 Thermoregulation during cold exposure: effects of prior exercise

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EXERCISE HAS BEEN CONJECTURED to increase an individual's risk of hypothermia during cold exposure (3, 5, 27). However, experimental and clinical evidence for this are largely anecdotal. Over 30 years ago, Pugh (18, 19) concluded that exercise-induced fatigue was an etiologic factor predisposing hikers, climbers, and outdoor-sportsmen to hypothermia, but he provided no data demonstrating this belief with a physiological mechanism for this predisposition. Recently, Thompson and Hayward (25) suggested that exercise during cold-wet exposure may fatigue shivering thermogenesis, but their findings did not definitively support their speculation. Others (16, 28) have reported that exercise performed before subsequent cold-water immersion exacerbates the fall in core temperature (T_core), but these results were inconclusive because premersion T_core differed between the experiments (16), or a cross-sectional methodology was employed (28). Furthermore, because water has such a high thermal conductivity, peripheral heat loss during cold-water immersion may be too pronounced for exercise effects on thermal balance and thermoregulatory effector responses to be detected.

Exercise could increase the risk of hypothermia during subsequent cold exposure for several reasons. First, exercise might mediate "thermoregulatory fatigue," which would blunt shivering responses and reduce vasoconstriction during subsequent cold exposure. For example, our laboratory (29) has observed that a prolonged period of physical exertion coupled with sleep deprivation and negative energy balance resulted in a lowered threshold for shivering, despite normal plasma glucose concentrations. Those findings, however, did not allow isolation of the effects of previous exercise from sleep deprivation and negative energy balance. Second, cold exposure immediately after performing leg exercise might result in accentuated heat loss from "thermoregulatory lag." Thermoregulatory responses are aimed at facilitating heat dissipation during exercise in temperate conditions (21), and substantial cool exposure might mediate a "lag" in switching from heat loss to conservation. Evidence for this might include increased heat loss from areas of active cutaneous vasodilation such as the torso and arms. Third, exercise might mediate greater heat loss during subsequent cold exposure due to "heat redistribution" to active limbs. During exercise, active skeletal muscle increases perfusion, and perfusion can remain elevated for extended durations (24), facilitating regional heat loss over these active limbs during exercise (20). Evidence for heat redistribution might include greater regional heat loss over the active limbs (legs) during subsequent cold exposure.

This study examined whether exercise impairs the body's capability to maintain thermal balance during subsequent cold exposure. To distinguish between these potential mechanisms, and the "thermal" consequences of exercise (increased T_core), control experiments were performed after passive heating to elevate the initial T_core to the same levels achieved by exercise.

METHODS

Subjects. Ten healthy men volunteered to participate in this study as test subjects. Physical characteristics were age, 24.7 ± 1.7 (SE) yr; height, 176.8 ± 2.1 cm; mass, 78.1 ± 3.5 kg; body surface area, 1.93 ± 0.05 m²; peak oxygen uptake

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Cold Exposure After Exercise

Preliminary testing. Body composition was measured by using dual-energy X-ray absorptiometry (model DPX-L, Lunar, Madison, WI). Mean skinfold thickness was calculated from 10 sites according to Allen et al. (1). All subjects completed an incremental cycle ergometer test for determination of VO2peak. Briefly, subjects pedaled at 70 W for 2 min, with the resistance increased by 35 W every 2 min until the subject was exhausted and could no longer maintain the exercise intensity.

Experimental design. Subjects completed two experimental trials, on separate days, spaced by 1 wk. Subjects refrained from smoking, taking medication, and exercising 12 h before any testing session. Each trial consisted of a standardized cold-air test (CAT) preceded by one of two manipulations: A) exercise (Ex) or B) passive heating (Heat). The Ex trial consisted of 60-min semirecumbent cycle ergometer exercise, with the subject immersed to should level in a water-immersion pool at 35.0 ± 0.1°C followed by the CAT. The immersion pool held ~36,000 liters and is controlled within 0.5°C by a temperature-control system. Mean exercise intensity was 55.4 ± 0.5°C by a temperature-control system. Mean exercise intensity was 55.4 ± 2.3% VO2peak for Ex. The Heat trial consisted of sitting in the immersion pool at 38.2 ± 0.0°C until rectal temperature (Tre) rose to match that at the completion of Ex followed by the CAT. This approach precluded using a randomized design, and the Heat trial always followed the Ex trial. Immediately after Ex or Heat, subjects towed off, changed into dry shorts and socks, and were taken to the anteroom of the cold chamber for baseline measurements. This took ~20 min. Five minutes of baseline data (body temperatures, heart rate, metabolic rate) were collected outside the cold-air chamber (22.8 ± 0.8°C) while the subjects sat quietly, and then they rose and walked into the cold-air chamber (4.6 ± 0.1°C) and reclined for up to 120 min in a nylon-mesh lounge chair. While reclining, the subjects sat quietly and were not allowed to employ behavioral thermoregulation. The trials were all conducted at the same time of day to control for the potential influence of circadian rhythmicity.

Measurements and calculations. Tre was measured by a thermistor inserted 10 cm past the anal sphincter. Integrated heat flow and skin temperature (Tsk) disks (Concept Engineering, Old Saybrook, CT) were secured at five (in water) and eight (CAT) sites (right side of the body). Mean weighted skin temperature (Tsk) during water immersion was calculated as follows: $T_{sk} = 0.28T_{subscapular} + 0.14T_{forearm} + 0.087T_{triceps} + 0.22T_{calf} + 0.28T_{lateral thigh}$. During CAT, $T_{sk}(°C)$ was calculated as follows: $T_{sk} = 0.06T_{foot} + 0.17T_{calf} + 0.28T_{lateral thigh} + 0.14T_{cheek} + 0.07T_{tricep} + 0.07T_{forearm} + 0.14T_{subscapular} + 0.07T_{hand}$. Mean weighted heat flow (RF, W·m⁻²) was calculated as follows: $RF = 0.06HF_{foot} + 0.17HF_{calf} + 0.28HF_{lateral thigh} + 0.14HF_{cheek} + 0.07HF_{tricep} + 0.07HF_{forearm} + 0.14HF_{subscapular} + 0.07HF_{hand}$. Tissue insulation was calculated as follows: $I_{ti} = (T_{re} - T_{sk})/RF(10)$. Mean body temperature ($T_b$) was calculated as follows: $T_b = 0.8T_{re} + 0.2T_{sk}$; during CAT, $T_b = 0.6T_{re} + 0.33T_{sk}$ (26). Temperature and heat flow measurements were made continuously by using an automated data-acquisition system.

Oxygen uptake (VO2) was measured by using an automated metabolic-measurement and -analysis system (model 2900, Sensormedics, Yorba Linda, CA) at minutes 0 (baseline) and 30 during the water immersion. During CAT, VO2 was measured at minutes 0 (baseline), 15, 35, 55, 75, 95, and 115. Metabolic heat production (M, W·m⁻²) was estimated from the VO2 and respiratory exchange ratio (R) by using the following equation (8): $M = [0.23(R) + 0.77] (5.873)(VO2)/(60A_b)$, where $A_b$ (6) is body surface area (m²).

Cumulative body heat debt was defined as the total negative heat storage integrated over time and expressed as a positive number. Body heat storage ($S$, W·m⁻²) was calculated: $S = M - W - L - K - E - (R + C)$, where $M$ is the metabolic rate, $W$ is work rate (0 in this experiment), $L$ is the respiratory heat losses by convection and evaporation, E is evaporative heat loss (set at 4.1 W·m⁻² in this experiment), $K$ represents conductive heat loss (0 in this experiment), and $R + C$ represents dry heat loss, measured by heat flow disks (8, 26).

Blood was drawn from an indwelling venous catheter (antecubital) in the left arm before the CAT began (minute 0) and at minutes 15, 30, 60, 90, and 120 during CAT. Catheter patency was maintained between blood draws by injecting heparinized saline into the catheter. Blood samples were analyzed to determine plasma glucose concentration by using an autoanalyzer (model 2300, Yellow Springs Instrument) to ensure that subjects maintained euglycemia. Plasma norepinephrine (NE) was determined by gas chromatography (31).

Statistical analyses. Data were analyzed by using a two-way repeated-measures analysis of variance. When significant F-ratios were calculated, paired comparisons were made post hoc by using Newman-Keuls tests. The slope and threshold of each individual $T_{re}$ vs. change in $M (∆M)$ relationship was determined by least squares linear regression. Paired t-tests were used to determine whether differences in slope or intercept data existed between Ex and Heat for $T_{re}$ vs. $∆M$. Data are reported as means ± SE. Significance was accepted at $P < 0.05$.

RESULTS

Water immersion. All subjects completed 60 min of cycling during Ex. The mean immersion time required during Heat to match the $T_{re}$ rise observed during Ex was 53.4 ± 5.0 min. The mean $T_{re}$ at the end of the immersion periods were 38.19 ± 0.14 and 38.08 ± 0.10°C, during Ex and Heat, respectively $(P > 0.05)$. The average VO2 during immersions were 1.97 ± 0.12 and 0.34 ± 0.02 l/min for Ex and Heat, respectively $(P < 0.05)$. For Ex, this VO2 corresponded to 55.4 ± 2.3% of the measured VO2peak. Final heart rates during immersion were 149.3 ± 6.1 and 102.1 ± 3.1 beats/min for Ex and Heat, respectively $(P < 0.05)$. Weight loss from sweat was 1.07 ± 0.15 and 1.06 ± 0.18 kg during Ex and Heat, respectively $(P < 0.05)$.

$T_{re}$ (CAT). During the transition from the immersion pool to the cold-air chamber, $T_{re}$ fell during Heat. Therefore, $T_{re}$ at minute 0 was slightly but significantly higher (0.14°C, $P < 0.05$) in Ex vs. Heat (Fig. 1). By minute 10 of cold-air exposure, differences between trials were no longer apparent. However, by minute 40 of CAT, $T_{re}$ had fallen lower $(P < 0.05)$ during Ex compared with Heat, and the difference between trials grew larger as exposure continued to minute 120. The cooling rate from minute 10 to the end of the exposure was faster $(P < 0.05)$ for Ex ($−0.64 ± 0.07°C/h)$ than Heat ($−0.57 ± 0.04°C/h)$.

$T_{sk}$ (CAT). $T_{sk}$ and the $T_{re}−T_{sk}$ gradient are shown in Fig. 2. Cold-air exposure caused $T_{sk}$ to decrease until a new steady-state value of ~23°C was achieved. There was a concomitant increase in the $T_{re}−T_{sk}$ gradient during CAT. The apparent tendency for higher $T_{sk}$ and lower $T_{re}−T_{sk}$ in Ex vs. Heat during the last 60 min of
the cold exposure did not achieve statistical significance.

Heat flow (CAT). HF was higher (P < 0.05) during CAT in Ex vs. Heat (Fig. 3). Also, Iti during CAT was lower (P < 0.05) in Ex compared with Heat (Fig. 3). Individual site heat flow and Iti are presented in Fig. 4. Calf heat flow and Iti demonstrated a significantly (P < 0.05) greater heat flow and lower Iti between Ex and Heat. Hand heat flow also tended (P = 0.06) to be higher in Ex.

M and heat debt (CAT). M did not differ between Ex and Heat at any time throughout CAT. The final M at minute 115 was 146.6 ± 6.5 and 136.1 ± 3.6 W·m⁻² for Ex and Heat, respectively. The relationships (slope and intercept) between Tb and the corresponding increment in M over pre-CAT values (∆M, a measure of shivering thermogenesis) did not differ between trials. Slopes were −33.8 ± 3.0 and −32.7 ± 3.4 W·m⁻²·°C⁻¹ for Ex and Heat, respectively. Intercepts were 34.5 ± 0.2 and 34.3 ± 0.1°C for Ex and Heat, respectively. Cumulative heat debt was not different between Ex (547.5 ± 47.0 W·m⁻²) and Heat (532.9 ± 28.5 W·m⁻²) after 120 min of exposure.

Plasma glucose and NE (CAT). Plasma glucose concentrations were not affected by CAT in either trial, and there were no differences between trials. Glucose concentrations were not affected by CAT in either trial, and there were no differences between trials. Glucose...
values averaged between 4 and 6 mmol/l throughout CAT. Plasma NE concentrations increased from 2.5 to 10–15 nmol/l during cold-air exposure, with no differences between Ex and Heat.

Heart rate (CAT). Heart rate tended (P = 0.06) to be higher from minutes 30–75 (~10 beats/min) in Ex during CAT compared with Heat.

DISCUSSION

This study determined whether exercise predisposes people to experience a greater decline in T_core during subsequent cold exposure. An expected response to exercise, even in temperate climates, is an increased T_core (21). Therefore, to isolate effects of body heat content and temperature changes from other exercise effects (thermoregulatory fatigue, thermoregulatory lag, heat redistribution), control experiments were needed in which initial pre-cold exposure T_core values had been passively elevated to the same value as those measured postexercise. If such controls were not employed, the T_core–T_sk gradient would be greater during cold exposure after exercise and, subsequently, heat loss would be facilitated. In addition, the absolute T_core could not be compared between trials beginning with different initial values. However, it is experimentally difficult to match both T_core and T_sk increases during exercise in air to increases induced by passive heating in air, especially if the durations of the interventions are also desired to be similar. Matching of T_core and T_sk changes during rest (passive heating) and exercise sessions of similar duration are better accomplished by, using water immersion. The exercise intensity (55% V̇O₂peak) was selected to represent moderately strenuous, fatigu- ing activities.

The primary finding from this study was that, when individuals exercised before cold exposure, they cooled faster than when rest preceded cold exposure. However, the data are not consistent with our hypothesis that exercise would lead to thermoregulatory fatigue of the shivering response to cold. We had based that hypothesis on findings from our laboratory (2) and those reported by others (18, 19, 25) suggesting that shivering can become fatigued. In this study, the shivering response to cold was the same regardless of whether exercise preceded the cold exposure. In contrast, HF measurements were higher and, concomitantly, tissue insulation less during cold exposure after exercise. T_sk during cold-air exposure also tended to be higher (0.2–0.5°C) after exercise. Collectively, these observations indicate that, after exercise, greater peripheral heat loss from the skin (thermoregulatory lag and/or heat redistribution) was responsible for the greater cooling rates during cold exposure.

Several factors might explain why peripheral heat loss during cold exposure was greater when preceded by exercise than passive heating. One possibility is that postexercise hyperemia in the leg muscles persists during cold exposure, increasing convective heat transfer from the body's core to the periphery overlying active muscle relative to cold exposure preceded by rest (heat redistribution). The higher heat flow and lower insulation in the calf during cold exposure after exercise, compared with passive heating, are consistent with this explanation. Another possibility is that the prior exercise blunted the drive for vasoconstriction normally elicited in response to cold (thermoregulatory lag). However, cold-induced vasoconstriction is sympathetically mediated, and the NE response to cold, considered reflective of sympathetic nervous activation (7), was the same whether cold exposure was preceded by exercise or passive heating. On the other hand, sensitivity of peripheral arterioles to NE released in response to cold might be diminished after exercise (12).

Our results contrast with those reported by Kenny et al. (13), who found that the threshold for vasoconstriction was elevated after exercise. They suggested that exercise would result in the retention of heat during subsequent recovery in a cold environment (13). However, our subjects exercised for 1 h in water, whereas those studied by Kenny et al. only completed a short exercise bout (15 min), and thus our subjects may have been more fatigued. In addition, Kenny et al. did not control for differences in initial T_core before cold exposure, but we matched initial T_core values before cold exposure between our trials. Finally, our volunteers were subjected to a whole-body cold-air exposure at a
constant temperature compared with the water-perfused suit that Kenny et al. used. Thus methodological differences probably account for discrepant observations in our study and that of Kenny et al.

Although we observed a lower T_core when cold exposure followed exercise as well as significantly higher peripheral heat flows and a tendency for higher M˙, compared with cold exposure after passive heating, we found no statistical difference in cumulative heat debt, measured by partitional calorimetry. There was a tendency for an increased M during cold exposure after exercise compared with exposures after passive heating, which probably offset the increased heat flow. Therefore, the greater fall in T_core during cold exposure after exercise may reflect a redistribution of body heat content (14, 15) from the core to the periphery because of a higher peripheral blood flow during, and for some time after, exercise (11).

The absence of an exercise effect on shivering thermogenesis suggests that this response to cold is not easily fatiguable. We observed no difference in the T_re vs. ΔM relationship between trials, suggesting that the differences in T_re between trials were not due to a change in central control of shivering thermogenesis. Perhaps exercise intensity and duration were not sufficient to fatigue the shivering mechanism, which is a relatively low-intensity activity (30), at least compared with exercise. In Pugh’s (18) case report of the Four Inns Walk, the participants were exercising up to 20 h in cold-wet conditions. Similarly, the subject in Thompson and Hayward’s study (25) who developed shivering fatigue was exercising for 4 h in severe cold-wet conditions. Another possibility is that shivering impairments observed in these earlier studies may not reflect fatigue, but rather hypoglycemia, which is known to impair shivering (9, 17). Plasma glucose levels were not measured in those previous studies (18, 25). In our study, plasma glucose concentrations remained normal throughout cold exposure.

A possible limitation to extrapolating our results to nonimmersed exercise relates to the potential effects of immersion, especially immersion-associated alterations in hormonal responses to exercise compared with exercise in air. However, during cycle exercise at ~60% V\textsubscript{O2peak}, performed in water up to the neck, there was no difference in catecholamine responses compared with cycling in air at the same intensity (4). Another study (22) also demonstrated that plasma osmolality, which is known to affect central temperature regulation (23), was not different during exercise at 60% V\textsubscript{O2peak} in water and air. In fact, hormonal responses to exercise have not been found to differ between air and immersion (22), except that plasma renin activity was lower and plasma atrial natriuretic peptide was higher during water exercise vs. air exercise at 60% V\textsubscript{O2peak}. However, there is no known influence of renin and atrial natriuretic peptide on hypothalamic neurons regulating thermoregulatory responses to cold. Studies comparing thermoregulatory responses to cold subsequent to exercise in air are warranted to confirm our findings. However, it seems reasonable to conclude that our data indicate that, after exercise, the ability to maintain thermal balance in the cold may be compromised.

This study was the first to examine the possibility that acute exercise performed before whole-body cold-air exposure impairs the ability to maintain thermal balance, as others have speculated. Our findings demonstrated that exercise before cold-air exposure may lead to a greater fall in T_core due to reduced insulation and increased heat loss and a redistribution of heat from the core to the periphery. The data also suggest that an exercise-related factor (heat redistribution) led to the greater fall in T_core and not the rise in T_core that accompanies exercise. These findings may also have potential implications for people who exercise hard and are then exposed to cold stress, or people who exercise hard outdoors in the cold and then stop, but do not return indoors immediately.

The authors thank the volunteers whose participation made this study possible. The expert technical assistance of Laurie Blanchard, Deb Kinsman, and Michelle Landry is gratefully acknowledged. Special thanks to Dr. Jiri Zameknic at the Defence and Civil Institute of Environmental Medicine, North York, Ontario, Canada, for measuring plasma norepinephrine.

The views, opinions and/or findings in this report are those of the authors and should not be construed as official Department of the Army position, policy, or decision unless so designated by other official documentation. Human subjects participated in these studies after giving their free and informed voluntary consent. Investigators adhered to AR 70-25 and USMRDC Regulation 70-25 on Use of Volunteers in Research.

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Received 26 October 1998; accepted in final form 25 March 1999.

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