Glycerol hyperhydration: physiological responses during cold-air exposure

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Submitted 14 February 2005; accepted in final form 4 April 2005

O’Brien, Catherine, Beau J. Freund, Andrew J. Young, and Michael N. Sawka. Glycerol hyperhydration: physiological responses during cold-air exposure. J Appl Physiol 99: 515–521, 2005. First published April 7, 2005; doi:10.1152/japplphysiol.00176.2005.—Hypohydration occurs during cold-air exposure (CAE) through combined effects of reduced fluid intake and increased fluid losses. Because hypohydration is associated with reduced physical performance, strategies for maintaining hydration during CAE are important. Glycerol ingestion (GI) can induce hyperhydration in hot and temperate environments, resulting in greater fluid retention compared with water (WI) alone, but it is not effective during cold-water immersion. Water immersion induces a greater natriuresis and diuresis than cold exposure; therefore, whether GI might be effective for hyperhydration during CAE remains unknown. This study examined physiological responses, i.e., thermoregulatory, cardiovascular, renal, vascular fluid, and fluid-regulating hormonal responses, to GI in seven men during 4 h CAE (15°C, 30% relative humidity). Subjects completed three separate, double-blind, and counterbalanced trials including WI (37 ml water/l total body water), GI (37 ml water/l total body water plus 1.5 g glycerol/l total body water), and no fluid. Fluids were ingested 30 min before CAE. Thermoregulatory responses to cold were similar during each trial. Urine flow rates were higher (P < 0.0001) with WI (peak 11.8 ml/min, SD 1.9) than GI (5.0 ml/min, SD 1.8), and fluid retention was greater (P = 0.0001) with GI (34%, SD 7) than WI (18%, SD 5) at the end of CAE. Differences in urine flow rate and fluid retention were the result of a greater free water clearance with WI. These data indicate glycerol can be an effective hyperhydrating agent during CAE.

Methods

Subjects. Seven healthy men participated in this study. The experimental procedures were explained in detail, and written free and informed voluntary consent was obtained from each subject. The study was approved by the US Army Research Institute of Environmental Medicine’s Scientific and Human Use Review Committees, and investigators adhered to Army Regulation 70-25 and US Army Medical Research and Materiel Command Regulation 70-25 on the Use of Volunteers in Research.

Research study design. Subjects reported to the laboratory on six occasions: three for preliminary testing and three for experimental testing. During the initial visit, demographic information, including subjects’ age, height, and body mass, were determined. Body composition was estimated by hydrostatic weighing, and maximal oxygen uptake was determined during continuous treadmill exercise (27). On the second visit, TBW was measured by deuterium oxide (2H₂O) dilution (10). On the third laboratory visit, subjects’ erythrocyte and plasma volume were measured with radiolabeled erythrocytes (⁵¹Cr) and albumin (¹²⁵I), respectively (28). Blood volume was calculated as the sum of the plasma volume and erythrocyte volume.

After preliminary testing, subjects completed three experimental trials, each separated by at least 1 wk. Two trials required hyperhydration, one by ingestion of water alone (WI) and one by ingestion of cognitive and physical performance (26). Previous studies have employed glycerol as a hyperhydration agent because it is rapidly absorbed and osmotically active; therefore, fluid intake with glycerol becomes distributed throughout TBW (25). Hyperhydration with glycerol in temperate (10, 20, 23) and hot (2, 4, 14, 19) environments increases fluid retention compared with water alone; thus it could be a strategy to limit dehydration during cold exposure.

Only one study has evaluated the efficacy of glycerol for fluid retention during cold exposure. Arnall and Goforth (3) reported that glycerol hyperhydration, compared with water alone, was ineffective at reducing fluid losses in divers immersed in a seated posture for 3 h in cold (13°C) water. The hydrostatic pressure associated with water immersion also causes a central fluid shift, but it is accompanied by a natriuresis that does not occur with cold exposure (7, 16). The resulting osmotic diuresis in thermoneutral-water immersion is greater than that observed with cold-air exposure (CAE), and the effect is additive during cold-water immersion (16). Thus their findings may be limited to the model of water immersion, rather than suggesting that glycerol hyperhydration is ineffective in a cold environment.

The present study was conducted to evaluate the effectiveness of hyperhydration with glycerol on fluid losses during CAE. It was hypothesized that hyperhydration with glycerol would improve fluid retention at the end of 4 h of CAE, compared with water alone.

Methods

HYPOTHYDRATION OFTEN OCCURS during cold exposure through combined effects of reduced fluid intake and increased fluid losses (11). Cold exposure is associated with blunted thirst, both during rest and exercise, and this occurs even when subjects are hypohydrated (15). Active individuals may also increase fluid losses through sweating, despite cold temperatures, and cold-induced diuresis (CID) can occur and may amount to 1–3% of body mass (8). This response is self-limiting; i.e., less CID is observed in subjects who are hypohydrated before cold exposure, compared with euhydrated subjects (22). However, greater CID will occur if fluid intake increases in an attempt to replace urinary losses (18).

Hypohydration may impair submaximal physical performance in the cold (24); therefore, hyperhydration strategies that sustain total body water (TBW) could potentially prevent or attenuate dehydration-mediated decreases in performance. This could also be important when cold individuals begin exercising in heavy clothing and subsequently experience heat strain, when hypohydration is known to adversely affect both

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glycerol and water (GI), and during the third trial (NF) no fluid was ingested. The order of the experimental trials was counterbalanced, and testing was conducted between 0700 and 1200.

Subject restrictions. To standardize the subjects' hydration state and reduce the variability in baseline fluid-regulating hormones and renal measurements (9), behavioral restrictions were imposed. On evenings before the preliminary measurements of TBW and erythrocyte and plasma blood volumes, as well as evenings before experimental trials, volunteers reported to a housing facility at 1900 to spend the night under supervision. They were not allowed tobacco products, alcohol, food, or drink for 12 h, nor were they allowed to exercise for 18 h before testing.

Experimental trials. All three experimental trials were performed in the manner described below. After arrival at the laboratory, subjects voided their bladder, were weighed, and then were instrumented with both rectal and skin thermocouples (4 sites: i.e., finger, upper arm, chest, and calf), electrocardiogram (ECG) electrodes (CM-5 configuration), a blood pressure cuff, and a flexible catheter inserted into a forearm vein for blood sampling. Subjects, wearing shorts and sneakers only, then sat for a 45- to 60-min control period (room temperature 20–24°C) during which skin and rectal temperatures were measured every 10 s; heart rate and blood pressure were measured every 10 min; and metabolic rate (model 2900, SensorMedics, Yorba Linda, CA) and cardiac output (CO2 rebreathing) were measured at the end of the control period. At the end of the control period, subjects had an initial blood sample drawn while remaining seated with arm position controlled, and then they stood to empty their bladders.

On the two fluid intake trials, subjects then drank 5.0 ml/l TBW of one of two experimental solutions containing water and a commercially available flavoring. The solutions differed only in that one also contained glycerol (1.5 g/l TBW, Penta Manufacturing, Fairfield, NY). Experimental solutions were similar in sweetness (aspartame), color, and flavor, and they were administered in a double-blind manner. After drinking the experimental solution, subjects drank 32 ml/l TBW distilled water for a total ingested volume (experimental solution plus distilled water) equal to 37 ml/l TBW (equivalent to 1,706 ± 173 ml for our subjects). Drinking was paced over 30 min to ensure similar intake rates among trials and subjects. Immediately after hyperhydration (or no hydration during the control trial), the subjects were moved to an adjacent environmental chamber (15°C, 40% relative humidity) where they sat for 4 h. During cold-air exposure, skin and rectal temperatures were measured every 10 s. Heart rate (obtained from ECG telemetry) and blood pressure (auscultated with sphygmomanometer) were measured after 15 min and at 30-min intervals thereafter so as to be taken at a time when subjects were not distracted by blood collection or metabolic measurements. The heart rate and blood pressure measurements at 15 min and 45 min of each hour were averaged for data analysis. Blood samples were collected every 60 min, and urine was collected immediately after blood sampling. Metabolic rate and cardiac output were measured after 30 min and at 60-min intervals thereafter so as to be off cycle to blood and urine collection.

Analyses for experimental trials. Venous blood samples were drawn by syringe, immediately aliquoted into tubes containing appropriate anticoagulant, and placed on ice. Hematocrit was determined by centrifugation and hemoglobin concentration by hemoglobinometer (Coulter Electronics). Lithium-heparinized plasma was analyzed for Na+, K+, and osmolality, creatinine, and glycerol using methods described above for plasma. Plasma hormone concentrations, i.e., antidiuretic hormone (ADH), atrial natriuretic peptide (ANP), aldosterone (Aldo), and cortisol (Cort), were determined by radioimmunooassay using methods previously described (10). For ADH, the within-assay coefficient of variation (CV) was 15%, with an extraction recovery of 86%. For ANP, within-assay CV was 5.8%, with an extraction recovery of 82%. The within-assay CVs for Aldo and Cort were 8.1 and 4.6%, respectively.

Data analyses. Mean skin temperature was calculated as the average of arm, chest, and calf skin temperatures. The percent changes in blood and plasma volume were calculated from hematocrit and hemoglobin values (6). Actual blood and plasma volume changes (in ml) were calculated by multiplying the percent changes by previously measured blood and plasma volumes. Changes in TBW were calculated by subtracting urine volumes from the volume of fluid ingested. The following excretion volumes and clearance values were calculated using the standard equations listed below: urinary osmolar excretion (UosmV), urinary Na+ excretion (UNaV), urinary K+ excretion (UKV), osmotic clearance (Cosem), free water clearance (CfH2O), and creatinine clearance (CCr), which was used as an estimate of glomerular filtration rate.

\[ Uo_{sm} V = U_{o} (meq/ml) \times V (ml/min) \]
\[ UNa V = U_{Na} (meq/ml) \times V (ml/min) \]
\[ C_{osm} = U_{osm} (mosmol/kg H_2O) \times V (ml/min) / P_{osm} (mosmol/kg H_2O) \]
\[ C_{Cr} = U_{Cr} (mg/ml) \times V (ml/min) / P_{cr} (mg/ml) \]
\[ C_{H_2O} = V (ml/min) - C_{osm} (ml/min) \]

where V is urine flow, Uosm is urinary osmolality, UNa is urinary Na+ concentration, UK is urinary K+ concentration, UCr is urinary creatinine concentration, Posm is plasma osmolality, and Pcr is plasma creatinine concentration.

All data were analyzed by univariate repeated-measures ANOVA using a general linear model procedure (SAS Institute, Cary, NC). When main effects or interactions were significant, Tukey’s honestly significant difference post hoc test was used to determine where significant differences between means existed. Significance was set at the P < 0.05 level. P values presented are for main effects (trial or time), except as noted for trial × time interactions. Data are presented as means (SD).

RESULTS

Preliminary and baseline measurements. Subjects had a mean age of 22 yr (SD 3), height of 175 cm (SD 2), body mass of 78.9 kg (SD 7.8), body fat of 18.8% (SD 6.1), maximal oxygen uptake of 55.7 ml·kg⁻¹·min⁻¹ (SD 7.8), maximal heart rate of 201 beats/min (SD 6), TBW of 46.1 liters (SD 3.9), and resting blood volume of 5.1 liters (SD 0.2). Behavioral restriction placed on subjects before testing were successful in standardizing baseline physiological measurements (i.e., renal function, hydration status, fluid-regulating hormones), because no differences in baseline (“Pre”) measurements were found among trials. No subjects had any negative reactions to glycerol, although they felt “full” after finishing the fluid intake during both WI and GI.

TBW and vascular fluid responses. The increased TBW due to fluid ingestion with GI and WI persisted throughout CAE (Fig. 1). At the end of cold exposure, ~34% (583 ml) of the 1,706 ml (SD 424) ingested fluid was retained with GI, which was significantly greater (P = 0.0001) than the ~18% (304 ml) retained with WI, and both were significantly higher (P = 0.0001) than with NF. Total urine volume postdrink was greater (P = 0.0001) with both GI (1,123 ml, SD 438) and WI.
Blood volume and plasma volume responded similarly across trials, with a decrease \( (P = 0.0001) \) by the end of cold exposure of \(-5\%\) for blood volume and \(-9\%\) for plasma volume, although with GI the decrease in plasma volume began after 60 min in the cold (Fig. 1). Plasma osmolality (trial \( \times \) time interaction, \( P = 0.0001 \)) initially fell by \(-7 \) mosmol/kgH\(_2\)O on cold exposure with WI, increased \(-5 \) mosmol/kgH\(_2\)O with GI, and did not change with NF (Fig. 2). Plasma glycerol concentrations increased (trial \( \times \) time interaction, \( P = 0.0001 \)) with GI to a peak of \(12 \) mmol/l (SD 1) at 60 min of cold exposure and were still elevated after 240 min of cold exposure (4 mmol/l, SD 12). Plasma glycerol did not change with NF or WI (both 0.1 mmol/l, SD 0.1). With both GI and WI, plasma Na\(^+\) concentration fell (trial \( \times \) time interaction, \( P = 0.0003 \)) \(-2\)–\(-3\) meq/l, and this decrease persisted throughout the experimental period with GI (Fig. 2). K\(^+\) concentration increased (\( P = 0.0004 \)) by \(-0.6 \) meq/l by the end of cold exposure, similarly on all trials (Fig. 2). Plasma protein also increased similarly on
all trials ($P = 0.0019$), from 6.9 meq/l (SD 0.4) to 7.5 meq/l (SD 0.5) at the end of cold exposure.

**Renal responses.** Urine flow, free water clearance, and osmotic clearance all increased (trial $\times$ time interactions, $P = 0.0001$) on cold exposure during both hyperhydration trials, but only osmotic clearance increased with NF (Fig. 3). Peak urine flow rate was higher (trial $\times$ time interaction, $P = 0.0001$) with WI (11.8 ml/min, SD 1.9) than with GI (5.0 ml/min, SD 2.3). Urine glycerol increased ($P = 0.0001$) with GI to a peak of 87 mmol/l (SD 55) at 120 min, was still elevated at 19 mmol/l (SD 15) at the end of cold exposure, and did not change with either NF or WI (both 1 mmol/l, SD 2). Urine osmolality decreased and electrolyte excretion increased (trial $\times$ time interaction, $P = 0.0001$) on both hyperhydration trials (Fig. 4). Glomerular filtration rate, estimated by creatinine clearance rate, did not change during cold exposure and was similar among trials (158 ml/min, SD 28).

**Hormonal responses.** The fluid-regulating hormone responses to hyperhydration are shown on Fig. 5. Plasma ADH activity was lower ($P = 0.0001$) during cold exposure, and did not change with either NF or WI (both 1 mmol/l, SD 2). Urine osmolality decreased and electrolyte excretion increased (trial $\times$ time interaction, $P = 0.0001$) on both hyperhydration trials (Fig. 4). Glomerular filtration rate, estimated by creatinine clearance rate, did not change during cold exposure and was similar among trials (158 ml/min, SD 28).

**Fig. 3.** Changes in urine flow, free water clearance, and osmotic clearance over time. Values are means (SD). For time $\times$ trial interactions: *significantly different from No Fluid; †significantly different from Water; $a,b,c,d$ significant difference compared with Pre, 60 min, 120 min, 180 min, respectively $P < 0.0001$.

**Fig. 4.** Changes in urinary sodium, potassium, and osmolality over time. Values are means (SD). ‡Significant main effect for time, Pre vs. 120-240 min cold exposure $P < 0.0001$. For time $\times$ trial interactions: *significant difference from No Fluid; †significant differences from Water; $a,b,c,d$ significant difference compared with Pre, 60 min, 120 min, 180 min, respectively $P < 0.0001$. 

J Appl Physiol • VOL 99 • AUGUST 2005 • www.jap.org
GLYCEROL HYPERHYDRATION DURING COLD-AIR EXPOSURE

Fig. 5. Changes in plasma antidiuretic hormone, atrial natriuretic peptide, and aldosterone over time. Values are means (SD). §Significant main effect for time, compared with subsequent values during cold exposure (P < 0.0001). #Significant main effect for trial, compared with No Fluid (P < 0.0001). For time × trial interactions: *significant difference from no fluid. #significant difference compared with Pre, 60 min, respectively (P < 0.025).

μU/ml) compared with NF (≈0.4 μU/ml). Plasma ANP increased (trial × time interaction, P = 0.0221) by ≈7 pg/ml during cold exposure with WI, and was elevated at the end of cold exposure with GI, but did not change during cold exposure with NF. During both hyperhydration trials, Aldo concentration initially increased (trial × time interaction, P = 0.0003) to a peak at 60 min of cold exposure. The values returned to near baseline levels at 120 min, but with WI Aldo continued to fall, and values at 180 min were lower than Pre. Cort responded similarly in all trials, with an initial fall (P = 0.0242) from 381 nmol/l (SD 102) before cold exposure to 298 nmol/l (SD 99) at 60 min of cold exposure. Cort at the end of cold exposure, 303 nmol/l (SD 97), was not significantly different from Pre.

Thermal, metabolic, cardiovascular, and hemodynamic responses. Both mean skin and finger temperatures fell (P = 0.0001) similarly in all trials, from 31.0°C (SD 0.8) and 31.6°C (SD 1.6), respectively, before cold exposure to 25.2°C (SD 1.0) and 17.9°C (SD 1.4), respectively, at the end of cold exposure. Core temperature increased (P = 0.0001) similarly in all trials, from 36.5°C (SD 0.4) before cold exposure to 36.8°C (SD 0.4) at the end of cold exposure. Metabolic rate increased (P = 0.0001) from 101 W (SD 17) at rest to 135 W (SD 32) after 30 min of cold exposure, and reached 152 W (SD 27) at the end of cold exposure, with no difference among trials. Heart rate (trial × time interaction, P = 0.0127) with GI fell from an initial 64 beats/min (SD 7) to 59 beats/min (SD 6) after 90 min of cold exposure, and with WI fell from 62 beats/min (SD 8) to 57 beats/min (SD 5) after 240 min of cold exposure, but heart rate with NF, 61 beats/min (SD 8), did not change. Stroke volume increased (P = 0.0008) from 69 ml (SD 19) to 87 ml (SD 13) by the end of cold exposure, with no difference among trials. Cardiac output increased (P = 0.0056) after 90 min of cold exposure, from 4.2 l/min (SD 0.9) Pre to 5.1 l/min (SD 0.7) at 210 min, with no difference among trials. Mean arterial pressure was higher (P = 0.0001) throughout cold exposure, compared with the baseline value of 89 mmHg (SD 7). The value at 30 min of cold exposure, 94 mmHg (SD 7), was also higher (P = 0.0001) than the final value during cold exposure of 91 mmHg (SD 8). There was no difference among trials. Total peripheral resistance decreased (P = 0.0106) from a baseline value of 21 mmHg·1−1·min (SD 3) to 18 mmHg·1−1·min (SD 3) at 150 and 210 min of cold exposure, with no difference among trials.

DISCUSSION

This was the first study to evaluate the effectiveness of glycerol as a hyperhydration agent during CAE. Nearly twice as much fluid was retained after 4 h of CAE with glycerol hyperhydration compared with water alone. This study also demonstrates that hyperhydration does not modify cardiovascular or thermoregulatory responses during resting CAE.

A previous study conducted in our laboratory under temperate ambient temperature (22°C) and using similar methodology and the same fluid intake as the present study also demonstrated the effectiveness of glycerol hyperhydration compared with water alone (10). In that study, after 3 h, 60% of fluid was retained after GI, compared with 32% with WI and a net fluid loss due to urine production of ~100 ml during NF. Slightly less fluid was retained in the present study after 3 h of cold exposure: 50% of fluid was retained with GI and 24% with WI, with ~142 ml loss with NF. Euhydrated individuals who increase fluid intake during cold exposure in attempt to offset fluid loss typically experience a greater CID (18), which could account for the reduced fluid retention in the cold, compared with temperate conditions. However, relatively less fluid was lost with GI, indicating that hyperhydration with glycerol was more effective than water alone. CID is minimal during short-term (1–4 h) upright CAE (1, 16) and did not occur during NF in the present study; however, over longer cold exposures.
Hypohydration has been suggested to increase susceptibility to hypothermia and peripheral cold injuries, although recent data from our laboratory do not support this idea (21, 22). The suggestion that decreased plasma volume due to hypohydration could increase risk of peripheral cold injury (12) appears to be unfounded, at least during short-term cold exposure. During cold exposure, plasma volume decreases in euhydration subjects primarily due to fluid shifts from intravascular to interstitial spaces (1, 29), and the magnitude of this fluid shift is similar even in hypohydrated subjects (22). In the present study, hyperhydration had no effect on thermoregulatory responses to cold, and hyperhydration was not effective at preserving plasma volume during cold exposure, although with GI, the rapid appearance of glycerol in the plasma (peak at 60 min in the present study) appears to have delayed the initial fall in plasma volume. Under temperate conditions, plasma volume increased sooner with GI than with water alone (10). In both environments, this delay in plasma volume shift is transient, and thereafter there is no difference in plasma volume between WI and GI, despite greater fluid retention with GI. This suggests that the central fluid shift that occurs with cold exposure influences plasma volume, whereas alterations in hydration status influence the extracellular space. Thus there is no basis for the supposition that moderate hypohydration reduces plasma volume and could thereby increase risk of cold injury.

Similar fluid shifts are observed with postural changes and onset of exercise, with an effective limit in the extent to which hemoconcentration occurs. For example, cycling exercise induces a somewhat larger hemoconcentration than a resting seated posture, yet running causes no further change than the “maximal” hemoconcentration of upright posture (13). Similarly, CID is reduced with upright posture and exercise (18), again suggesting a set point for plasma volume reduction toward which the various stimuli (cold, posture, exercise) additively contribute, but do not exceed. Furthermore, fluid intake during exercise does not alter plasma volume, but instead it preserves the extravascular fluid volume (5). Because glycerol is freely distributed in body water, hyperhydration with GI may better preserve the extravascular fluid volume, accounting for the improved TBW, compared with water alone. This extravascular “reserve” could later be called on during exercise or heat stress, when hydration becomes important to performance and thermoregulation. Whether the degree of hyperhydration achieved in the present study is sufficient to improve physical performance in the cold or thermoregulation during subsequent body warming due to exercise or heat exposure remains to be demonstrated. It should be noted that when rehydration during exercise is possible, the hyperhydrating effects of glycerol are no more beneficial than water alone (17); therefore, the use of glycerol is only pertinent to situations where rehydration is not possible for several hours.

Because glycerol is distributed throughout body water, the most appropriate way to ensure that all subjects receive the same relative glycerol dose is to base the dose on TBW. Previous studies have based the dose on total body mass (2, 4, 14, 19, 23) and lean body mass (3), which avoids the expensive and time-consuming precise measurement of TBW. Data from Riedesel et al. (23) suggest that a dose of 1.5 g/kg body mass has no greater effect on fluid retention than 1.0 g/kg body mass but that a dose higher than 0.5 g/kg body mass is needed for maximum benefit. The dose in the present study, 1.5 g/l TBW, would have been equivalent to a dose of 0.9 g/kg body mass, with a range of 0.8—1.0 g/kg body mass.

This study has several important new findings. First, glycerol hyperhydration is more effective at increasing TBW during CAE than water alone. Second, hyperhydration has no effect on limiting hemoconcentration during CAE, with fluid moving instead into the extravascular spaces. Third, hyperhydration has no effect on thermoregulation during resting CAE. This study supports the previous suggestion (10) that glycerol
induces hyperhydration through renal reabsorption of glycerol and water. Finally, this study provides insight into the hormonal mechanisms of cold-induced diuresis and fluid shifts due to hyperhydration.

ACKNOWLEDGMENTS

The authors are grateful for the technical assistance of Dr. C. Robert Valeri, Spec., Gerald Shoda, Sgt. James McKay, Janet Laird, Jane DeLuca, and Aileen Sato. The authors also recognize the volunteers for the time and effort they devoted to the study.

DISCLOSURES

The opinions or assertions contained herein are the private views of the authors and are not be construed as official or reflecting the views of the U.S. Army or the Department of Defense. The investigators have adhered to the policies for the protection of human subjects as prescribed in Army Regulation 70-25, and the research was conducted in adherence with the provisions of 45 CFR Part 46. Any citations of commercial organizations and trade names in this report do not constitute an official Department of the Army endorsement of approval of the products or services of these organizations.

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