascular responses was significantly elevated during unloaded active cycling. These data suggest that, after exercise, sweat rate is influenced by either central command and/or muscle mechanoreceptor stimulation during unloaded active cycling. However, the contribution of muscle mechanoreceptor stimulation relative to central command in causing the elevated sweating response during active recovery could not be identified in that study.

Passive limb movement using a tandem ergometer has been employed to investigate the role of muscle mechanoreceptor stimulation, independent from the contribution of central command, during exercise (3, 18, 19, 29). The present study used this approach to test the hypothesis that sweat rate is modulated by muscle mechanoreceptor stimulation during the recovery period from exercise.

METHODS

Subjects. Seven healthy male subjects (age, 20.4 ± 0.2 yr) participated in this study. All subjects were of normal weight (64.0 ± 1.6 kg) and height (172.9 ± 2.0 cm). They were informed of the purpose and risks of this institutionally approved study, and all participants reviewed and signed an informed consent document. All subjects were healthy nonsmokers and free of any known cardiovascular, neurological, or metabolic diseases. Subjects refrained from alcohol and stimulants such as caffeine for 24 h before testing.

Experimental protocol. Each subject performed the exercise protocol described below on separate days, with each bout being separated from the prior bout by a minimum of 48 h. Experiments were performed in a temperature-controlled laboratory ($27.5 \pm 0.5^\circ\text{C}$). After being exposed to this temperature for 1 h, the subject lay supine at the rear position of an adapted supine tandem cycle ergometer (Panasonic bicycle). The subject remained in this position for 30 min while additional measurement devices were attached. After 5 min of

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baseline data collection, each subject performed 1 min of loadless exercise while remaining in the supine position. This was immediately followed by the subject exercising for 20 min at a workload that elicited 65% of the individual's predicted maximal heart rate at a pedaling cadence of 60 rpm (Fig. 1). After the exercise bout, the subject either stopped all leg movement or, on a separate day, the subject's legs were passively moved (at 60 rpm) via a second person cycling the tandem ergometer. The order of postexercise treatment was randomized, and each bout of exercise was performed at the same time of day (1). Data were obtained throughout exercise and for 20 min of recovery. Recovery period consisted of two phases (Fig. 1). During the first 10 min of recovery, the subjects either rested or passively cycled as described above. During the subsequent 10 min, subjects rested without any leg movements (inactive recovery) to confirm whether the differences between recovery modes in the first 10 min, if any, resulted from the passively leg movements.

Measurements. Each subject was instrumented for the measurement of esophageal temperature with a probe (thermistor) swallowed to the level of the left atrium (15). Skin temperatures were measured at seven sites by thermocouples, and mean skin temperature was calculated according to the method of Hardy and DuBois (8). Heart rate was obtained from the electrocardiogram signal (model 1000PN, NEC). Systolic and diastolic blood pressures were recorded every minute by electrophygmomanometry of the left upper arm (model STBP780, Colin). MAP was calculated as diastolic pressure plus one-third pulse pressure. Electromyography (EMG) was continuously measured from the rectus femoris muscle to confirm the absence of voluntary muscle contracting during recovery modes.

Sweat rate was continuously recorded from two sites (right forearm and chest) by using the ventilated capsule method with compressed nitrogen as the perfusion gas delivered at a rate of 500 ml/min. Capsules (surface area = 2.01 cm²) were attached on the skin by using circular double-stick tape. Absolute humidity was calculated from relative humidity and temperature of the gas exiting the chambers, with the detector (model HMP 233, Vaisala) positioned 1 m from the capsule on the skin. Sweat rate was calculated as

$$\text{Sweat rate (mg}\cdot\text{cm}^{-2}\cdot\text{min}^{-1}) = \text{absolute humidity (mg/m}^3) \times \text{gas flow (m}^3\text{/min)/capsule surface area (cm}^2\text{)}$$

Chest and forearm skin blood flows were monitored from integrated laser-Doppler flowmetry probes (Moor) placed adjacent to each sweat capsule. After the exercise bout and recovery periods, local skin temperature was elevated to 43°C by a 3-cm heating device that housed the laser-Doppler flow probe. Local temperature was held at this level for 40 min to elicit maximal cutaneous vasodilation (27). An index of cutaneous vascular conductance (CVC) was calculated from the ratio of laser Doppler flux to MAP. CVC was then expressed as a

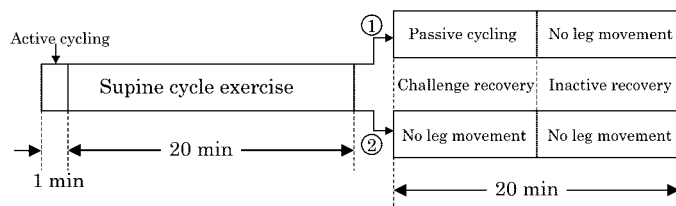


Fig. 1. Schematic depicting the exercise and recovery protocols. Each subject exercised in the supine position for 20 min, followed by either 10 min of passive cycling or no leg movement. Passive cycling was achieved by a second person pedaling the tandem ergometer while the subject's legs passively moved. This 10-min period of recovery was termed "challenge recovery." After this 10-min period, data were collected for an additional 10 min (inactive recovery) during which time no leg movement occurred for either recovery mode. For data analysis, the first 10 min of recovery were subdivided into early recovery and late recovery phases (not illustrated in this figure). The order of recovery modes was randomized.

percentage of maximal cutaneous vasodilation as determined from local heating. Oxygen consumption was measured with an automated metabolic analyzer (Arco system) at rest and the last 5 min of exercise through the end of the recovery period.

Data analyses. Data were continuously acquired (Biopac, Santa Barbara, CA) and averaged into the following 1-min stages for both protocols: preexercise, last minute of exercise, and recovery minutes 1–10. Average data before exercise were statistically compared with data at the end of exercise by using Student's paired *t*-test. Data were analyzed via a two-way repeated-measures ANOVA with main factors of recovery mode (i.e., rest or passive limb movement) and stage of recovery (i.e., early and late recovery). Early recovery was defined as the first 5 min of the initial recovery period, and late recovery was defined as the last 5 min of the initial recovery period. At minutes 5 and 10 of the challenge recovery period, differences between recovery modes were compared by using Student's paired *t*-test. Statistical significance was set at an α level of 0.05. All data are presented as means \pm SE.

RESULTS

The increase in sweat rate during exercise was similar between exercise bouts such that there was no difference in this variable at the end of exercise (Fig. 2). Forearm sweat rate was significantly greater throughout passive cycling recovery relative to during no-pedaling recovery. Midway through the recovery period, forearm sweat rate was significantly greater in the passive cycling recovery mode relative to the no-pedaling condition (0.30 ± 0.05 vs. 0.12 ± 0.02 mg \cdot cm⁻² \cdot min⁻¹; $P < 0.05$) and remained greater through the end of the challenge recovery period (0.14 ± 0.02 vs. 0.07 ± 0.01 mg \cdot cm⁻² \cdot min⁻¹; $P < 0.05$). In contrast, chest sweat rate was significantly greater at the midrecovery period during passive cycling (0.57 ± 0.13 vs. 0.39 ± 0.14 mg \cdot cm⁻² \cdot min⁻¹; $P < 0.05$) but not at the end of the challenge recovery period.

Preexercise chest and forearm CVC were not different between the two trials (Table 1). Both chest and forearm CVC significantly increased during exercise, whereas no differences in either variable were observed at the end of exercise between exercise bouts. In contrast to sweat rate, there were no differences in chest or forearm CVC between recovery modes.

The average workload subjects performed was 85.7 ± 7.5 W. Before exercise, and at the 20th min of exercise, heart rate and oxygen consumption were not different between the two exercise bouts. However, both of these variables were slightly, but significantly, greater throughout passive cycling recovery relative to no-pedaling recovery. Both of these variables were not different between recovery modes during the final 10 min of the 20-min recovery period, when no leg movement occurred.

MAP increased to the same level during both exercise bouts, and it decreased similarly during both recovery modes (all $P > 0.05$). Esophageal temperature at rest, throughout exercise, and throughout recovery was not significantly different between exercise bouts or modes of recovery (Fig. 2, Table 1). Although there was a slight difference in mean skin temperature before exercise, after that time there were no significant differences in mean skin temperature throughout the protocol (Table 1).

Figure 3 shows EMG activity throughout exercise and recovery from a subject. Coordinated EMG activity during passive cycling recovery was clearly diminished, whereas EMG during active unloaded (0 W) cycling showed visible bursts.

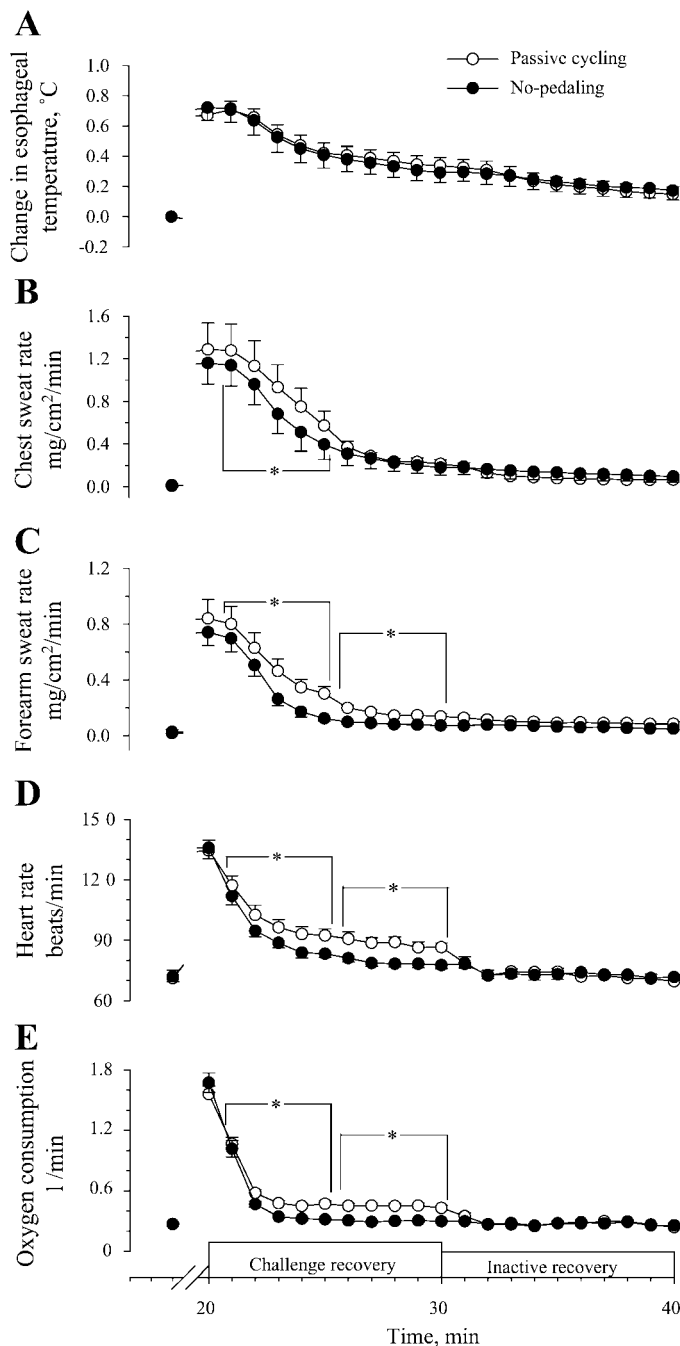


Fig. 2. Effect of mode of exercise recovery after 20 min of cycle ergometry exercise on the change in esophageal temperature (A), chest (B) and forearm (C) sweat rate, heart rate (D), and oxygen consumption (E). At the end of exercise (20 min), there were no differences in any variable. At no time during the experimental protocol was esophageal temperature different between trials. Forearm sweat rate was significantly greater during passive cycling relative to no-pedaling recovery, whereas chest sweat rate was significantly greater only during the early recovery period. Similarly, heart rate and oxygen consumption were significantly greater during passive cycling relative to no-pedaling recovery. 20-min Data point, data from the final minute of exercise; 20- to 30-min period, data during passive cycling and no-pedaling recoveries (challenge recovery); 30- to 40-min period, no leg movement for either trial (inactive recovery). Values are means \pm SE. *Values during passive cycling are significantly greater than the no-pedaling recovery mode, $P < 0.05$.

DISCUSSION

The primary finding of this study is that, despite no differences in sweat rate at the end of the two exercise bouts, when muscle mechanoreceptors were stimulated during postexercise recovery (i.e., passive cycling), forearm and chest sweat rate were greater during passive cycling recovery relative to no-pedaling recovery. Throughout exercise and recovery, there were no significant differences in esophageal or mean skin temperatures between bouts. These data suggest that muscle mechanoreceptor stimulation is capable of modulating sweat rate independent from the contributions of central command and thermal factors.

During the passive cycling recovery period, another person pedaled the ergometer to stimulate muscle mechanosensitive afferents of the subject without voluntary movement by the subject. The observation, shown in Fig. 3, was consistent across subjects, indicating that voluntary movements during passive cycling recovery were minimized. Heart rate was slightly but significantly greater during passive cycling recovery relative to no-pedaling recovery. Despite some studies showing that muscle afferent stimulation is capable of increasing heart rate (16, 17), other studies have not reported increases in heart rate during passive cycling via a tandem ergometer (3, 19). However, in those studies, the subjects were in the upright posture and exercise was performed over a shorter period of time and in a cooler environment relative to the present study. Thus it is possible that differences in heart rate during passive cycling in the aforementioned studies, relative to the present study, may be related to these methodological differences. Nevertheless, the absence of coordinated EMG activity during passive cycling in all subjects supports the conclusion that passive cycling primarily stimulated muscle mechanoreceptors with minimal activation of central command.

Forearm sweat rate during passive cycling recovery was greater relative to no-pedaling recovery throughout the recovery period (Fig. 2). It is interesting to note that, shortly after the cessation of passive cycling recovery, forearm sweat rate was no longer different between recovery modes. However, it is recognized that there was a general trend for differences in forearm sweat rate between recovery modes to be minimized near the end of the passive cycling recovery period. In contrast, chest sweat rate was significantly greater during passive cycling recovery early in the recovery period (i.e., first 5 min), followed by an absence of difference in chest sweat rate between recovery modes (Fig. 2). The observation that differences in sweat rate between recovery modes were minimized near the end of the recovery period suggests that muscle mechanoreceptor stimulation itself may be insufficient to maintain elevated sweating responses. Therefore, two conceivable hypotheses may explain this response. First, during the initial period after the end of exercise, muscle metaboreceptors will remain stimulated until the substances responsible for this stimulation are removed. Given prior findings that accumulated metabolites in the exercising muscle sensitize muscle afferent (5, 6), then mechanoreceptor stimulation of sweat rate may wane as muscle metaboreceptor stimulation is minimized. Second, elevated muscle temperature has been shown to sensitize muscle metaboreceptor activation (21). Although speculative, elevated muscle temperature due to exercise may sensitize muscle mechanoreceptor activation. As the recovery

Table 1. Esophageal and mean skin temperatures, mean arterial pressure, and chest and forearm cutaneous vascular conductance at rest, at the end of exercise, at minutes 5 and 10 of the recovery period, and at the end of the test

	Rest	End of Exercise	Recovery Minute 5	Recovery Minute 10	End of Test
T_{es}					
Passive	36.8±0.1	37.4±0.1	37.2±0.1	37.1±0.1	36.9±0.1
No pedaling	36.7±0.1	37.4±0.1	37.1±0.1	37.0±0.1	36.9±0.1
\bar{T}_{sk}					
Passive	33.0±0.1*	32.7±0.2	32.5±0.1	32.2±0.2	32.6±0.2
No pedaling	32.7±0.2	32.5±0.2	32.6±0.2	32.7±0.2	32.8±0.2
MAP					
Passive	86.9±3.0	98.0±3.7	88.0±3.2	87.6±3.4	87.8±2.4
No pedaling	85.0±2.0	100.2±1.9	87.9±1.7	86.8±1.7	86.4±1.9
CVC chest					
Passive	21.0±6.1	60.5±6.8	40.1±7.1	32.6±6.4	20.1±3.6
No pedaling	22.9±7.2	56.1±4.9	39.3±5.9	30.3±5.5	15.9±1.9
CVC forearm					
Passive	18.7±3.5	57.4±3.6	47.7±6.9	41.3±6.4	32.1±6.5
No pedaling	16.5±2.7	57.0±3.5	45.8±6.3	41.4±7.0	27.2±5.0

Values are means ± SE. T_{es} , esophageal temperature; \bar{T}_{sk} , mean skin temperature; MAP, mean arterial pressure; CVC, cutaneous vascular conductance. *Significantly greater than the no-pedaling recovery mode, $P < 0.05$.

period persists, and muscle temperature returns to preexercise levels (12), then the hypothesized sensitization of muscle mechanoreceptors would be minimized, resulting in similar sweating responses between recovery modes.

It remains controversial whether baroreceptors modulate sweat rate, with some studies supporting this hypothesis (7, 14, 25) and others not supporting it (22, 28, 31). Nevertheless, given the possibility that baroreceptors can modulate sweat rate, the present study was conducted with the subjects in the supine posture to eliminate differences in baroreceptor-loading status between recovery modes (30). This contention is supported by a prior observation that central venous pressure and thoracic impedance were not different during no-load pedaling relative to no-pedaling conditions when the subjects were supine (30), coupled with the present findings of no differences in arterial blood pressure between recovery modes (Table 1). Thus differences in sweat rate in the present study were not due to differences in baroreceptor loading between recovery modes.

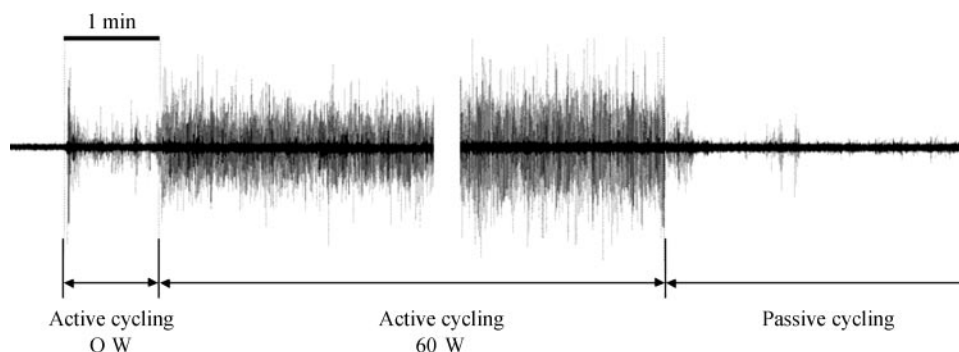
Consistent with the aforementioned study (30), with the subjects in the supine position, there were no differences in CVC between recovery modes (Table 1). These data confirm what was previously supposed (30): that it is unlikely that muscle mechanoreceptor stimulation modulates neural control of skin blood flow during recovery from exercise.

Limitation of this study. Esophageal temperature was measured as an index of internal temperature because of its rapid

response when internal temperature changes (2). Neither hypothalamic, spinal, nor muscle temperatures were recorded in the study, and thus we do not know whether temperatures in these regions were higher during passive cycling despite the absence of a difference in esophageal temperature. Oxygen consumption was slightly elevated during passive cycling. Therefore, of the aforementioned sites, muscle temperature would have the greatest possibility of being elevated during passive cycling recovery due to increased metabolism. However, we are unaware of findings that support the hypothesis that slightly elevated muscle temperatures, if present in this study, would alter sweat rate. Moreover, if during passive exercise hypothalamic, spinal, or muscle temperature was slightly elevated and this was the mechanism for elevated sweat rate, then CVC would similarly be expected to be affected by these thermal afferents, which it was not (see Table 1). Therefore, it is unlikely that elevated temperatures in other regions of the body were the stimulus for the separation in sweat rate between recovery modes.

Conclusion. In this study, 20 min of exercise elevated internal temperature and subsequently increased sweat rate and skin blood flow. During the subsequent recovery period, when muscle mechanoreceptors were stimulated via passive cycling, forearm and chest sweat rates were significantly elevated relative to recovery without muscle mechanoreceptor stimulation. The absence of a difference in esophageal and skin temperatures between recovery modes strongly suggests that

Fig. 3. Electromyograph from a representative subject during loadless cycling (0 W), 60-W cycling, and passive cycling (i.e., legs passively moved by additional person pedaling on a tandem ergometer). This figure demonstrates the absence of coordinated electromyograph activity during passive pedaling relative to 0- and 60-W cycling.



muscle mechanoreceptor stimulation was the primary stimulus leading to this elevation in sweat rate. In contrast, the absence of a difference in CVC between recovery modes supports a prior observation (30) that muscle metaboreceptor stimulation does not affect CVC during the recovery from exercise. The present and prior findings strongly suggest that sweat rate is capable of being modulated by a variety of factors associated with exercise such as activation of central command (24), muscle metaboreceptor stimulation (22), and now muscle mechanoreceptor stimulation.

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