Hyperhydration: tolerance and cardiovascular effects during uncompensable exercise-heat stress

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Latzka, William A., Michael N. Sawka, Scott J. Montain, Gary S. Skrinar, Roger A. Fielding, Ralph P. Matott, and Kent B. Pandolf. Hyperhydration: tolerance and cardiovascular effects during uncompensable exercise-heat stress. J. Appl. Physiol. 84(6): 1858–1864, 1998.—This study examined the efficacy of glycerol and water hyperhydration (1 h before exercise) on tolerance and cardiovascular strain during uncompensable exercise-heat stress. The approach was to determine whether 1-h preexercise hyperhydration (29.1 ml H2O/kg lean body mass with or without 1.2 g/kg lean body mass of glycerol) provided a physiological advantage over euhydration. Eight heat-acclimated men completed three trials (control euhydration before exercise, and glycerol and water hyperhydrations) consisting of treadmill exercise-heat stress (ratio of evaporative heat loss required to maximal capacity of climate = 4:1). During exercise (~55% maximal O2 uptake), there was no difference between glycerol and water hyperhydration methods for increasing (P < 0.05) total body water. Glycerol hyperhydration endurancetime (33.8 ± 3.0 min) was longer (P < 0.05) than for control (29.5 ± 3.5 min), and was not different (P > 0.05) from that of water hyperhydration (31.3 ± 3.1 min). Hyperhydration did not alter (P > 0.05) core temperature, whole body sweating rate, cardiac output, blood pressure, total peripheral resistance, or core temperature tolerance. Exhaustion from heat strain occurred at similar core and skin temperatures and heart rates in each trial. Symptoms at exhaustion included syncope and ataxia, fatigue, dyspnea, and muscle cramps (n = 11, 10, 2, and 1 cases, respectively). We conclude that 1-h preexercise glycerol hyperhydration provides no meaningful physiological advantage over water hyperhydration and that hyperhydration per se only provides the advantage (over euhydration) of delaying hypohydration during uncompensable exercise-heat stress.

MATERIALS AND METHODS

Subjects and preliminary measurements. Subjects were fully informed of all aspects of the study and signed a statement of informed consent. This study was approved by the appropriate institutional review boards. All subjects were medically cleared before any testing. Subjects were required to be healthy with no history of heat injuries, cardiovascular disease, or renal abnormalities. Eight men completed three exercise-heat-stress tests (HSTs). The subject’s average age was 23 ± 6 (SD) yr (range 19–36 yr), body mass was 76 ± 15 kg (range 56–100 kg), lean body mass (LBM) was 63 ± 9 kg (range 53–73 kg), maximal O2 uptake (V̇O2max) was 56 ± 8 ml·kg–1·min–1 (range 42–69 ml·kg–1·min–1), and TBW was 46.4 ± 6.4 liters (range 38–54 liters).

Preliminary measurements (V̇O2max, hydrostatic weighing, and TBW measurements) were made before any HSTs by be of limited thermal benefit. UCHS is associated with heat exhaustion occurring at relatively low core temperatures (19, 33). This is likely because the displacement of blood to the skin (via increased cutaneous dilation and compliance) results in an inability to sustain adequate cardiac output, blood pressure, and perfusion in the brain, leading to syncope (28, 31).

During UCHS conditions, a hyperhydration method that defends or expands blood volume may theoretically reduce cardiovascular strain and improve exercise-heat performance. Ideally, hyperhydration should increase total body water (TBW) for extended periods (at least several hours), and the increased TBW should allow plasma volume to be maintained or expanded. Glycerol hyperhydration has been shown to increase TBW, and the extra water has been retained for extended periods at rest (6, 24). We recently reported (15) similar increases in TBW for both glycerol and water hyperhydration approaches during compensable exercise-heat stress in which neither glycerol nor water hyperhydration modified the core temperature or sweating response, compared with the sustained euhydration trials. It is to be determined whether hyperhydration provides an advantage during UCHS when cardiovascular strain, and not thermoregulatory factors, limits performance.

Previous research has not evaluated the effects of hyperhydration during UCHS. The purpose of this study was to determine the efficacy of two hyperhydration strategies on tolerance during UCHS. Glycerol hyperhydration was compared with water hyperhydration and euhydration without fluid replacement during UCHS. It was hypothesized that preexercise hyperhydration would improve physiological tolerance (i.e., greater core temperature at exhaustion) compared with the preexercise euhydration condition during UCHS.
using procedures previously reported (15). Before HSTs, the subjects were heat acclimated to the exercise conditions. Nude body mass was measured for 2 wk in the morning before breakfast and after the subject voided. These body masses were used to establish baseline body weights, which represent euhydration in the control trial. TBW was measured at the end of heat acclimation by using the deuterium-labeled-water-dilution technique (7). The error of the method, including purification and analysis, was calculated as $\pm 0.1$ g/l (7). The TBW measurement was used to calculate the change in TBW during the HSTs. Before HSTs, subjects practiced walking at the selected treadmill grade and speed and the selected cardiac output procedures (CO2 rebreathing) to become familiar with methods used during the HSTs. VO2 is measured in the 1st wk of acclimation by using a progressive intensity-continuous effort treadmill protocol (13). Each subject completed five compensable HSTs (15) before UCHS trials were initiated.

Subjects were heat acclimated by walking at $\sim 45\%$ VO2max on a treadmill for two 50-min bouts, spaced by a 10-min rest period, for 6–10 days in a hot dry climate (ambient temperature $= 35^\circ C$, relative humidity $= 45\%$, dew-point temperature $= 25^\circ C$, air velocity $= 1$ m/s). Heat acclimation was determined when nonsignificant differences were observed for final exercise core temperatures and heart rates on 2 continuous days of acclimation. During rest and exercise in the heat, the subjects were encouraged to drink water or a commercial electrolyte beverage. Heart rate and rectal temperatures (Tr) were measured during the heat acclimation sessions. Body masses (nude and clothed) were obtained before and after exercise in the heat. Subjects were asked to participate in additional heat acclimation sessions on nonest days if they had gone more than 2 consecutive days without an exercise-heat exposure.

Hydration procedures. Hyperhydration began immediately after venipuncture for the initial plasma osmolality measurement. No experiment was performed unless subjects were euhydrated before HSTs were initiated. The euhydration condition criteria required an initial plasma osmolality of $< 286$ mosmol/kgH2O and body weight within $\pm 0.5$ kg of the baseline value. In the hyperhydration trials, subjects first drank 3.9 ml/kg LBM of the experimental solution (i.e., either glycerol or water solution sweetened with aspartame). The experimental solution administration was double blind, and the water and glycerol solutions were of similar sweetness (aspartame), color, flavor, and temperature ($10^\circ C$) to mask the taste of glycerol. The glycerol solution contained 1.2 g glycerol/kg LBM and was adequate for human consumption. After ingestion of the experimental solution, the subject drank a large volume (25.2 ml/kg LBM) of water (temperature $= 36^\circ C$). The total volume of fluid consumed in a 30-min period was 29.1 ml/kg LBM. This hyperhydration method is selected cardiac output procedures (CO2 rebreathing) to be measured in the 1st wk of acclimation by using the deuterium-labeled-water-dilution technique (7). The error of the method, including purification and analysis, was calculated as $\pm 0.1$ g/l (7).

RESULTS

Before each trial, predrink body masses were similar (P > 0.05), i.e., 76.0 $\pm$ 5.4, 76.1 $\pm$ 5.2, and 75.9 $\pm$ 5.2 kg for Con, G, and W trials, respectively. The predrink plasma osmolalities were 283 $\pm$ 2, 285 $\pm$ 2, and 284 $\pm$ 1 mosmol/kgH2O for Con, G, and W, respectively, and were not different (P > 0.05). The average volume of fluid consumed before each hyperhydration trial was $1.84$ $\pm$ 0.09 liters (adjusted according to body weight); no fluid was given during HSTs or before the Con trial. Two of the eight subjects on one occasion became nauseated after drinking the glycerol solution; therefore, the trial was aborted and repeated on another day.

Figure 1 presents the change in TBW during each trial. For G and W, TBW increased (P < 0.05) by $1.50$ $\pm$ 0.15
and 1.42 ± 0.11 liters, respectively, at 60 min after drinking, with no difference (P > 0.05) between trials. In all trials, TBW decreased (P < 0.05) during exercise to 1.35 ± 0.08, 1.63 ± 0.12, and 1.64 ± 0.08 liters for Con, G, and W, respectively. There were no differences (P > 0.05) in TBW between G and W before or during exercise, but TBW was greater (P < 0.05) in G and W trials than in the Con trial. Total urine volume was less (P < 0.05) in the Con trial than in either G or W trials, and urine volumes were not different (P > 0.05) between G and W trials. The total urine volumes were 0.11 ± 0.04, 0.34 ± 0.07, and 0.42 ± 0.07 liter for Con, G, and W trials, respectively.

Metabolic and exercise performance. The time to exhaustion was not different (P > 0.05) between G and W trials; however, time to exhaustion was greater (P < 0.05) in the G trial than in the Con trial. Endurance times were 29.9 ± 1.2, 33.8 ± 1.1, and 31.3 ± 1.1 min for Con, G, and W trials, respectively. As shown in Fig. 2, seven of the eight subjects had greater endurance in the G trial than in the Con trial, and three of the eight subjects had greater endurance in the W trial than in the Con trial. Metabolic rates were not different (P > 0.05) among trials or over time. The average metabolic rates were 414 ± 8, 413 ± 9, and 418 ± 7 W/m² for Con, G, and W trials, respectively, which corresponded to relative O₂ uptakes of 54, 54, and 55% of VO₂max.

Figure 3 presents heart rate, cardiac output, TPR, and MAP during exercise for each trial. Heart rates were not different (P > 0.05) at rest among trials and increased (P < 0.05) over time during exercise. At 10 min of exercise, heart rates were greater (P < 0.05) in the Con trial than in G and W trials (165 ± 3 vs. 156 ± 2 and 156 ± 4 beats/min, respectively); however, this difference diminished as exercise progressed. Heart rate responses during exercise were similar between G and W trials. Heart rates at exhaustion from heat strain were not different (P > 0.05) among trials. Final heart rates were 187 ± 4, 187 ± 3, and 185 ± 4 beats/min for Con, G, and W trials, respectively.

Cardiac output values were not different (P > 0.05) among trials. The average cardiac output values during exercise were 17.1 ± 1.0, 17.9 ± 0.9, and 17.1 ± 0.9 l/min for Con, G, and W trials, respectively. Cardiac output increased (P < 0.05) from 10–20 min of exercise, and average values were 18.1 ± 0.9 and 17.2 ± 0.9 l/min, respectively. MAP responses during rest or exercise were not different (P > 0.05) among trials. MAP increased (P < 0.05) over time during exercise. Final MAP values were 98 ± 5, 92 ± 3, and 96 ± 6 mmHg for Con, G, and W trials, respectively. TPRs were not different among trials. Final mean TPR values were 5.8 ± 0.3, 5.5 ± 0.3, and 5.8 ± 0.2 peripheral resistance units for Con, G, and W trials, respectively.

Body temperature and sweating. Figure 4 presents the Tₑ responses during exercise for each trial. Tₑ values were not different (P > 0.05) among trials either before or during exercise. The Tₑ responses were similar (P > 0.05) among trials both before and during exercise. The Tₛₑ values were lower (P < 0.05) at 5 min of exercise compared with before exercise, and, from 5 min of exercise, the Tₛₑ values continued to increase (P < 0.05) over time. Final exercise Tₛₑ values for Con, G, and W trials were 37.4 ± 0.2, 37.6 ± 0.2, and 37.1 ± 0.4°C, respectively. The Tₑ−Tₛₑ gradient values were similar (P > 0.05) among trials and, at exhaustion, were 1.3 ± 0.3, 1.2 ± 0.3, and 1.3 ± 0.3°C for Con, G, and W trials, respectively. Tₑ values were not different (P > 0.05) among trials and increased (P < 0.05) over time during exercise. Final exercise Tₑ values were 38.5 ± 0.1, 38.6 ± 0.2, and 38.4 ± 0.2°C for Con, G, and W trials, respectively. Whole body sweating rates were not different (P > 0.05) among trials. The sweating rates were 608 ± 42, 698 ± 56, and 683 ± 32 g·m⁻²·h⁻¹ for Con, G, and W trials, respectively.

Physiological tolerance to heat strain. Table 1 presents the physiological responses: Tₑ responses during exercise for each trial. Tₑ values were not different (P > 0.05) among trials either before or during exercise. The Tₑ responses were similar (P > 0.05) among trials both before and during exercise. The Tₛₑ responses were lower (P < 0.05) at 5 min of exercise compared with before exercise, and, from 5 min of exercise, the Tₛₑ values continued to increase (P < 0.05) over time. Final exercise Tₛₑ values for Con, G, and W trials were 37.4 ± 0.2, 37.6 ± 0.2, and 37.1 ± 0.4°C, respectively. The Tₑ−Tₛₑ gradient values were similar (P > 0.05) among trials and, at exhaustion, were 1.3 ± 0.3, 1.2 ± 0.3, and 1.3 ± 0.3°C for Con, G, and W trials, respectively. Tₑ values were not different (P > 0.05) among trials and increased (P < 0.05) over time during exercise. Final exercise Tₑ values were 38.5 ± 0.1, 38.6 ± 0.2, and 38.4 ± 0.2°C for Con, G, and W trials, respectively. Whole body sweating rates were not different (P > 0.05) among trials. The sweating rates were 608 ± 42, 698 ± 56, and 683 ± 32 g·m⁻²·h⁻¹ for Con, G, and W trials, respectively.
The change in $T_{re}$ from rest to exhaustion ranged from 0.9 to 2.4°C. Final $T_{sk}$ ranged from 35.8 to 38.7°C. Final heart rate ranged from 162 to 204 beats/min, and heart rate values at exhaustion represented 95, 95, and 94% of maximal heart rate for Con, G, and W trials, respectively. Four out of eight subjects had greater core temperature tolerance in the G trial than in the Con trial, and three out of eight subjects had greater core temperature tolerance in the W trial than in the Con trial.

Table 2 presents the frequency of symptoms reported at exhaustion relative to final temperature. At exhaustion, the predominant reasons for discontinuing exercise were syncope, ataxia and fatigue. No apparent pattern was observed for frequency of symptoms relative to final $T_{re}$ or to the method of hyperhydration (water vs. glycerol).

**DISCUSSION**

This study examined the efficacy of two hyperhydration approaches on physiological tolerance and cardiovascular effects during UCHS. A time schedule was used that initiated exercise-heat stress (60 min post-drink) when TBW increases were expected to be greatest and similar for both G and W (6, 15). Therefore, the design should have been able to discriminate any initial hydrational advantage between trials during exercise-heat stress. A design emphasis in this study was to ensure that valid "baseline" conditions were attained. The baseline condition was initial euhydration with subsequent progressive dehydration during exercise. This baseline condition was selected because, during UCHS, people rapidly store heat (14, 19, 33); thus exposure times are short, with few opportunities to
rehydrate. Both methods of hyperhydration were equally effective at increasing TBW. At 30 min post-drink, TBW increased an average of 1.5 and 1.4 liters during G and W trials, respectively. Similar increases in TBW between G and W were reported and addressed in our previous study (15).

This study was the first to examine the efficacy of hyperhydration for UCHS. The data clearly demonstrate that G (compared with W) provides no physiologic or tolerance advantage during UCHS. Compared with W, G did not modify $T_{re}$, skin temperature, whole body sweating, MAP, TPR, cardiac output, heart rate, responses, endurance time, or physiological strain at exhaustion. Compared with control conditions (euhydration with no fluid replacement), hyperhydration demonstrated several minor advantages. G increased endurance time (~4 min) and lowered heart rate (~10 beats/min), whereas W lowered heart rate (~10 beats/min) relative to Con. These advantages probably reflect the adverse effects of dehydration observed in the Con trial. By the end of the Con trial, subjects were hypohydrated by ~1.5 liters (~2% of body weight). Hypohydration of this magnitude will increase physiological strain during compensable exercise-heat stress (18, 22, 34, 35); however, its effects on short-term UCHS are not well studied (33).

Hyperhydration did not alter the physiological strain (core temperature, core temperature increase, heart rate, and skin temperature) tolerated at exhaustion during UCHS. The reasons for discontinuing exercise include syncope and ataxia, fatigue, dyspnea, and muscle cramps ($n = 11, 10, 2, 1$ cases, respectively). Endurance time averaged 32 min and ranged from 24 to 38 min. Exhaustion from heat strain occurred at a mean core temperature of $38.7 \pm 0.2^\circ C$, which compares with values ($38.6 \pm 0.2^\circ C$) reported by Montain et al. (19) for subjects wearing protective clothing for a variety of different exercise intensities and climatic conditions (all UCHS). Sawka et al. (33) reported that exhaustion from heat strain occurred at a $T_{re}$ of $39.1 \pm 0.3^\circ C$ for euhydrated and $38.7 \pm 0.7^\circ C$ for hypohydrated subjects who wore only shorts during UCHS. Nielsen et al. (21) reported that exhaustion from heat strain occurred at similar core temperatures throughout a heat acclimation program.

Our research approach should have had sufficient resolution to observe any meaningful hyperhydration effects on physiological tolerance to UCHS. With the use of the same research approach, physiological tolerance to UCHS was reduced both by hypohydration (33) and by wearing protective clothing (19). Reductions of core temperature tolerance by ~0.4 and ~0.3°C were induced with hypohydration and wearing protective clothing, respectively. Hypohydration was thought to reduce physiological tolerance to heat strain because of decreased blood volume, which results in less cardiac filling (32, 33). Wearing protective clothing was thought to reduce physiological tolerance to heat strain, because higher skin temperatures cause greater blood displacement from the central circulation to the skin, resulting in less cardiac filling (2, 19). Reductions in cardiac filling are believed to be responsible for differences in physiological tolerance to UCHS.

During exercise-heat stress, the simultaneous dilation of cutaneous and skeletal muscle vascular beds is associated with reduced cardiac filling and stroke volume (20, 30, 32). During severe exercise-heat stress, cardiac filling can decline to a level at which cardiac output cannot be maintained by increasing heart rate (27, 30). Rowell et al. (30) reported that, during UCHS, cardiac output was reduced by ~1 l/min compared with control values measured in a temperate climate. In the present study, cardiac output decreased (1 l/min) and stroke volume decreased (14 ml) despite a heart rate

### Table 1. Physiological responses at exhaustion from heat strain for control and hyperhydration trials during uncompensable exercise-heat stress

<table>
<thead>
<tr>
<th>Variable</th>
<th>Control</th>
<th>Glycerol Hyperhydration</th>
<th>Water Hyperhydration</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rectal temperature, °C</td>
<td>$38.7 \pm 0.2$</td>
<td>$38.8 \pm 0.2$</td>
<td>$38.6 \pm 0.2$</td>
</tr>
<tr>
<td>Rectal temperature change, °C</td>
<td>$1.5 \pm 0.2$</td>
<td>$1.7 \pm 0.2$</td>
<td>$1.6 \pm 0.2$</td>
</tr>
<tr>
<td>Mean skin temperature, °C</td>
<td>$37.4 \pm 0.3$</td>
<td>$37.6 \pm 0.3$</td>
<td>$37.1 \pm 0.4$</td>
</tr>
<tr>
<td>Heart rate, beats/min</td>
<td>$187 \pm 4$</td>
<td>$187 \pm 3$</td>
<td>$185 \pm 4$</td>
</tr>
</tbody>
</table>

Values are means ± SE for 8 subjects.

### Table 2. Frequency of symptoms reported at exhaustion relative to final rectal temperature during uncompensable exercise-heat stress

<table>
<thead>
<tr>
<th>Rectal Temperature, °C</th>
<th>Syncope and Ataxia</th>
<th>Fatigue</th>
<th>Dyspnea</th>
<th>Muscle Cramp</th>
</tr>
</thead>
<tbody>
<tr>
<td>38.0</td>
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<td>2</td>
<td>1</td>
<td>1</td>
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<tr>
<td>38.2</td>
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<td>1</td>
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<td>38.4</td>
<td>0</td>
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<td>1</td>
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<td>38.6</td>
<td>0</td>
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<td>38.8</td>
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<td>39.0</td>
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<td>39.2</td>
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<td>39.4</td>
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<td>39.6</td>
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<tr>
<td>39.8</td>
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<tr>
<td>Total</td>
<td>11</td>
<td>10</td>
<td>2</td>
<td>1</td>
</tr>
</tbody>
</table>

$n$, No. of cases.
increase of 14 beats/min. It was anticipated that the increase in TBW associated with hyperhydration would be available to better maintain blood volume during UCHS, thereby allowing improved cardiac filling, cardiac output, and blood pressure preservation (compared with control). It is unclear, however, why hyperhydration mediated slightly lower heart rates with no other cardiovascular or heat-tolerance advantages. The lower heart rate might suggest a larger blood volume in the hyperhydration trials than in the Con trial, but, since the Con group finished exercise hypohydrated, the response may reflect the effect of dehydration and not hyperhydration.

Physiological mechanisms responsible for exhaustion from heat strain include the following: high circulatory strain (29), high body temperatures (3), increased discomfort from hot and/or wet skin (8, 19), reduced skeletal muscle blood flow (4, 9), increased anaerobic muscle metabolism and glycogen utilization (5, 36), and reduced perfusion of the brain, which causes syncope (11). It is possible that combinations of these factors were responsible for exhaustion from heat strain. As discussed above, there was some evidence that hyperhydration reduced circulatory strain with no heat-tolerance advantage. Therefore, cardiovascular strain was not the sole reason for exhaustion. Numerous subjects discontinued exercise at relatively low body temperatures, as also reported by others (14, 19, 33); therefore, the effects of high body temperature are not the sole reason for discontinuing exercise. Exhaustion occurred after a relatively short period for this exercise intensity; hence it is doubtful that differences in glyco- gen depletion were an important factor for exhaustion in this study. The high incidence of reported syncope and ataxia (11 of 24 cases) at exhaustion suggests that the cardiovascular system may have contributed to exhaustion in many instances. The combination of physiological stresses (i.e., high circulatory strain, high body or skin temperatures) could account for a reduction in motivation to continue exercise (3), or, if motivation were maintained, exercise would continue until physiological limitations were incurred (e.g., ataxia and syncope).

In summary, 1-h preexercise hyperhydration with glycerol or water does not alter core temperature, whole body sweating rate, cardiac output, blood pressure, TPR, or core temperature tolerance during UCHS. In addition, glycerol ingestion caused nausea and headaches on several occasions and provided no advantage over water hyperhydration. It is concluded that glycerol hyperhydration provides no meaningful physiological advantage over water hyperhydration during UCHS and that hyperhydration per se only provides the advantage (over euhydration) of delaying hypohydratation during UCHS.

We gratefully acknowledge the technical assistance of Janet Staab and Gerard Shoda, and we thank Drs. Richard R. Gonzalez and Kenneth K. Kranning for expert advice and support. We also express our gratitude to all the test subjects who participated in this study. The views and findings in this report are those of the authors and should not be construed as an official Department of the Army position, policy, or decision unless designated by other official documentation.

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Received 9 April 1997; accepted in final form 29 January 1998.

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