Hypervolemia in Men from Fluid Ingestion at Rest and During Exercise

John E. Greenleaf, Ph.D., Robin Looft-Wilson, M.S., Jeni L. Wishered, B.S., Catherine R. Jackson, Ph.D., Paul P. Fung, M.S., Andrew C. Ertl, Ph.D., Paul R. Barnes, Ph.D., Christopher D. Jensen, M.S., and James H. Whittam, Ph.D.

Background: Plasma osmolality (Osm) is important for controlling and maintaining plasma volume (PV) and body water. The effect of oral rehydration fluids for ameliorating dehydration is well-established; but optimal composition and Osm of fluids for hyperhydrating normally hydrated subjects is less clear. Methods: Six treatments were used without and with oral fluids of varying ion and constituent concentrations for hyperhydrating six previously euhydrated men (30 ± 8 yr, 76.6 ± 16.1 kg, 73 ± 12 ml·kg⁻¹ PV, 40 ± 10 ml·min⁻¹·kg⁻¹ peak V̇O₂ sitting at rest for 90 min (V̇O₂ = 0.39 ± 0.02 L·min⁻¹) and during subsequent 70 min of submaximal exercise (V̇O₂ = 2.08 ± 0.33 L·min⁻¹, 70 ± 7% peak V̇O₂). The hypothesis was that the fluid composition is more important than plasma Osm for increasing PV in euhydrated subjects at rest and maintaining it during exercise. Drink formulation compositions, given at 10 ml·kg⁻¹ body wt, (X = 768 ml), for the sitting period were: Performance 1 (P1; 55 mEq Na⁺, 365 mOsm·kg⁻¹, P2 (97 mEq Na⁺, 791 mOsm·kg⁻¹), P2G (113 mEq Na⁺, 4% glycerol, 1382 mOsm·kg⁻¹), AstroAde (AA; 164 mEq Na⁺, 253 mOsm·kg⁻¹), and O1 and O2 (no drinking). The exercise drink (10 ml·kg⁻¹, 768 ml) was P1 for all treatments except O2 (no drinking): thus, drink designations were: P1/P1, P2/P1, P2G/P1, AA/P1, O1/P1, and O2. Results: PV at rest increased (p < 0.05) by 4.7% with P1 and by 7.9% with AA. Percent change in PV during exercise was +1% to +3% (NS) with AA/P1; +6% to 0% (NS) with P1/P1; +10% to +14% (NS) with P2/P1, P2G/P1, AA/P1, and O1/P1; and -6% to -5% (p < 0.05) with O2. AA, with the lowest Osm of 253 mOsm·kg⁻¹, increased PV at rest (as did P1) and maintained it during exercise, whereas the other drinks with lower Na⁺ and higher Osm of 365–1382 mOsm·kg⁻¹ did not. Conclusion: Drink composition appears to be more important than its Osm for increasing PV at rest and for maintaining it during exercise in previously euhydrated subjects.

Fatigue in People at rest and during exercise involves both physiological and psychological factors (2,3,6). Reduction in vascular fluid volume (hypovolemia) and chronic decrease in total body water (hypohydration) caused by part in voluntary dehydration (12,13,34), defined as the delay in full fluid replacement (eucydration) during and following loss of body fluid, adversely affect exercise performance. Stress from performing mental arithmetic can also cause hypovolemia (31). In addition, plasma volume (PV) and the ionic-osmotic constituent concentration of plasma and cells are also regulatory factors for body thermoregulation which is often compromised with exercise-induced hypovolemia and hypohydration (10,14,22). Thus, people subjected to acute or chronic stress may be somewhat "dehydrated" as well as fatigued.

Research on body fluid distribution and rehydration fluid formulations, stimulated by demands on troops during World War II (1,32), has continued with increasing intensity for military personnel (25) with application for recreational exercisers and competitive athletes (29). Many current rehydration formulations are more concentrated (hypertonic-hyperosmotic) than the normal plasma osmolality (285 mOsm·kgH₂O⁻¹), with more osmoles contributed by carbohydrate than by ionized solute (29). Optimal fluid composition for rapid gastric emptying and transfer through the gastrointestinal system appears to be 20–30 mEq·L⁻¹·Na⁺, 5–10 mEq·L⁻¹·K⁺ (with Cl⁻ as the only anion), and 0.9%–10% carbohydrate, preferably glucose (9). However, measurement of gastric and gastrointestinal emptying of fluid does not necessarily reflect change in plasma or interstitial fluid volumes.

Recent findings from our laboratory have indicated that fluid formulations containing greater concentrations of ionized solute up to 164 mEq·L⁻¹·Na⁺ induced significantly greater levels of hypervolemia in supine, resting, moderately (24 h) dehydrated men, and were also better than water for attenuating the hypovolemia during supine, submaximal, leg ergometer exercise (17). From those findings the present study was designed to (From the Laboratory for Human Environmental Physiology, Gravitational Research Branch, NASA, Ames Research Center, Moffett Field, CA (J. E. Greenleaf, R. Looft-Wilson, J. L. Wishered, C. G. R. Jackson, P. P. Fung, A. C. Ertl); Department of Biological Sciences, San Francisco State University, San Francisco, CA (P. R. Barnes); and Shaker Tech, Inc., San Francisco, CA (C. D. Jensen, J. H. Whittman).

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determine the effect of intermittent ingestion of two previously tested and two new hypertonic fluid formulations containing various osmotic and carbohydrate concentrations on PV in euhydrated men at rest followed by upright (sitting) submaximal ergometer exercise. To test the physiological effect of the hyperhydration; endurance (18), thermoregulatory, and body water balance parameters were measured. The hypothesis was that fluid composition is more important than plasma osmotic concentration for increasing plasma volume in euhydrated subjects at rest and during exercise.

**METHODS**

**Subjects:** There were six men who gave written informed consent for this study which was approved by the Ames Research Center and San Francisco State University Human Research Experiments Review Boards. Their mean (±SD) anthropometric data were: 30 ± 8 yr, 182 ± 7 cm ht, 76.84 ± 16.19 kg wt, 1.98 ± 0.22 m² S.A., 3124 ± 505 ml PV, 5522 ± 923 ml BV, and 2.99 ± 0.45 L·min⁻¹ peak V0₂. The men passed a comprehensive medical examination which included their history, urine and blood analyses, and a treadmill exercise test. All were non-smokers and none took non-prescribed drugs.

**Procedure:** Six treatments for each subject were conducted in a semi-random balanced design at weekly intervals. The experimental protocol consisted of intermittent drinking during 90 min of sitting rest, 15 min to move to the cycle ergometer and to readjust sensors, intermittent drinking during 70 min of upright (sitting) submaximal leg exercise (70 ± SD 7% V0₂ peak), followed by 10 min of sitting recovery (Fig. 1).

The subjects arrived at the laboratory at 0700 h and ate a standard carbohydrate breakfast: 220 ml of reconstituted frozen orange juice and two toasted English muffins with jelly. After breakfast they urinated and inserted a rectal thermistor 16 cm. Dressed in shorts (weighed dry), they were weighed (+5 g) on a digital scale (model 5780, National Controls, Inc., San Carlos, CA). The men then sat in a chair for 90 min while skin probes and sensors (EKG, skin temperature) were attached and a forearm venous catheter (Quik-Cath, Travenol Laboratories, Inc., Deerfield, IL) was inserted. Body weight was measured again and additional urine samples were collected after the rest and exercise periods (Fig. 1).

**Formulations and drinking:** The subjects drank one of four fluid formulations (Table I), each divided into seven portions, during the rest period (Fig. 1). The formulations were designated P1 (Performance 1), P2 (2 × P1 concentration), P2G (P2 +4% glycerol), AA (AstroAde), or 0 (no drinking). Performance 1 is a commercial product of the Shaklee Corporation; AstroAde was designed at Ames Research Center and also packaged by Shaklee. All drinks, in powder form, were mixed just prior to testing. The high salt content of AA, as well as the very sweet taste of P2G, were apparent to the subjects. Glycerol was used for its water-retaining properties. Drink volume was 10 ml·kg⁻¹ body wt for both rest and exercise phases. Performance 1 was consumed during exercise with five treatments; there was no drinking during the sixth. Thus, drink designations for the six treatments in the rest/exercise phases, respectively, were: P1/P1, P2/P1, P2G/P1, AA/P1, 0/P1, and 0/0. One subject exhibited mild gastrointestinal disturbance during these experiments.

**Physiological measurements:** After three familiarization sessions, peak oxygen uptake (V0₂ peak) was measured with the subjects in the upright sitting position on a model 846 cycle ergometer (Quinton Instruments Co., Seattle, WA). The respiratory measurement system used a low-resistance, low-dead-space Rudolph valve (model 2700, Hans Rudolph, Inc., Kansas City, MO), a Tissot tank calibrated electronic spirometer (model S-301 Pneumoscan, K.L. Engineering Co., Sylmar, CA), and a 3-L mixing chamber from which expired gas was sampled at 0.5 L·min⁻¹ and then drawn through and dried by anhydrous calcium sulfate (N.A. Hammond Drierite Co., Xenia, OH) to oxygen and carbon dioxide analyzers (Applied Electrochemistry models S-3A1 and CD-3A, respectively; Ametek, Thermox Instruments Division, Pittsburgh, PA). The analyzers were calibrated with standardized gases (Lloyd-Haldane apparatus). Analog data were processed on-line with an analog-to-digital converter (VISTA system IBM model 17002, Vacumed, Ventura, CA) and transmitted to an IBM (model AT) computer: peak data were the mean of the final four 15-s values. Mean (±SE) peak exercise data were: load = 1550 ± 92 kg·m⁻¹·min⁻¹, RE = 1.25 ± 0.04, V̇O₂peak = 126.00 ± 6.14 L·min⁻¹, HR = 187 ± 7 b·min⁻¹, and V̇CO₂ = 40 ± 4 ml·min⁻¹·kg⁻¹. The submaximal exercise load (817 ± SD 133 kg·m⁻¹·min⁻¹) corresponded to an oxygen uptake of 70 ± SD 7% of the measured peak V̇O₂.

Heart rate was determined with a cardiographometer (model 78203C, Hewlett-Packard, Waltham, MA) via three skin electrodes (Silvon No. 01-3630 Ag/AgCl, NDM, Dayton, OH); two on the anterior shoulders and the third over the fifth intercostal space.

Rectal and skin temperatures were measured with series 400 thermistors (Yellow Springs Instrument Co., Yellow Springs, OH). Skin thermistors, attached with holders that permitted free movement of air (20), were located
TABLE I. FLUID COMPOSITION (PER 2000 ML) AND MEASURED DRINK SOLUTE CONCENTRATIONS.

<table>
<thead>
<tr>
<th>Package Label Data</th>
<th>P1*</th>
<th>P2†</th>
<th>P2C§</th>
<th>AA²</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sodium Chloride (gm)</td>
<td>—</td>
<td>9.00</td>
<td>15.44</td>
<td>—</td>
</tr>
<tr>
<td>Sodium Citrate (gm)</td>
<td>3.87</td>
<td>7.74</td>
<td>7.74</td>
<td>—</td>
</tr>
<tr>
<td>Dextrose (gm)</td>
<td>41.12</td>
<td>82.24</td>
<td>82.24</td>
<td>—</td>
</tr>
<tr>
<td>Aspartane (gm)</td>
<td>—</td>
<td>—</td>
<td>0.72</td>
<td>—</td>
</tr>
<tr>
<td>Glycerol (gm)</td>
<td>100.87</td>
<td>100.87</td>
<td>100.87</td>
<td>—</td>
</tr>
<tr>
<td>Other Constituents</td>
<td>177.29</td>
<td>354.58</td>
<td>354.58</td>
<td>—</td>
</tr>
<tr>
<td>Total</td>
<td>545.43</td>
<td>545.43</td>
<td>545.43</td>
<td>—</td>
</tr>
</tbody>
</table>

Ionic Concentration: (mEq·L⁻¹/ %wt·vol⁻¹)

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<tr>
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<tbody>
<tr>
<td>Na⁺</td>
<td>19.61/0.04</td>
<td>39.22/0.09</td>
<td>39.22/0.09</td>
<td>157/0.36</td>
</tr>
<tr>
<td>K⁺</td>
<td>5.01/0.02</td>
<td>10.02/0.04</td>
<td>10.02/0.04</td>
<td>—</td>
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<tr>
<td>Cl⁻</td>
<td>4.98/0.02</td>
<td>9.96/0.04</td>
<td>9.96/0.04</td>
<td>76/0.27</td>
</tr>
<tr>
<td>Mg²⁺</td>
<td>0.40/0.01</td>
<td>0.80/0.01</td>
<td>0.80/0.01</td>
<td>—</td>
</tr>
<tr>
<td>Ca²⁺</td>
<td>1.96/0.02</td>
<td>3.92/0.03</td>
<td>3.92/0.03</td>
<td>—</td>
</tr>
<tr>
<td>P²⁺</td>
<td>0.51/0.01</td>
<td>1.02/0.02</td>
<td>1.02/0.02</td>
<td>—</td>
</tr>
<tr>
<td>Total</td>
<td>32.47/0.11</td>
<td>69.94/0.22</td>
<td>69.94/0.22</td>
<td>233/0.63</td>
</tr>
</tbody>
</table>

Sodium Chloride (gm) = 9.00
Sodium Citrate (gm) = 3.87
Dextrose (gm) = 41.12
Aspartane (gm) = —
Glycerol (gm) = 100.87
Other Constituents = 177.29
Total = 545.43

Carbohydrate (%wt·vol⁻¹)

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<table>
<thead>
<tr>
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<tbody>
<tr>
<td>Glucose</td>
<td>1.85</td>
<td>3.70</td>
<td>3.70</td>
</tr>
<tr>
<td>Fructose</td>
<td>2.43</td>
<td>4.85</td>
<td>4.85</td>
</tr>
<tr>
<td>Maltodextrin</td>
<td>5.44</td>
<td>10.88</td>
<td>10.88</td>
</tr>
<tr>
<td>Total</td>
<td>9.72</td>
<td>19.43</td>
<td>19.43</td>
</tr>
</tbody>
</table>

Measured Drink Solute Concentrations

<p>| | | | |</p>
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<thead>
<tr>
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</tr>
</thead>
<tbody>
<tr>
<td>Na⁺ (mEq·L⁻¹)</td>
<td>55.2</td>
<td>97.1</td>
<td>112.7</td>
</tr>
<tr>
<td>K⁺ (mEq·L⁻¹)</td>
<td>5.3</td>
<td>10.3</td>
<td>10.7</td>
</tr>
<tr>
<td>Osmolality (mOsm·kg·H₂O⁻¹)</td>
<td>365</td>
<td>791</td>
<td>1,382</td>
</tr>
<tr>
<td>Glycerol (mg·dl⁻¹)</td>
<td>2.0</td>
<td>4.0</td>
<td>2,916</td>
</tr>
<tr>
<td>Glucose (mg·dl⁻¹)</td>
<td>2,049</td>
<td>3,579</td>
<td>3,543</td>
</tr>
<tr>
<td>Citrate (mg·dl⁻¹)</td>
<td>416</td>
<td>753</td>
<td>731</td>
</tr>
</tbody>
</table>

Volume change = [(weight change - (blood + urine loss))] (5)

Blood measurements:

Plasma Na⁺, K⁺, glucose, glycerol and citrate concentrations were measured with a Cobas Mira S analyzer (Roche Diagnostic Systems, Inc., Branchburg, NJ); Na⁺ (glass membrane) and K⁺ (PVC valinomycin membrane) with ion-selective electrodes; glucose with hexokinase-NAD reactions and NADH read at 340 nm; glycerol with glycerokinase-glycerophosphate oxidase-peroxidase reac-
tions with the quinoneimine complex read at 490-550

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Plasma Osm was measured by freezing-point depression (model 3DI, Advanced Instruments Digi-
matic Osmometer, Needham Heights, MA).

Plasma volume was measured on frozen plasma with the Evans blue dye (T-1824, New World Trading Corp., DeBary, FL) dilution technique from one 10-min post-dye injection blood sample (4,16). Freezing does not change T-1824. Plasma was eluted through machine-packed chromatographic columns (model PD-10, Sepha-
dex G-25 M, Pharmacia LKB, Uppsala, Sweden) and the elutriate was read at 615 nm; PV = (V·St·v)/ (T·1.03); where: V = volume T-1824 injected, D = dilu-
tion of standard, St = standard absorbance, v = volume of sample extracted, t = test sample absorbance (subtract plasma blank), and 1.03 = correction factor for slow dye uptake by tissues. Percent change in PV was calculated using the Hb-Hct transformation equation (16).

Data from the Sephadex columns were compared with data from the standard manually packed columns (16). The optical density of 0.2 ml T-1824/10 ml acetone standard was measured (0.130); then 0.2 ml T-1824 were mixed with Tepol-phosphate and eluted through nine

at six sites. A Squirrel meter/logger (Grant model 1200, Science/Electronics Inc., Miamisburg, OH) was used for processing sensor signals. Mean skin temperature (Tsk) = 0.06 (TARM) + 0.13 (TFOREARM) + 0.21 (TTHIGH) + 0.21 (TCAFE) + 0.19 (TCHESST) + 0.20 (TRACK) (14,20). Mean room dry-bulb temperature was 21.8 ± 0.3°C, and relative humidity was 50 ± 2%. A fan increased air flow to 16.2 ± 1.2 m·min⁻¹ over the subject during exercise.

Body water balance = [weight change - (blood + urine loss)] (5) + drink volume = (CO₂ out - O₂ in) (5).
manually packed chromatographic columns and nine Sephadex columns. Mean (±SD, ±SE) optical density for the manual and Sephadex columns was 0.1103 (±0.0041, ± 0.0014) and 0.0989 (±0.0026, ±0.0006), respectively (ΔX = 14.0%, p < 0.0001). Thus, optical density from the Sephadex columns was lower and variability of the elutriate was about half that of the manually packed columns.

Mean corpuscular volume (MCV) = 10 (Hct · 9.6)/(RBC) in 10^6 µl.

Hct and Hb concentration were determined two ways: manually and with calculated values from the Coulter counter. The manual Hb values were 0.7 (rest) to 0.5 (exercise) g·dl⁻¹ higher than those calculated, and manual Hct values were 2.6 (rest) to 3.6 (exercise) units lower than those calculated; manual values were used for the plasma and blood volume determinations.

Urine measurements: Urine volume, collected at the end of rest (−15 min) and after exercise (+10 min of recovery), was timed and measured in a graduated cylinder. Urinary excretion rate (V) was expressed in ml·min⁻¹. Urinary Na⁺ (UNa⁺), K⁺ (UK⁺), and osmotic (UoSM) concentrations were determined with the same methods as the respective plasma variables. Other urine functions were calculated: Osm clearance (CosM) was urine Osm excretion (UoSMV) divided by plasma Osm (PosM) averaged over the urine collection period; free water clearance (CF) was V-CosM; and fractional ionic excretion was UNa⁺ + V and UK⁺ + V.

Percent change in the content of plasma constituents was constituent concentration times percent change in PV (35). Content data, which are independent of ΔPV, are discussed without presentation in tables or figures.

Statistical analysis: The data were analyzed with Student's t-test for dependent variables. The null hypothesis was rejected when p < 0.05, and nonsignificant differences were denoted by NS or trend or tendency. All variability is ± SE unless noted otherwise.

RESULTS

Blood Data

Plasma and mean corpuscular volumes: Mean percent change in PV from −105 min (Fig. 2, upper panel) indicates that, at the end of the rest phase (−15 min), the greater (p < 0.05) increases in PV occurred with the AA/P1 (by 7.9%) and P1/P1 (by 4.7%) treatments, and the lesser increases (NS) were with the 0/0 (by 1.7%) and 0/P1 (by 1.0%) treatments. Thus, the two non-drinking treatments at rest responded similarly. Change from sitting upright with the thighs positioned at a more downward angle (position change), resulted in decreasing trends in PV by time zero with all treatments—which probably resulted from the increased hydrostatic pressure and capillary filtration in the lower extremities.

During exercise, AA/P1 maintained the highest PV followed by P1/P1, 0/P1, P2G/P1, P2/P1, and 0/0 in decreasing order (Fig. 2, upper panel). Reduction in %ΔPV occurred with all treatments from 4–9 percentage units at 10 min of exercise, with essentially similar rates of recovery regardless of drinking or not; i.e., the 0/0 response was similar to the P2G/P1 response (Fig. 2, lower panel). Thus, the rate of PV restitution during exercise appeared to be independent of drink composition and whether or not fluid was consumed.

Mean corpuscular volumes (range 82–84 µl) were not different from each other or over time at rest or during exercise indicating no appreciable net change of vascular fluid into or from red blood cells.

Osmolality: Mean plasma osmotic concentrations were within the upper half of the normal range (277–297 mOsm·kg H₂O⁻¹); they varied between 288 and 293 mOsm·kg H₂O⁻¹ in the rest phase (Fig. 3, upper panel). In both non-drinking treatments (0/P1 and 0/0) plasma Osm remained constant during the first hour of rest. Osmolality varied ± 2 mOsm·kg⁻¹ by the end of rest; it increased with P1/P1 and P2G/P1 and decreased with AA/P2 and 0/P1 (Fig. 3, middle panel). Plasma Osm increased during exercise with all treatments, especially 0/P1 (with drinking P1) and 0/0 (without drinking). Intake of P1 during exercise tended to attenuate the increase in Osm. Treatments P1/P1, 0/P1, and AA/P1 had the lower Osm at the end of exercise (Fig. 3, lower panel).
onset of exercise (Fig. 5, lower panel). The K⁺ content in the drinks did not appear to influence the concentration or content responses at rest or during exercise. At 70 min of exercise the larger percent change in content occurred in AA/P1, 0/P1, and P2/P1 (containing K⁺), and the smallest change occurred in P1/P1 (also containing K⁺), with 0/0 (containing no K⁺) in the middle. Thus K⁺, the major intracellular cation, did not accompany the shift of Na⁺ and water from the vascular space at the beginning of exercise.

**Glucose**: Mean plasma glucose was elevated above the normal range of 64–115 mg·dl⁻¹ at the beginning of the rest period, probably a result of the high carbohydrate breakfast (Fig. 6, upper panel). Glucose concentration decreased with all treatments at rest and position change, with the greater decrease in those with no carbohydrate (AA/P1, 0/0, 0/P1). With exception of AA/P1, [glucose] and content decreased immediately with onset of exercise (similar to Osm and Na⁺), and then increased as exercise continued. Treatments 0/PI and 0/0 [glucose] were similar at time zero but, by the end of exercise (70 min), 0/P1 increased most (to 110 mg·dl⁻¹) while 0/0

which accompanied the greater increases in PV (Fig. 2, upper panel). As expected, treatment 0/0 exhibited the greatest increase in Osm by the end of exercise, while AA/P1 had the least increase (Fig. 3, lower panel). Also AA/P1, with the highest ionic Osm, had the greatest increase in plasma Osm content. Osmotic content of the remaining treatments returned to normal by the end of exercise. The acute decrease in plasma osmotic content at the beginning of exercise accompanied the shift of plasma from the vascular space, but there was a residual increase in plasma Osm of 2–6 mOsm·kg⁻¹.

**Sodium**: Plasma Na⁺ concentration [Nat] (Fig. 4) generally followed the comparable Osm. Because Na⁺ and accompanying anions account for a large part (80–90%) of plasma Osm (plasma [Na⁺] vs. Osm r = 0.93), the Osm contribution of other constituents (proteins, carbohydrates, non-electrolytes) was <14% (100 – r²).

**Potassium**: Mean plasma K⁺ was within the normal range at rest (Fig. 5, upper panel) and, unlike Na⁺, both K⁺ and content exhibited immediate increases with the onset of exercise (Fig. 5, lower panel). The K⁺ content in the drinks did not appear to influence the concentration or content responses at rest or during exercise. At 70 min of exercise the larger percent change in content occurred in AA/P1, 0/P1, and P2/P1 (containing K⁺), and the smallest change occurred in P1/P1 (also containing K⁺), with 0/0 (containing no K⁺) in the middle. Thus K⁺, the major intracellular cation, did not accompany the shift of Na⁺ and water from the vascular space at the beginning of exercise.

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that P1 was consumed during exercise with all treatments except 0/0, [citrate] in the four citrated drinks at rest converged at about 0.75 mg·dl⁻¹ at 70 min of exercise; there was a pronounced decrease in [citrate] with AA as consumption decreased from 15.44 g·2 L⁻¹ at rest to 3.87 g·2 L⁻¹ during exercise. But reducing citrate consumption by only 50% from rest to exercise did not appreciably alter the change in [citrate] in the P1/P1, P2/P1, and P2G/P1 treatments (Fig. 8, lower panel).

Urine Data

Excretion rate and electrolyte-osmotic concentration: Mean urine excretion rate (liters) at rest varied from 1.2 ± 0.3 ml·min⁻¹ (0/PT) to 3.2 ± 1.2 ml·min⁻¹ (P2G/P1), with a mean (n = 6) level of 2.3 ± 0.3 ml·min⁻¹ (Fig. 9, upper panel, solid line). Normal resting V is about 1.0 ml·min⁻¹. Excretion rate during exercise varied from 0.8 ± 0.3 ml·min⁻¹ (0/PT and 0/0) to 3.2 ± 0.8 ml·min⁻¹ (AA/PI), with a mean (n = 6) rate of 1.8 ± 0.4 ml·min⁻¹ (Fig. 9, upper panel, dashed line) which was not signifi-

increased least (to 85 mg·dl⁻¹) (Fig. 6, upper panel). Similar results were evident with Δglucose (Fig. 6, lower panel) and content. Thus, consumption of glucose during exercise resulted in the increase of both plasma [glucose] and content.

Glycerol: Only P2G contained appreciable (4%) glycerol. Mean plasma [glycerol] increased to 168 ± 33 mg·dl⁻¹ at 0 min of rest, maintained that level during the first 30 min of exercise, and then decrease to 116 ± 18 mg·dl⁻¹ at 70 min (Fig. 7, upper and lower panels). Apparently there was some glycerol metabolism, as the change in glycerol content decreased from 3462 ± 1430% at 0 min to 2208 ± 768% at 70 min of exercise.

Citrate: Mean resting plasma [citrate] varied from 1.7 ± 0.2 to 2.2 ± 0.3 mg·dl⁻¹, within the normal range of 1.7–3.0 mg·dl⁻¹ (Fig. 8, upper panel). Citrate was present in all drinks: 3.87 g·2 L⁻¹ in P1, 7.74 g·2 L⁻¹ in P2 and P2G, and 15.44 g·2 L⁻¹ in AA (Table II). Plasma [citrate] at 0 min increased by 0.5 pg·ml⁻¹ (P2G) to 1.7 pg·ml⁻¹ (AA), and remained essentially constant with 0/P1 and 0/0 (Fig. 8, lower panel). In spite of the fact
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Sodium excretion: Mean UNa\cdot V during exercise for the six treatments of 120 ± 29 μEq⋅min⁻¹ (Fig. 10, upper panel, dashed line) was lower (p < 0.05) than that at rest of 168 ± 19 μEq⋅min⁻¹ (solid line). The larger increase in Na⁺ excretion at rest and during exercise with AA/P1 was probably due to its higher Na⁺ of 164 mEq L⁻¹.

Potassium excretion: There was no significant difference between mean Ur·K at rest (58 ± 8 μEq⋅min⁻¹) and during exercise of 75 ± 20 μEq⋅min⁻¹ (Fig. 10, lower panel). The large increase in K⁺ excretion with AA/P1 during exercise probably accompanied a fluid shift from muscle cells to the interstitial and vascular spaces.

Physiological Data

Heart rate: Mean heart rate varied from 71 ± 6 to 87 ± 8 b⋅min⁻¹ at rest, and from 149 ± 9 to 160 ± 8 b⋅min⁻¹ at 70 min of exercise. The increased heart rate during exercise was lowest (Δ = 61 ± 10 b⋅min⁻¹) with P1/P1, and highest (Δ = 74 ± 10 b⋅min⁻¹) with AA/P1.

In general, urine Na⁺, K⁺, and Osm concentrations were lower with P1/P1 and P2/P1, and higher with AA/P1, 0/P1, and 0/0 treatments (Table II). The former reflected the lower drink Osm, while the latter resulted from the greater ionic content of AA/P1 (in spite of its lower Osm); the urine response to dehydration was similar to that following high salt consumption. The generally lower-than-Rest mean rates at rest. Exercise V was depressed similarly with P2/P1, P2G/P1, O/P1, and O/0; but not with AA/P1 with its higher Na⁺ content.

Osmotic clearance: There was no significant difference between mean Uosm·V/Posm at rest (3.0 ± 0.2 ml⋅min⁻¹) and during exercise of 2.4 ± 0.4 ml⋅min⁻¹ (Fig. 9, middle panel). The slight increase in Osm clearance with AA/P1 during exercise reflected the concomitant increased excretion of Na⁺ and K⁺ (Fig. 10).

Free water clearance: There was no significant difference between mean CH₂O at rest (−0.74 ± 0.23 ml⋅min⁻¹) and during exercise of −0.60 ± 0.24 ml⋅min⁻¹ (Fig. 9, lower panel). Treatments with higher ionic content (AA/P1) and dehydration (0/P1 and 0/0) had the lower free water clearances indicating greater water retention.

In general, urine Na⁺, K⁺, and Osm concentrations were lower with P1/P1 and P2/P1, and higher with AA/P1, 0/P1, and 0/0 treatments (Table II). The former reflected the lower drink Osm, while the latter resulted from the greater ionic content of AA/P1 (in spite of its lower Osm); the urine response to dehydration was similar to that following high salt consumption. The generally lower-than-Rest mean rates at rest. Exercise V was depressed similarly with P2/P1, P2G/P1, O/P1, and 0/0; but not with AA/P1 with its higher Na⁺ content.
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TABLE II. MEAN (±SE) URINE ELECTROLYTE CONCENTRATIONS AT REST (−105 TO −15 MIN) AND DURING EXERCISE (−15 TO +10 MIN) FOR THE SIX TREATMENTS.

<table>
<thead>
<tr>
<th>Variable</th>
<th>P1/P1</th>
<th>P/P1</th>
<th>P2G/P1</th>
<th>AA/P1</th>
<th>0/P1</th>
<th>0/0</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rest phase</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Urine Na⁺ (μEq·L⁻¹)</td>
<td>63.3 (17.4)</td>
<td>65.8 (17.5)</td>
<td>81.1 (22.8)</td>
<td>100.4 (18.6)</td>
<td>113.1 (17.2)</td>
<td>111.2 (21.7)</td>
</tr>
<tr>
<td>Urine K⁺ (μEq·L⁻¹)</td>
<td>18.2 (4.1)</td>
<td>17.8 (4.5)</td>
<td>29.0 (8.9)</td>
<td>39.8 (8.2)</td>
<td>51.7 (14.1)</td>
<td>66.8 (22.1)</td>
</tr>
<tr>
<td>Osmolality (mOsm·kgH₂O⁻¹)</td>
<td>328 (56)</td>
<td>368 (62)</td>
<td>443 (121)</td>
<td>498 (79)</td>
<td>752 (146)</td>
<td>712 (135)</td>
</tr>
<tr>
<td>Exercise phase</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Urine Na⁺ (μEq·L⁻¹)</td>
<td>47.6 (8.6)</td>
<td>72.9 (22.4)</td>
<td>55.1 (7.8)</td>
<td>80.6 (19.9)</td>
<td>102.3 (12.2)</td>
<td>126.5 (18.9)</td>
</tr>
<tr>
<td>Urine K⁺ (μEq·L⁻¹)</td>
<td>27.4 (3.8)</td>
<td>53.6 (24.5)</td>
<td>27.1 (2.1)</td>
<td>58.5 (6.2)</td>
<td>85.9 (13.4)</td>
<td>90.2 (19.2)</td>
</tr>
<tr>
<td>Osmolality (mOsm·kgH₂O⁻¹)</td>
<td>280 (33)</td>
<td>451 (124)</td>
<td>397 (87)</td>
<td>442 (89)</td>
<td>781 (116)</td>
<td>843 (105)</td>
</tr>
</tbody>
</table>

Fig. 9. Mean (±SE) urine excretion (upper panel), osmotic clearance (middle panel), and free water clearance (lower panel), at rest and during exercise for the six treatments.

Fig. 10. Mean (±SE) urine sodium excretion (upper panel) and potassium excretion (lower panel), at rest and during exercise for the six treatments.

Dehydration (0/0) did not result in the characteristic elevated heart rate at rest or during exercise.

Rectal and mean skin temperatures: Mean rectal temperature (Tᵣₑₜₐₜ) was stable with each treatment at rest; it varied from 36.6 ± 0.2°C with P2G/P1 to 37.2 ± 0.1°C with 0/P1 (Fig. 11, upper panel). The range and variability of resting Tᵣₑₜₐₜ decreased somewhat by time zero. Equilibrium levels of Tᵣₑₜₐₜ at min 70 of exercise varied from 37.98 ± 0.10°C (P1/P1) to 38.29 ± 0.17°C (0/P1), appropriate
levels with an exercise load of 70% peak \( \dot{V}O_2 \). Mean change in \( T_{Re} \) during exercise (Fig. 11, lower panel) did not exhibit the expected non-drinking response where the 0/0 (dehydration) increase in \( T_{Re} \) should have been the greatest. In fact, P2G/P1 showed the greatest increase (\( \Delta = 1.41 \pm 0.13 \)°C), followed by P2/P1 (\( \Delta = 1.34 \pm 0.17 \)°C), 0/0 (\( \Delta = 1.33 \pm 0.14 \)°C), AA/P1 (\( \Delta = 1.31 \pm 0.14 \)°C), 0/P1 (\( \Delta = 1.25 \pm 0.15 \)°C), and P1/P1 (\( \Delta = 1.14 \pm 0.08 \)°C). Thus, it appears that glycerol ingestion tends to elevate \( T_{Re} \), while P1 tends to attenuate the increase.

Absolute average mean skin temperatures (Tsk) and \( \Delta Tsk \) were not significantly different among the six treatments. Treatment 0/0 \( \Delta Tsk \) was nearest zero, while treatment 0/P1 tended to have the greater decrease. Lower Tsk suggests reduced blood flow reflecting perhaps greater sweating and evaporative water (heat) loss.

**Body water balance and sweat rate:** Mean total body water balance for the six treatments was 42 ± 7 ml at rest (Fig. 12, solid line) and −650 ± 81 ml (p < 0.01) during exercise (Fig. 12, dash line). Treatments P1/P1 and P2/P1 had mean positive balances at rest even with increased sweating (Table III) and AA/P1 had the greatest negative balance; whereas P2/P1 had the greatest negative balance during exercise, and P2G/P1 and AA/P1 the lesser negative balances (Fig. 12) reflecting reduced reduced sweating (Table III). Treatments 0/P1 and 0/0 had virtually similar unchanged rest water balances, and similar negative exercise balances indicating no effect of drinking P1 (Fig. 12, Table III).

**Salient Responses During Each Treatment**

**P1/P1:** Significant increase in \( \%\Delta PV \) at rest (by 4.7%), only positive exercise urinary free water clearance (0.30 ± 0.23 ml \( \cdot \) min\(^{-1} \)), lowest increase in exercise heart rate (\( \Delta = 61 \pm 10 \) b \( \cdot \) min\(^{-1} \)), and lowest increase in exercise TRE (\( \Delta = 1.14 \pm 0.08 \)°C).

**P2/P1:** No effect of double strength P1 on rest (2.6%) or exercise (−1.7%) \( \%\Delta PV \), low exercise urinary excretion rate (1.0 ± 0.2 ml \( \cdot \) min\(^{-1} \)), highest positive water balance at rest (93 g \( \cdot \) m\(^{-2} \cdot \) h\(^{-1} \)), and greatest negative exercise water balance (−315 g \( \cdot \) m\(^{-2} \cdot \) h\(^{-1} \)).

**P2G/P1:** Compared with P2/P1, there was no effect of 4% glycerol intake on rest or exercise \( \%\Delta PV \), higher urinary excretion rate at rest (3.2 ± 1.2 ml \( \cdot \) min\(^{-1} \)), and lowest increase in exercise TRE (\( \Delta = 1.41 \pm 0.13 \)°C).

**AA/P1:** Greatest increase in \( \%\Delta PV \) at rest (by 7.9%), highest level of exercise \( \%\Delta PV \) (by ~2% units), highest level of rest and exercise plasma Na\(^+\) K\(^+\), and Osm contents, lowest plasma glucose concentration (72 mg \( \cdot \) dl\(^{-1} \)) and increase in content (\( \Delta = 37 \)%) at rest, high reduction in exercise plasma glucose content (\( \Delta = -15 \)%) in presence of glucose intake, high exercise urinary excretion rate (3.2 ± 0.8 ml \( \cdot \) min\(^{-1} \)), highest rest (226 ± 45 \( \mu \) Eq \( \cdot \) min\(^{-1} \)) and exercise (254 ± 72 \( \mu \) Eq \( \cdot \) min\(^{-1} \)) urinary Na\(^+\) excretion, highest exercise urinary K\(^+\) excretion (174 ± 33 \( \mu \) Eq \( \cdot \) min\(^{-1} \)) and Osm clearance (4.36 ± 1.04 ml \( \cdot \) min\(^{-1} \)), lower rest (−1.29 ± 0.36 ml \( \cdot \) min\(^{-1} \)) and exercise (−1.16 ± 0.89 ml \( \cdot \) min\(^{-1} \)) urinary free water clearances, greatest negative water balance at rest (−78 g \( \cdot \) m\(^{-2} \cdot \) h\(^{-1} \)), and greatest increase in exercise heart rate (\( \Delta = 74 \pm 10 \) b \( \cdot \) min\(^{-1} \)).

**0/P1:** Lowest increase in \( \%\Delta PV \) at rest (by 0.7%); com-
pared with no drinking (0/0), P1 increased exercise \%ΔPV by 3.4% units, greatest reduction in exercise plasma glucose content (Δ = -9.8%), low rest (1.2 ± 0.3 ml·min⁻¹) and exercise (0.8 ± 0.3 ml·min⁻¹) urinary excretion rates, and low rest (−1.01 ± 0.29 ml·min⁻¹) and exercise (−0.91 ± 0.26 ml·min⁻¹) urinary free water clearances.

0/0: Second lowest increase in \%ΔPV at rest (by 1.2%), lowest exercise plasma glucose concentration (86 ± 3 mg·dl⁻¹) and reduction in content (Δ = −31%), low rest (1.8 ± 0.6 ml·min⁻¹) and exercise (0.8 ± 0.2 ml·min⁻¹) urinary excretion rates, and low rest (−1.38 ± 0.76 ml·min⁻¹) and exercise (−1.12 ± 0.21 ml·min⁻¹) free water clearances.

**Discussion**

Plasma volume—rest: The significant increases in PV in the present study with P1 (by 4.7%) and with AA (by 7.9%) after 90 min rest in euhydrated men confirmed similar significant increases in PV in a prior study of 24-h dehydrated men (17) with P1 (by 4.6%) and AA (by 7.6%) after 70 min rest. In the prior study the six formulations consumed at 12 ml·kg⁻¹ were ingested within 5 min at the beginning of the 70-min rest period. In the present study the drinks were ingested at 10 ml·kg⁻¹ intermittently throughout the initial 60 min of rest. It appears that drink composition, rather than moderate dehydration or rate of drinking, is the more important factor influencing the magnitude of rest hypervolemia. This conclusion is strengthened with findings from resting (supine) euhydrated men who ingested (16–17 ml·kg⁻¹) isotonic NaCl (0.9%, 315 mOsm·kg⁻¹) and hypertonic NaCl (1.5%, 493 mOsm·kg⁻¹) over 60 min (14). Percent change in PV at 60 and 90 min after drinking 1.5% saline was about +5% and +6.5%, respectively; after 0.9% saline it was about +7% and +11.5%, respectively. Compared with isotonic saline, hypertonic saline attenuated the hypervolemic response. But even greater and similar hypervolemia (to about 20%) was induced after subjects drank both iso- and hypertonic saline solutions in an ambient temperature of 39.4°C (14). Thus, excessive NaCl ingestion accentuates, and increased ambient temperature further enhances, resting hypervolemia.

Position change: Plasma volume with all treatments (except 0/P1) decreased during position change from the chair to cycle, where the arms were at the same level but the lower extremities were at a more dependent (downward) position facilitating increased capillary filtration.

Plasma volume—exercise: Plasma volume exhibited its characteristic decrease with onset of exercise to provide fluid for working muscles. The greatest decrease was with 0/0 and the least decrease was with AA/P1, attesting to the plasma-retaining property of Na⁺ and its anions. The slope of the increasing PV curves after 10 min of exercise appeared to be independent of P1 ingestion because the slope of the 0/0 curve was similar to the others, with exception of AA/P1 which tended to level off after 30 min of exercise. These data question the efficacy of drinking at all during 70 min of moderate exercise to maintain plasma and extracellular fluid volumes because the rate of PV increase, after the initial decrease when exercise commenced, was independent of drinking or no drinking. In general, it is clear that the greater the PV at rest, the greater the PV at the end of exercise; i.e., AA and P1 had the higher PV after rest and exercise. So pre-exercise hyperhydration seems better than dehydration during short-term exercise for maintenance of PV.

Somewhat similar PV responses occurred in a prior study (17) during exercise after drinking the six formulations at 12 ml·kg⁻¹ within 5 min. There was little restoration of PV during exercise with this bolus-drinking procedure. PV decreased by 4–10% (p < 0.05) within the first 15 min and remained more or less depressed throughout the 70 min of exercise. Water exhibited the greatest ΔPV of −14% (p < 0.05) throughout exercise suggesting that bolus ingestion of water before exercise is contraindicated for prompt elevation of PV. Similar reduction in PV, that remained essentially depressed.
during exercise, also occurred after drinking iso- and hypertonic NaCl solutions in a cool, and especially in a hot, environment (14).

Perhaps the inhibition of voluntary fluid intake; i.e., involuntary dehydration (13), may facilitate active muscle function during the initial period of exercise when hypovolemia occurs. Hemoconcentration is a fast mechanism for increasing plasma hormone parameters needed to facilitate the increased metabolism and cardiovascular adjustments during exercise. The shift of extracellular (interstitial) fluid and electrolytes into active muscle cells and the shift of cellular fluid, electrolytes, and metabolic end products from cells may require more than a few minutes to reach equilibrium. Oral intake and vascular uptake of fluids too soon during exercise may inhibit this equilibrium. Once equilibrium is reached, perhaps after 1 h of exercise, fluid intake could commence to prevent excessive total body dehydraton.

Plasma osmolality and electrolytes: Plasma Osm, with its main constituent Na⁺ and accompanying anions, is a major factor for maintenance and restoration of vascular volume homeostasis both directly, via its osmotic fluid-retention action, and indirectly through kidney fluid reabsorption via vasopressin (13). Morimoto and associates (19) have shown conclusively that restoration and maintenance of the extracellular volume by voluntary drinking depends on replacement of its Na⁺ content.

Plasma Osm, Na⁺, and K⁺ concentrations were remarkably stable during drinking at rest (also with no drinking) suggesting that the rate of drinking was such that the various fluid formulation constituents were sufficiently buffered to maintain plasma homeostasis. The increase in resting plasma Na⁺ with P2G, compared with that of only P2, suggests glycerol acts to hold body water in part by also increasing plasma Na⁺ content because P2G plasma volume changed minimally (by 2%). The fact that P2G Osm of 1382 mOsm · kg⁻¹ was so much greater than that of AA (253 mOsm · kg⁻¹), and that resting AA hypervolemia was significantly greater than that with P2G, support the conclusion that fluid formulation ionic contents are more important than its osmotic content for increasing PV at rest and, indirectly, for maintaining it during exercise. Change in plasma Osm at 70 min of exercise was approximately inversely related to change in PV for the formulations, which suggests that Osm was responding to change in PV rather than vice versa; otherwise high Osm would have been positively associated with high PV. Again, this observation indicates Osm as the dependent variable.

The essentially consistent increase in plasma K⁺ during exercise with all treatments indicates, unlike plasma Osm and Na⁺, that the increase was independent of drinking P1 because 0/0 responded similarly, and that it was responding to the exercise stimulus per se.

Plasma glucose, glycerol, citrate: The uniform decrease of plasma glucose at rest, from above to within its normal range, was generally greater with those treatments not containing carbohydrate (AA/P1, 0/P1, and 0/0). Because of the minimal change in plasma glucose during exercise with no drinking (0/0), the progressive increase in its concentration with the other formulations could be attributed to consumption of P1.

Addition of glycerol to P2 did not significantly affect the ΔPV from that of P2 without glycerol. While consumption of glycerol plus water induces total body hydration by inhibiting diuresis (24,33), it has not been possible during submaximal exercise to show a positive effect of glycerol on cardiovascular, thermoregulatory, or hormonal responses (30); or that it acts as an exogenous substrate to spare carbohydrate utilization (26,28). Also, glycerol ingestion in the present study resulted in the greatest increase in exercise rectal temperature (15). Because glycerol distributes into the total body water (all fluid compartments), it may have less of an hypervolmic effect than Na⁺-containing compounds which distribute mainly into the extracellular fluid space.

Citic acid is often added to beverages to lower pH, which acts as a preservative, and to improve taste. It was used in AstroAde to replace about half the chloride in NaCl to improve taste. There was no specific effect of citrate on the level of hypovolemia at rest because both P1 (with the lowest citrate content) and AA (with the highest citrate content) had the greater (significant) increases in PV.

Body water balance: urinary excretion and sweating: With such fluid intake at rest (561–977 ml · h⁻¹), a small positive total water balance—within the range of normal variability (±0.22% body wt, or ±150 ml)—would be expected. However, the consistently negative water balances during exercise were attenuated with both P2G/P1 (with intra- and extracellular distribution of glycerol) and AA/P1 (with mainly extracellular distribution of NaCl/NaCitrate). These attenuated negative balances were caused by decreased sweating because of their above normal urinary flows (2–3 ml · min⁻¹). Conversely, the greatest negative exercise water balance with P2/P1 was caused by 2–5 fold greater sweating because its urine excretion was normal. Thus, there was no consistent mechanism for variations in water balance with the various fluid formulations; some were caused by change in urinary flow, and others by change in sweating.

Rectal temperature and thermoregulatory parameters: There was no consistent inverse relationship between the magnitude of change in TRE and sweat rate during exercise (15). Changes in TRE were 1.41°C (P2G/P1), 1.34°C (P2/P1), 1.33°C (0/0), 1.31°C (AA/P1), 1.25°C (0/P1), and 1.14°C (P1/P1); corresponding sweat rates were 55, 247, 154, 84, 169, and 130 g · m⁻² · h⁻¹, respectively. Nor was there any regular relationship between ΔTRE and equilibrium (70 min) levels of plasma Na⁺ or Osm; factors influencing thermoregulation (10,22) which varied within their respective normal ranges: 146–149 mEq · L⁻¹ and 292–296 mOsm · kg⁻¹, respectively. Essentially similar ΔTRE (range = 0.3°C) and equilibrium levels of Na⁺ and Osm occurred during exercise with drinking these various fluid formulations before and during exercise. Thus, intake of these various fluid formulations does not appear to significantly influence ΔTRE or thermoregulatory parameters during 70 min of submaximal exercise.

Astronauts: reentry and landing: What appears to be a unique human hydration situation is that of astronauts in microgravity, where their PV and total body water are reduced chronically (11) while most plasma constituent concentrations remain at normal 1-G (eugravity) levels (7). This adaptive microgravic-hypovolemic-hypohy-
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Hydrated condition does not appear to result in adverse health or performance unless exacerbated by significant additional fluid loss from vomiting, diarrhea, or sweating. However, the microgravity-induced "latent" hyperhydration becomes functional dehydration during reentry as spacecraft deceleration and accompanying increased +Gz (head-to-foot) gravitational force on the astronauts reestablishes their hydropo...