Thermoregulatory responses to cold water at different times of day

JOHN W. CASTELLANI, ANDREW J. YOUNG, JAMES E. KAIN, AND MICHAEL N. SAWKA

Thermal and Mountain Medicine Division, US Army Research Institute of Environmental Medicine, Natick, Massachusetts 01760-5007

Castellani, John W., Andrew J. Young, James E. Kain, and Michael N. Sawka. Thermoregulatory responses to cold water at different times of day. J. Appl. Physiol. 87(1): 243–246, 1999.—This study examined how time of day affects thermoregulation during cold-water immersion (CWI). It was hypothesized that the shivering and vasoconstrictor responses to CWI would differ at 0700 vs. 1500 because of lower initial core temperatures (T\text{core}) at 0700. Nine men were immersed (20°C, 2 h) at 0700 and 1500 on 2 days. No differences (P > 0.05) between times were observed for metabolic heat production (M\dot{}), heat flow (250 W m\(^{-2}\)), mean skin temperature (T\text{sk}, 21°C), and the mean body temperature-change in M (\(\Delta M\)) relationship. Rectal temperature (T\text{re}) was higher (P < 0.05) before (\(\Delta = 0.4°C\)) and throughout CWI during 1500. The change in T\text{re} was greater (P < 0.05) at 1500 (−1.4°C) vs. 0700 (−1.2°C), likely because of the higher T\text{re}−T\text{sk} gradient (0.3°C) at 1500. These data indicate that shivering and vasoconstriction are not affected by time of day. These observations raise the possibility that CWI may increase the risk of hypothermia in the early morning because of a lower initial T\text{core}.


circadian rhythm; immersion; norepinephrine; shivering; vasoconstriction

RESTING CORE TEMPERATURES (T\text{core}) vary throughout the day, according to an intrinsic circadian rhythm (13). Typically, T\text{core} achieves its nadir in the early morning and then rises to a peak in the late afternoon. Thermoregulatory responses to exercise and heat stress (e.g., T\text{core} threshold for the initiation of sweating and forearm blood flow) also vary over the day (16, 19). These threshold changes closely parallel the change in resting T\text{core}. Whether human thermoregulatory responses to cold stress exhibit similar rhythmicity has not been documented. Information regarding a possible circadian pattern to thermoregulatory responses to cold stress has important implications for experimental designs and perhaps predicting susceptibility to cold injury.

This study examined whether shivering thermogenesis or vasoconstriction during cold-water immersion differs between morning and afternoon exposure. It was hypothesized that the shivering response to cold exposure would vary with time of day, such that, with the circadian rise in T\text{core}, the onset of shivering would occur at a higher body temperature. We also hypothesized that the vasomotor responses governing peripheral heat loss during cold exposure would also exhibit a “time-of-day effect,” although we could not predict the direction of that effect. Experimental findings reported here may support a shift in either direction. On the one hand, plasma norepinephrine (NE) has been shown to be higher in the afternoon vs. morning (12), so peripheral heat losses may also be less in the afternoon because of greater sympathetically mediated peripheral vasoconstriction. On the other hand, radiative and convective heat loss (via an increase in resting forearm blood flow) is greater in the afternoon vs. morning (9, 15), which would lead to greater heat loss.

METHODS

Subjects. Nine men participated in this study after being fully briefed on the risks and giving informed consent. Physical characteristics were the following: age, 23.8 ± 1.1 (SE) yr; height, 178.3 ± 2.8 cm; mass, 77.8 ± 2.9 kg; body surface area, 1.95 ± 0.05 m\(^{2}\); peak oxygen uptake (V\dot{}O\text{peak}), 50.2 ± 1.6 ml·kg\(^{-1}\)·min\(^{-1}\); body fat, 14.0 ± 1.2%; and skinfold thickness, 2.8 ± 0.5 mm. Subjects had no history of cardiovascular or metabolic disease or prior cold injuries. All procedures were approved by the appropriate Institutional Review Board.

Preliminary testing. Body density was determined from underwater weighing with percent fat calculated according to Siri (11). Mean skinfold thickness was calculated from 10 sites according to Allen et al. (1). Two weeks before beginning the experimental protocol, all subjects completed an incremental-effort cycle ergometer test to exhaustion for determination of V\dot{}O\text{peak}. Briefly, each subject pedaled (60 rpm) at a resistance of 60 W for 2 min. The resistance was increased 30 W every 2 min thereafter until the subject reached exhaustion.

Experimental design. Subjects reported to the laboratory 1 h before the experiment, and instrumentation was affixed. They then sat quietly for 15 min on a platform suspended above the water (0700 ambient temperature, 24.3 ± 0.2°C; 1500 ambient temperature, 24.7 ± 0.4°C) while staff obtained preimmersion measurements of body temperature and metabolic heat production (M). After these baseline measurements were made, subjects were quickly lowered into 20°C water to shoulder level where they remained immersed for 120 min. Tests were terminated if rectal temperature (T\text{re}) reached 35°C (7). Each subject completed two immersions on separate days. One experiment began at 0700 and the other at 1500. These trials were separated by at least 1 wk, and the order was randomized. Subjects refrained from using alcohol, medications, or tobacco products and did not exercise for 12 h before testing. Approximately 1−1.5 h before the 0700 trial, subjects consumed a light breakfast (piece of fruit, juice). Before the 1500 experiment, subjects ate lunch (sandwich, soda) 3−3.5 h before immersion and were involved in only light activities (i.e., desk work). The rationale for these feedings was to prevent hypoglycemia.

Measurements. T\text{re} was measured by a thermistor inserted 10 cm past the anal sphincter. Mean weighted skin temperature (T\text{sk}, °C) and mean weighted heat flow (HF, W m\(^{-2}\)) were
measured by using an integrated disk system (Concept Engineering heat flow sensor with integral linear thermistor, Old Saybrook, CT). \( T_{sk} \) was calculated as follows: \( T_{sk} = 0.06T_{foot} + 0.17T_{calf} + 0.14T_{medial\ thigh} + 0.14T_{lateral\ thigh} + 0.14T_{chest} + 0.07T_{triceps} + 0.07T_{forearm} + 0.14T_{subscapular} + 0.07T_{hand} \) (18). Calculation of \( HF \) (W·m\(^{-2}\)) was as follows: \( HF = 0.26H_{subscapular} + 0.14H_{forearm} + 0.08H_{triceps} + 0.22H_{calf} + 0.28H_{medial\ thigh} \) (18), where \( H \) is heat flow. Mean body temperature (\( T_b \)) was calculated as follows: \( T_b = 0.8T_{re} + 0.2T_{sk} \); during immersion, \( T_b = 0.67T_{re} + 0.53T_{sk} \) (5). Temperature and heat flow measurements were continuously recorded by using a computer-automated data-acquisition system.

Oxygen uptake (\( V_{O2} \)) was measured by using an automated metabolic analysis system (model 2900, Sensormedics, Yorba Linda, CA) before and after 90 min of immersion via an indwelling venous catheter (18 gauge) placed in a superficial forearm vein. Aliquots were centrifuged at 4°C to separate the plasma. Plasma NE was determined (8) in duplicate via high-performance liquid chromatography with electrochemical detection (model 460, Waters). Plasma samples were frozen at \(-40\)°C before analysis.

Statistical analyses. A two-way repeated-measures analysis of variance (trial \( \times \) time) was used to determine whether significant differences existed between the 0700 and 1500 trials. When significant \( F \)-ratios were detected, paired comparisons were analyzed post hoc by using the Newman-Keuls test. Statistical significance was accepted at \( P < 0.05 \).

RESULTS

Immersion time. The immersion time for seven subjects was the same (120 min) for both trials. However, the immersion times for two of the subjects were lower at 0700 (56.6 and 66.5 min) vs. 1500 (120 min for both subjects) because they reached the \( T_{re} \) safety limit of 35°C.

Temperature and heat flow responses. \( T_{re} \) was significantly higher (\( P < 0.05 \)) at 1500 compared with 0700 before and throughout the 120-min immersion (Fig. 1). The change in \( T_{re} \) was greater at 1500 (\( P < 0.05 \)) from minute 60 through minute 120 (Fig. 1). \( T_{sk} \) was higher (\( P < 0.05 \)) at minute 0 in the 1500 trial (\( \Delta = 0.4\)°C), but, after immersion, no differences were observed between trials (Fig. 2). The gradient between \( T_{re} \) and \( T_{sk} \) was higher (\( P < 0.05 \)) during the 1500 trial (Fig. 2). During immersion, \( HF \) (W·m\(^{-2}\)) did not differ (\( P > 0.05 \)) between trials (Fig. 3).

M. M was similar between trials throughout the 120-min immersion (Fig. 3). Analysis of the \( T_{re} \)-\( \Delta M \) relationship demonstrated no differences in either the slope (\( -64.0 \pm 4.2 \) vs. \(-68.0 \pm 4.9 \)) or intercept (\( 32.7 \pm 0.2 \) vs. \( 32.9 \pm 0.2 \)) for 0700 and 1500, respectively.

Plasma NE. There were no significant differences in plasma NE concentration at minute 0 (290 ± 60 vs. 342 ± 87 pg/ml) and minute 90 (1,560 ± 448 vs. 1,564 ± 361 pg/ml) for 0700 and 1500, respectively.

DISCUSSION

Human heat stress studies have demonstrated a circadian shift in the onset of thermoregulatory responses, similar to the circadian shift in \( T_{core} \) (14, 16, 19). However, this study is the first to focus on whether thermoregulatory responses to cold-water immersion are affected by time of day. Overall, the data indicate little difference in thermoregulatory responses to acute cold exposure as a function of time of day.

This study found that \( T_{re} \) remained higher throughout the immersion period at 1500 compared with 0700. This is because the higher initial \( T_{core} \) at 1500 vs. 0700, a well-documented circadian variation (14, 16, 19). Interestingly, the change in \( T_{re} \) during cold-water immersion was greater at 1500. This is most likely because of the higher \( T_{re} \)-\( T_{sk} \) gradient at 1500, promoting a greater transfer of heat from the core to the periphery. Whether the faster decrease in \( T_{re} \) at 1500 would be sustained after the \( T_{re} \) had reached the same temperature as at the end of the immersion begun at 0700 cannot be determined from these data.
We observed no time-of-day effect on the two principal physiological responses elicited in humans exposed to cold. Absolute $\dot{M}$ was similar, as were the slope and intercept of the relationship of $T_b$ to $\Delta M$. This suggests that time of day has no effect on the onset or sensitivity of shivering thermogenesis during cold-water immersion. Time of day also appears to have no effect on cold-induced vasoconstriction. Skin temperatures and heat flow during cold-water immersion were the same at 0700 and 1500, suggesting no effect on peripheral heat loss. Plasma NE, a marker of sympathetic nervous system activation (3), was also the same between the two time points. Together, these observations suggest no effect on the sympathetically mediated vasoconstrictor response to cold. This observation is consistent with those reported from the only other investigation of circadian variations in thermoregulatory effector responses to cooling (17). Tayefeh et al. (17) observed no difference in the cooling-induced vasoconstriction threshold between 0700 and 1600, although a shift was apparent by 0300. However, it is unclear to what extent that shift resulted from sleep deprivation as opposed to circadian rhythm effects (17). We can only speculate what may occur were we to perform our experiments at 0300.

One potential reason differences in thermoregulatory responses to cold exposure were not observed between 0700 and 1500 was that the cold stimulus used was severe. Cold-water immersion elicits maximal cutaneous vasoconstriction and high levels of shivering thermogenesis. Thus it was possible that cold-water immersion elicited maximal responses during both trials, and thus time-of-day differences in thermoregulatory responsiveness may have been masked. Therefore, further studies using a less-severe stimulus (cold air) that does not cause maximal constriction and shivering may be warranted.

Two subjects’ 0700 experiments were terminated early because their $T_{re}$ achieved the safety limit of 35°C. For one subject, there were no differences in $M$, peripheral heat loss, or cooling rate. It appears that $T_{re}$ reached the safety limit earlier in the 0700 trial simply because of the 0.6°C lower initial $T_{re}$ compared with 1500. Therefore, this subject’s overall responses to cold at different times are similar to those of the other seven volunteers. The data from the other volunteer whose test was terminated early suggest that a blunted shivering response might have been responsible for the faster drop in $T_{core}$. The slope of this subject’s $T_b-\Delta M$ relationship was less, and therefore $M$ lower, at any given $T_b$ at 0700 vs. 1500; there was no difference in the intercept (onset) of shivering. The mechanism by which the thermogenic response to cold might have been blunted at 0700 in this individual is not apparent. Factors known to impair shivering during cold expo-
Individuals typically have a lower resting T core in the circadian rhythm compared to their more active state, enough to cause Tcore to decrease, dangerously low in the morning; thus, when morning cold exposures are severe enough to cause Tcore to decrease, dangerously low Tcore levels may be achieved sooner than when cold exposure takes place in the afternoon, when resting Tcore is elevated.

The authors thank the volunteers whose participation made this study possible. The expert technical assistance of Douglas Zamistil, Laurie Blanchard, Amy Rouse, Michelle Mayo, Kristine Bailey, and Dean Rios is gratefully acknowledged.

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 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.

Address for reprint requests and other correspondence: J. W. Castellani, Thermal and Mountain Medicine Division, US Army Research Institute of Environmental Medicine, 15 Kansas St., Natick, MA 01760-5007 (E-mail: john.castellani@na.amedd.army.mil).

Received 16 November 1998; accepted in final form 15 March 1999.

REFERENCES