Hypervolemia in Men from Fluid Ingestion at Rest and During Exercise

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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 compostion and Osm of fluids for hyperhydrating normally hydrated subjects is less clear. ***Methods:*** Six treatments were used without and with oral fluids of varying ionic and constituent concentrations for hyperhydrating six previously euhydrated men (30 ± SD 8 yr, 76.84 ± 16.19 kg, 73 ± 12 ml·kg^{-1} PV, 40 ± 10 ml·min^{-1}·kg^{-1} peak $\dot{V}O_2$) sitting at rest for 90 min ($\dot{V}O_2$ = 0.39 ± SE 0.02 L·min^{-1}) and during subsequent 70 min of submaximal exercise ($\dot{V}O_2$ = 2.08 ± SE 0.33 L·min^{-1}, 70 ± 7% peak $\dot{V}O_2$). 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, ($\bar{X}$ = 768 ml), for the sitting period were: Performance 1 (P1; 55 mEq Na^+, 365 mOsm·kg H_2O^{-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 01 and 02 (no drinking). The exercise drink (10 ml·kg^{-1}, 768 ml) was P1 for all treatments except 02 (no drinking); thus, drink designations were: P1/P1, P2/P1, P2G/P1, AA/P1, 0/P1, and 0/0. ***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, P2/P1, P2G/P1, and 0/P1; and −8% to −5% ($p < 0.05$) with 0/0. 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 in part by involuntary dehydration (12,13,34), defined as the delay in full fluid replacement (euhydration) 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·kgH_2O^{-1}), with more osmols 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. There have been few definitive studies on the efficacy of various drink formulations for increasing body fluid compartment volumes, especially PV in resting euhydrated subjects (23,27,29).

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

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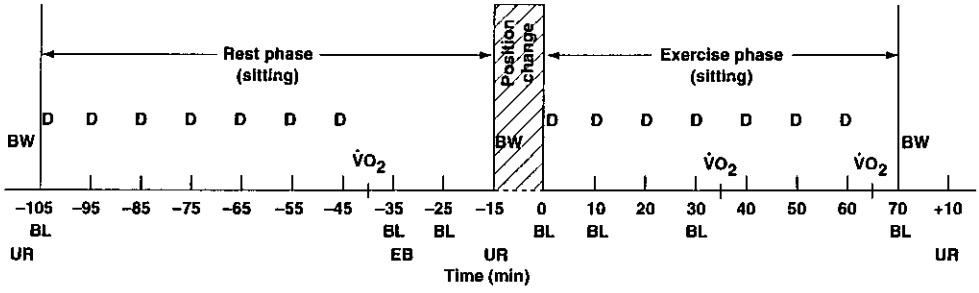

Fig. 1. Experimental protocol. BW = body weight, BL = blood sample, UR = urine, D = drinking (1/14 of total rest and exercise vol.), $\dot{V}O_2$ = oxygen uptake, and EB = Evans blue injection.

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 m^2 S.A., 3124 ± 505 ml PV, 5522 ± 923 ml BV, and 2.99 ± 0.45 $L \cdot min^{-1}$ peak $\dot{V}O_2$ 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% $\dot{V}O_2$ 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 $ml \cdot kg^{-1}$ 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 ($\dot{V}O_2$ 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., Slymar, CA), and a 3-L mixing chamber from which expired gas was sampled at 0.5 $L \cdot min^{-1}$ 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-3AI and CD-3A, respectively; Ametek, Thermox Instruments Division, Pittsburg, 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 $kg \cdot m^{-1} \cdot min^{-1}$, RE = 1.25 ± 0.04, $\dot{V}_{EBTPS}$ = 126.00 ± 6.14 $L \cdot min^{-1}$, HR = 187 ± 7 $b \cdot min^{-1}$, and $\dot{V}O_2$ = 40 ± 4 $ml \cdot min^{-1} \cdot kg^{-1}$. The submaximal exercise load (817 ± SD 133 $kg \cdot m^{-1} \cdot min^{-1}$) corresponded to an oxygen uptake of 70 ± SD 7% of the measured peak $\dot{V}O_2$.

Heart rate was determined with a cardiotachometer (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.

	P1*	P2†	P2G§	AA‡
Package Label Data				
Sodium Chloride (gm)	—	—	—	9.00
Sodium Citrate (gm)	3.87	7.74	7.74	15.44
Dextrose (gm)	41.12	82.24	82.24	—
Aspartame (gm)	—	—	—	0.72
Glycerol (gm)	—	—	100.87	—
Other Constituents	177.29	354.58	354.58	—
Total	222.28	444.56	545.43	25.16
Ionic Concentration: ($mEq \cdot L^{-1}$,/$\%wt \cdot vol^{-1}$)				
Na^+	19.61/0.04	39.22/0.09	39.22/0.09	157/0.36
K^+	5.01/0.02	10.02/0.04	10.02/0.04	—
Cl^-	4.98/0.02	9.96/0.04	9.96/0.04	76/0.27
Mg^{2+}	0.40/0.01	0.80/0.01	0.80/0.01	—
Ca^{2+}	1.96/0.02	3.92/0.03	3.92/0.03	—
P^{4+}	0.51/0.01	1.02/0.02	1.02/0.02	—
Total	32.47/0.11	69.94/0.22	69.94/0.22	233/0.63
Carbohydrate ($\%wt \cdot vol^{-1}$)				
Glucose	1.85	3.70	3.70	—
Fructose	2.43	4.85	4.85	—
Maltodextrin	5.44	10.88	10.88	—
Total	9.72	19.43	19.43	—
Measured Drink Solute Concentrations				
Na^+ ($mEq \cdot L^{-1}$)	55.2	97.1	112.7	163.7
K^+ ($mEq \cdot L^{-1}$)	5.3	10.3	10.7	<0.1
Osmolality ($mOsm \cdot kgH_2O^{-1}$)	365	791	1,382	253
Glycerol ($mg \cdot dl^{-1}$)	2.0	4.0	2,916	1.0
Glucose ($mg \cdot dl^{-1}$)	2,049	3,579	3,543	<0.5
Citrate ($mg \cdot dl^{-1}$)	416	753	731	854

* Shaklee Performance, Shaklee U.S., Inc., San Francisco, CA.
† Double strength Shaklee Performance.
§ Double strength Shaklee Performance plus 4% glycerol.
‡ AstroAde – NaCl/NaCitrate.

at six sites. A Squirrel meter/logger (Grant model 1200, Science/Electronics Inc., Miamisburg, OH) was used for processing sensor signals. Mean skin temperature ($\bar{T}_{SK}$) = 0.06 (T_{ARM}) +0.13 ($T_{FOREARM}$) +0.21 (T_{THIGH}) +0.21 (T_{CALF}) +0.19 (T_{CHEST}) +0.20 (T_{BACK}) (14,20). Mean room dry-bulb temperature was 21.8 ± SD 0.3°C, and relative humidity was 50 ± 2%. A fan increased air flow to 16.2 ± 1.2 $m \cdot min^{-1}$ over the subject during exercise.

Body water balance = [(weight change − (blood + urine loss) + drink volume − (CO_2 out − O_2 in)] (5).

Blood measurements: Blood samples (15 ml each; 20 ml each at −25 and −35 min, 115 ml/experiment) were withdrawn through an 18G catheter (Quik-Cath, Baxter Healthcare Corp., Deerfield, IL) inserted into the right antecubital vein. Blood samples were placed into four Vacutainer® tubes: tube 1 = 2 ml for hemoglobin (Hb) and hematocrit (Hct); tube 2 = 3 ml for glucose; tube 3 = 10 ml for Na^+, K^+, Osm, RBC, and glycerol; and tube 4 = 5 ml for Evans blue dye (PV) analysis. Hemoglobin and Hct were measured immediately (manually). Hemoglobin was measured (cyanomethemoglobin method) with the Coulter Diluter II and Hemoglobinometer (Coulter Electronics, Hialeah, FL). Blood for Hct was drawn into four capillary tubes, centrifuged for 10 min at 11,500 rpm (model MB, International Equipment Co., Needham Heights, MA), and read with a modified microcapillary tube reader (model CR, International Equipment Co.). Hemoglobin and Hct were also calculated automatically with a Coulter model STKS analyzer. Plasma was frozen (−20°C) for subsequent analysis.

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 glycerolkinase-glycerophosphate oxidase-peroxidase reactions with the quinoneimine complex read at 490–550 nm; and citrate with citrate lyase (NADH to NAD^+ at 340 nm). Plasma Osm was measured by freezing-point depression (model 3DII, Advanced Instruments Digimatic 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, Sephadex G-25 M, Pharmacia LKB, Uppsala, Sweden) and the elutriate was read at 615 mμ. PV = $(V \cdot D \cdot St \cdot v)/(T \cdot 1.03)$; where: V = volume T-1824 injected, D = dilution 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 Teepol-phosphate and eluted through nine

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 0.0949 (±0.0026, ± 0.0008), respectively ($\Delta\bar{X}$ = 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·0.96)/([RBC] in $10^6 \cdot \mu^3$).

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) $g \cdot dl^{-1}$ 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 ($\dot{V}$) was expressed in $ml \cdot min^{-1}$. Urinary Na^+ (U_{Na^+}), K^+(U_{K^+}), and osmotic (U_{OSM}) concentrations were determined with the same methods as the respective plasma variables. Other urine functions were calculated: Osm clearance (C_{OSM}) was urine Osm excretion ($U_{OSM}\dot{V}$) divided by plasma Osm (P_{OSM}) averaged over the urine collection period; free water clearance (C_{H_2O}) was $\dot{V}$-C_{OSM}; and fractional ionic excretion was $U_{Na}+\dot{V}$ and $U_K+\dot{V}$.

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.

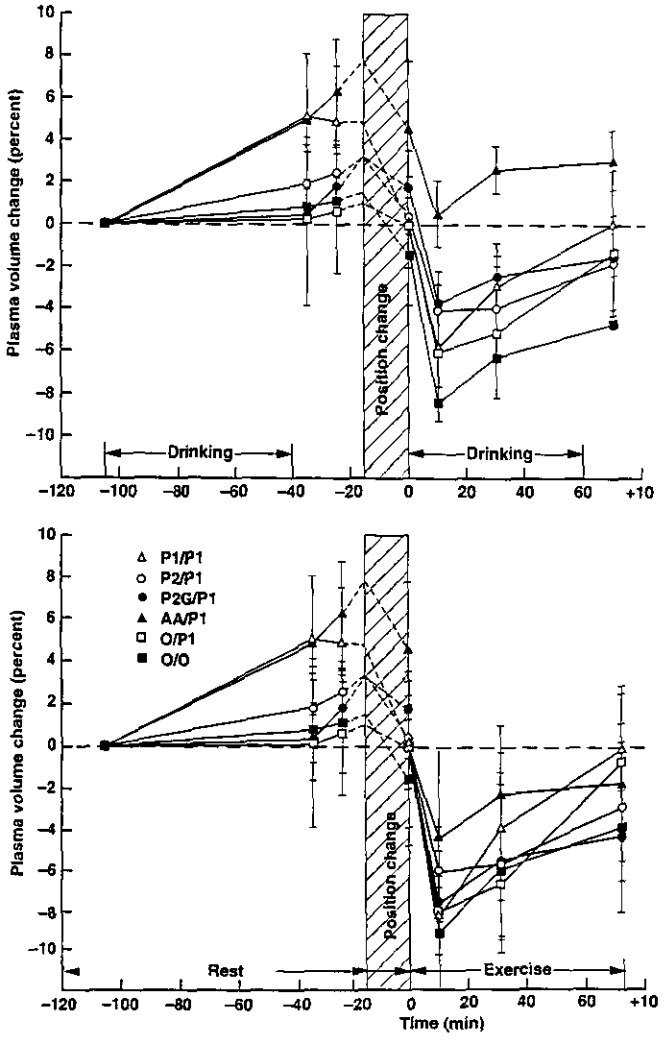

Fig. 2. Mean (±SE) percent change in plasma volume at rest and during exercise for the six treatments. Change from −105 min (upper panel), and change from −105 min and from 0 min (lower panel).

RESULTS

Blood Data

Plasma and mean corpuscular volumes: Mean percent change in PV from −105 min (**Fig. 2**, upper panel) indicate 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 in a chair with the thighs horizontal, to sitting upright on the cycle with thighs and legs 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 μ^3) 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 $mOsm \cdot kg\ H_2O^{-1}$); they varied between 288 and 293 $mOsm \cdot kg\ H_2O^{-1}$ 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 $mOsm \cdot kg^{-1}$ 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)

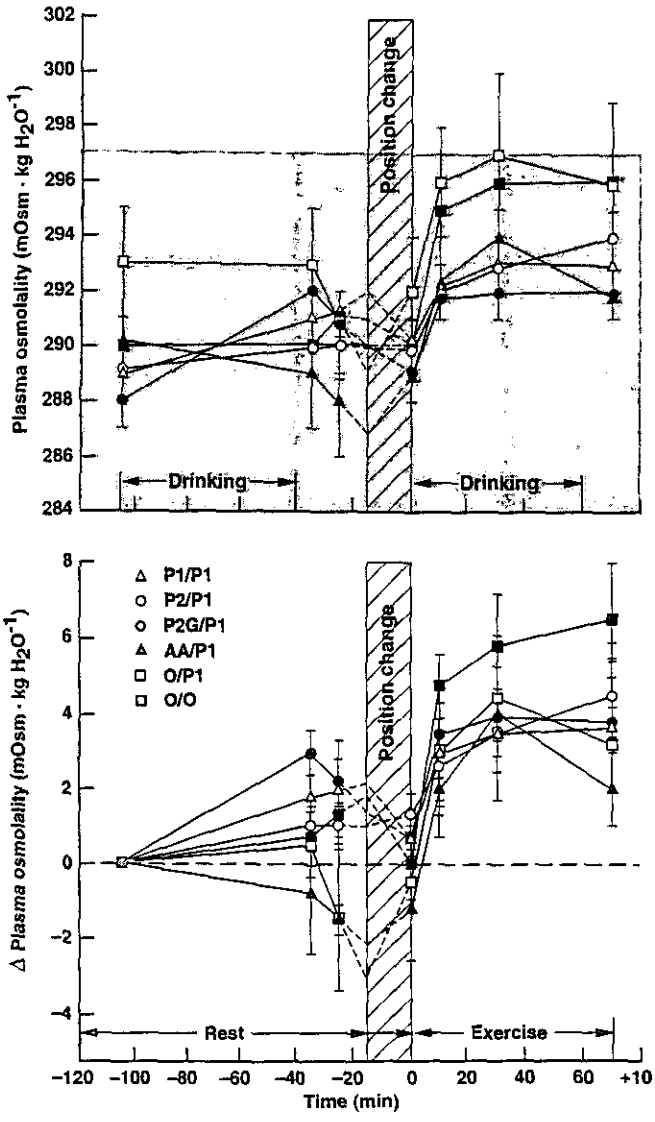

Fig. 3. Mean (±SE) plasma osmotic concentration (upper panel), and change in concentration from −105 min (lower panel), at rest and during exercise for the six treatments. Shaded area is normal range.

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 [Na^+] (**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 − r^2).

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.

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/P1 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

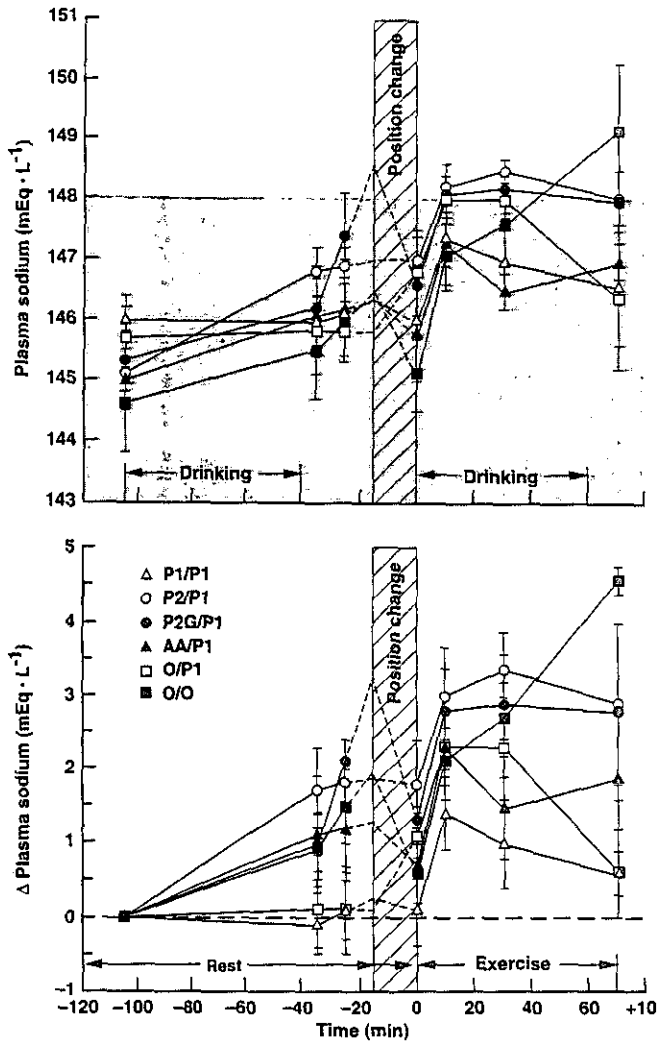

Fig. 4. Mean (±SE) plasma sodium concentration (upper panel), and change in concentration from −105 min (lower panel), at rest and during exercise for the six treatments. Shaded area is normal range.

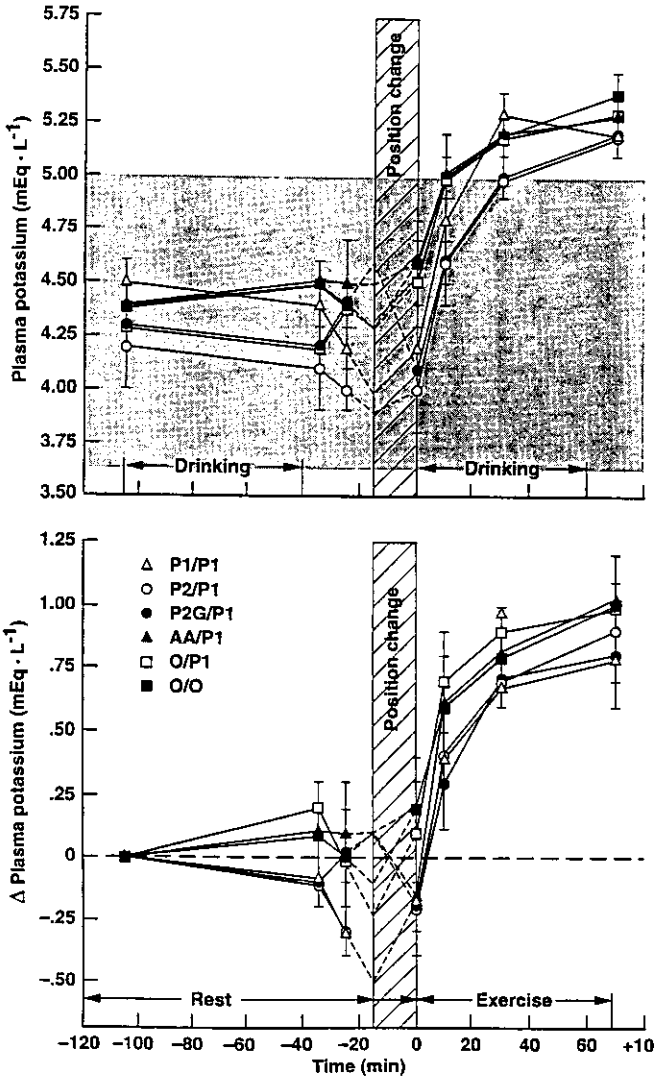

Fig. 5. Mean (±SE) plasma potassium concentration (upper panel), and change in concentration from −105 min (lower panel) at rest and during exercise for the six treatments. Shaded area is normal range.

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 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 ($\dot{V}$) at rest varied from 1.2 ± 0.3 ml · min^{-1} (0/P1) 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 $\dot{V}$ is about 1.0 ml · min^{-1}. Excretion rate during exercise varied from 0.8 ± 0.3 ml · min^{-1} (0/P1 and 0/0) to 3.2 ± 0.8 ml · min^{-1} (AA/P1), with a mean (n = 6) rate of 1.8 ± 0.4 ml · min^{-1} (Fig. 9, upper panel, dashed line) which was not signifi-

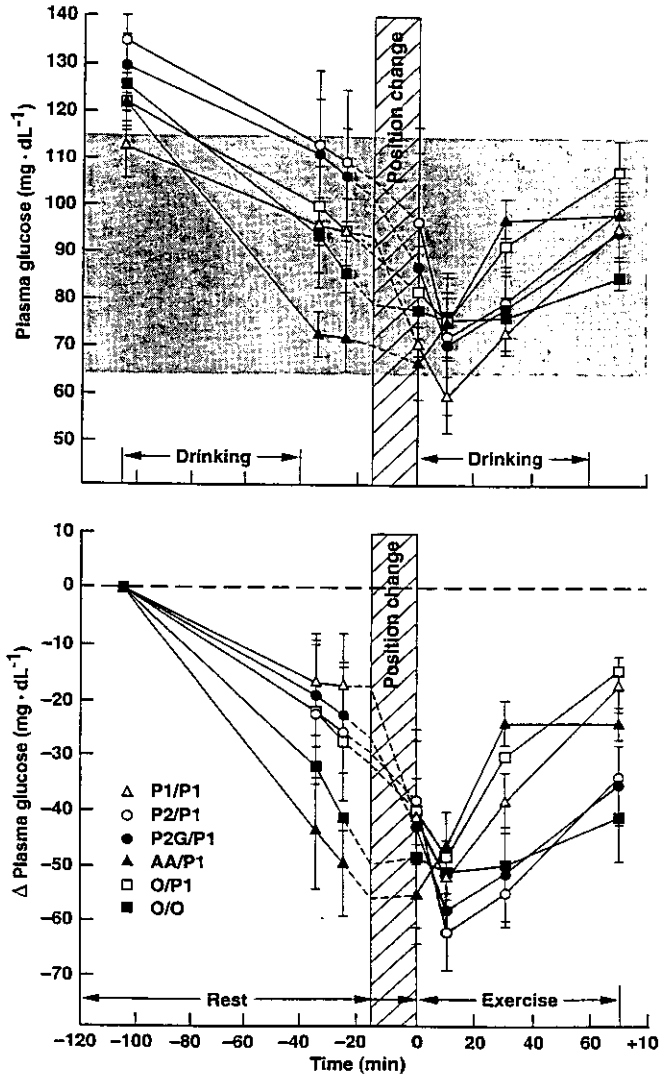

Fig. 6. Mean (±SE) plasma glucose concentration (upper panel), and change in concentration from −105 min (lower panel), at rest and during exercise for the six treatments. Shaded area is normal range.

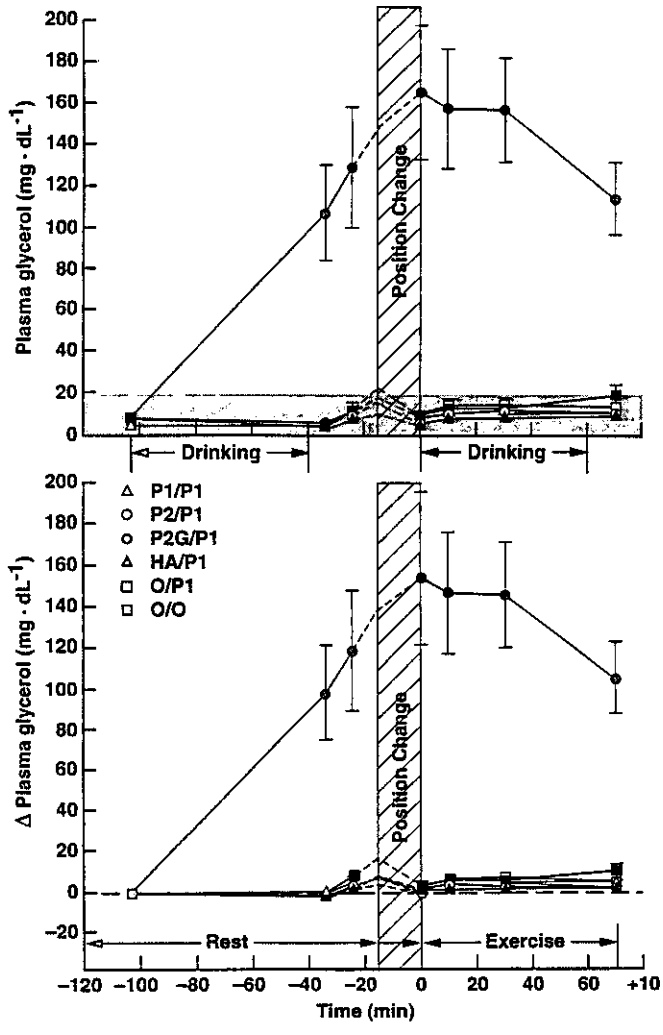

Fig. 7. Mean (±SE) plasma glycerol concentration (upper panel), and change in concentration from −105 min (lower panel), at rest and during exercise for the six treatments. Shaded area is normal range.

cantly lower than the mean rate at rest. Exercise $\dot{V}$ was depressed similarly with P2/P1, P2G/P1, 0/P1, and 0/0; but not with AA/P1 with its higher Na^+ content.

Osmotic clearance: There was no significant difference between mean $U_{OSM} \cdot \dot{V}/P_{OSM}$ 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 C_{H_2O} 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 elevated urine $[K^+]$ during exercise over those at rest probably resulted from increased muscle activity.

Sodium excretion: Mean $U_{Na} \cdot \dot{V}$ 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 $U_K \cdot \dot{V}$ 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 ($\Delta = 61 \pm 10$ b · min^{-1}) with P1/P1, and highest ($\Delta = 74 \pm 10$ b · min^{-1}) with AA/P1.

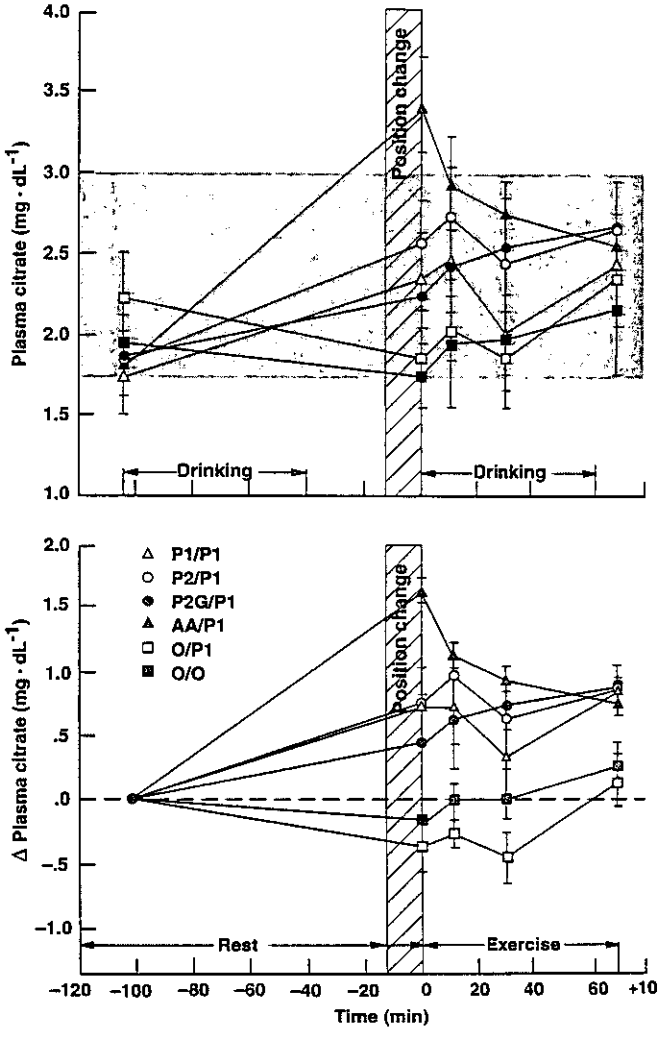

Fig. 8. Mean (±SE) plasma citrate concentration (upper panel) and change in concentration from −105 min (lower panel), at rest and during exercise for the six treatments. Shaded area is normal range.

TABLE II. MEAN (±SE) URINE ELECTROLYTE CONCENTRATIONS AT REST (−105 TO −15 MIN) AND DURING EXERCISE (−15 TO +10 MIN) FOR THE SIX TREATMENTS.

Variable	P1/P1	P/P1	P2G/P1	AA/P1	0/P1	0/0
			Rest phase			
Urine Na^+ ($\mu Eq \cdot L^{-1}$)	63.3 (17.4)	65.8 (17.5)	81.1 (22.8)	100.4 (18.6)	113.1 (17.7)	111.2 (21.7)
Urine K^+ ($\mu Eq \cdot L^{-1}$)	18.2 (4.1)	17.8 (4.5)	29.0 (8.0)	39.8 (8.2)	51.7 (14.1)	66.8 (22.1)
Osmolality ($mOsm \cdot kgH_2O^{-1}$)	328 (56)	368 (62)	443 (121)	498 (79)	752 (146)	712 (135)
			Exercise phase			
Urine Na^+ ($\mu Eq \cdot L^{-1}$)	47.6 (8.6)	72.9 (22.4)	55.1 (7.8)	80.6 (19.9)	102.3 (12.2)	126.5 (18.9)
Urine K^+ ($\mu Eq \cdot L^{-1}$)	27.4 (3.8)	53.6 (24.5)	27.1 (2.1)	58.5 (6.2)	85.9 (13.4)	90.2 (19.2)
Osmolality ($mOsm \cdot kgH_2O^{-1}$)	280 (33)	451 (124)	397 (87)	442 (89)	781 (116)	843 (105)

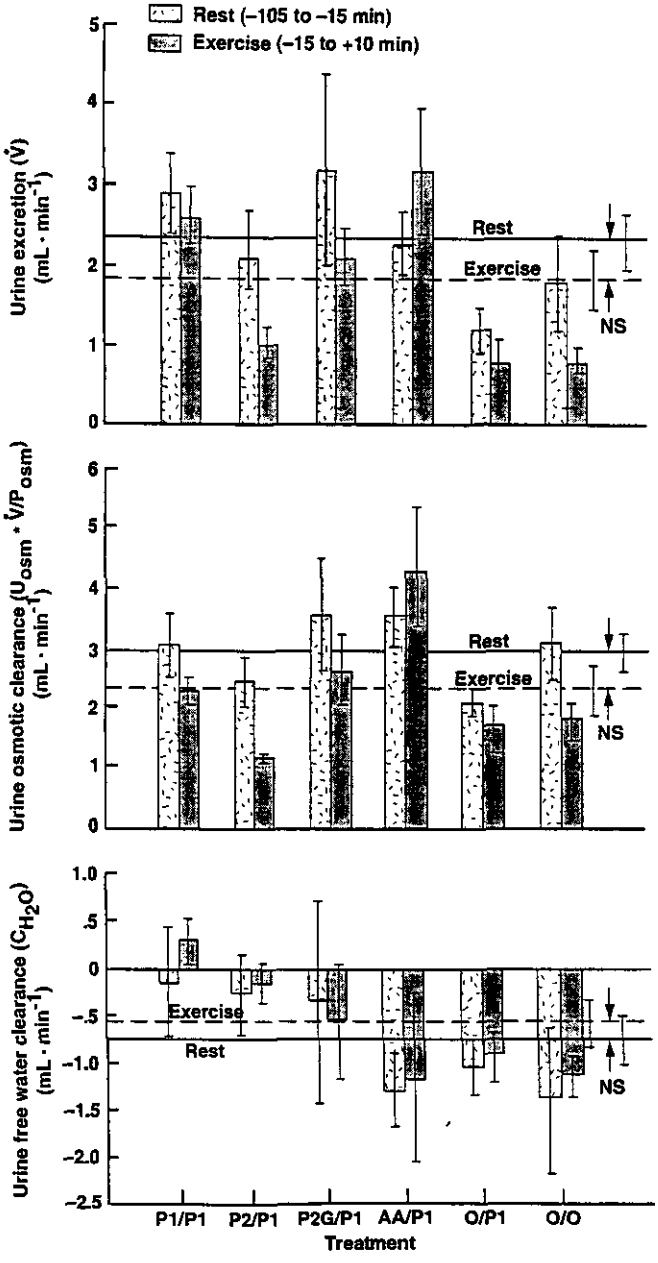

Fig. 9. Mean (±SE) urine excretion (upper panel), osmotic clearance (middle panel), and free water clearance (lower panel), at rest and during exercise for the six treatments.

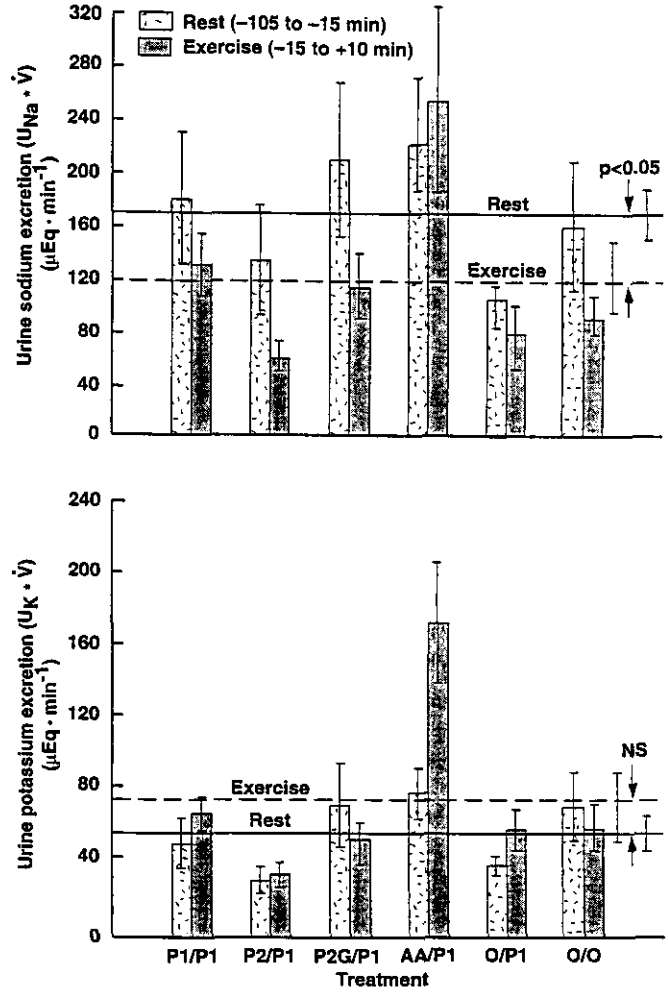

Fig. 10. Mean (±SE) urine sodium excretion (upper panel) and potassium excretion (lower panel), at rest and during exercise for the six treatments.

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 (T_{RE}) 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 T_{RE} decreased somewhat by time zero. Equilibrium levels of T_{RE} at min 70 of exercise varied from 37.98 ± 0.10°C (P1/P1) to 38.29 ± 0.17°C (0/P1), appropriate

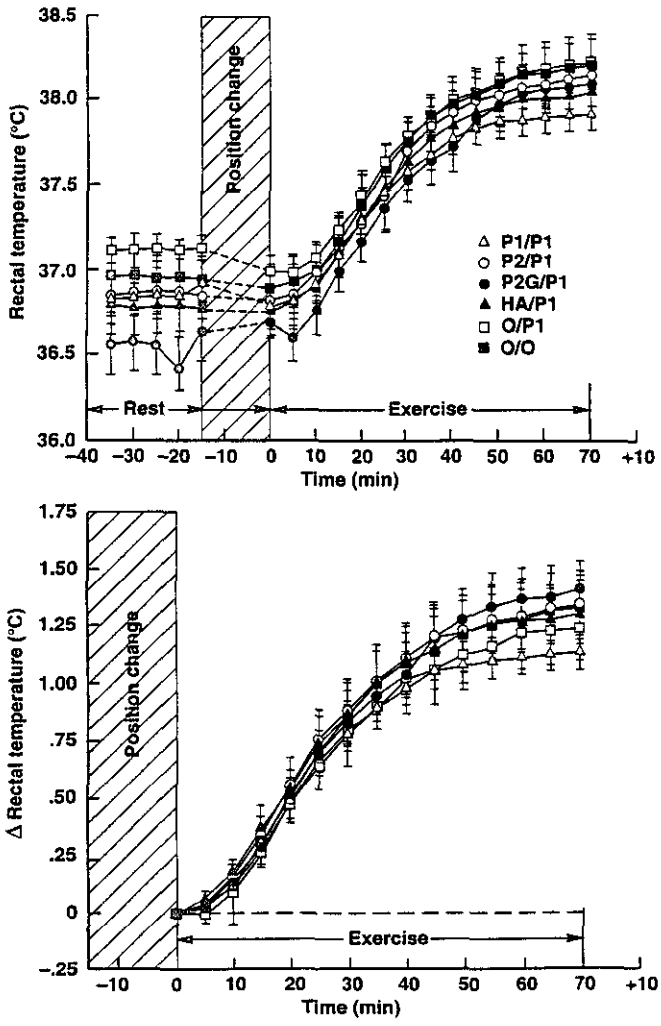

Fig. 11. Mean (±SE) rectal temperature at rest and during exercise (upper panel), and change in rectal temperature from time zero during exercise (lower panel), for the six treatments.

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 (Δ = 1.41 ± 0.13°C); followed by P2/P1 (Δ = 1.34 ± 0.17°C), 0/0 (Δ = 1.33 ± 0.14°C), AA/P1 (Δ =1.31 ± 0.14°C), 0/P1 (Δ = 1.25 ± 0.15°C), and P1/P1 (Δ = 1.14 ± 0.08°C). Thus, it appears that glycerol ingestion tends to elevate ΔT_{RE}, while P1 tends to attenuate the increase.

Absolute average mean skin temperatures ($\bar{T}_{SK}$) and $\Delta \bar{T}_{SK}$ were not significantly different among the six treatments. Treatment 0/0 $\Delta \bar{T}_{SK}$ was nearest zero, while treatment 0/P1 tended to have the greater decrease. Lower $\bar{T}_{SK}$ 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 greater 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 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 %ΔPV at rest (by 4.7%), only positive exercise urinary free water clearance (0.30 ± 0.23 ml · min^{-1}), lowest increase in exercise heart rate (Δ = 61 ± 10 b · min^{-1}), and lowest increase in exercise T_{RE}. (Δ = 1.14 ± 0.08°C).

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

P2G/P1: Compared with P2/P1, there was no effect of 4% glycerol intake on rest or exercise %ΔPV, higher urinary excretion rate at rest (3.2 ± 1.2 ml · min^{-1}), and greatest increase in exercise T_{RE} (Δ = 1.41 ± 0.13°C).

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

0/P1: Lowest increase in %ΔPV at rest (by 0.7%); com-

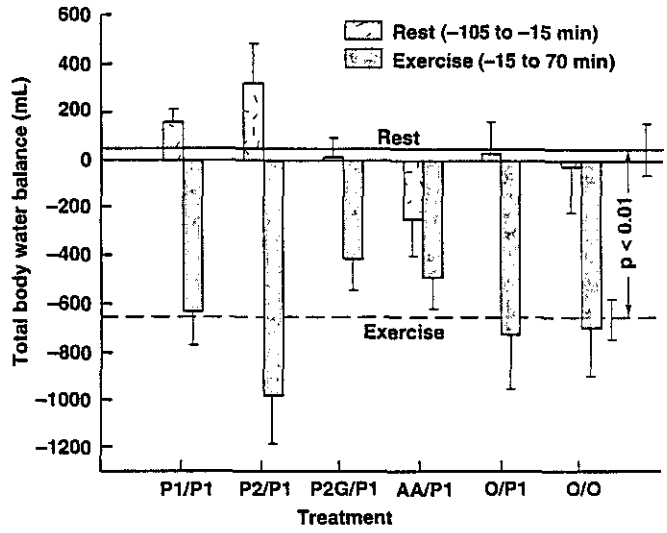

Fig. 12. Mean (±SE) total body water balance at rest and during exercise for the six treatments. Solid horizontal line is mean (n = 6) of rest values; dash horizontal line is mean of exercise values.

TABLE III. MEAN (±SE) WATER BALANCE, RESPIRATORY WATER LOSS, INSENSIBLE WATER LOSS, AND SWEAT RATE AT REST (−105 TO −15 MIN) AND DURING EXERCISE (−15 TO 70 MIN) FOR THE SIX TREATMENTS.

Variable	P1/P1	P2/P1	P2G/P1	AA/P1	0/P1	0/0	$\bar{X}$
			Rest phase				
Water balance ($g \cdot m^{-2} \cdot hr^{-1}$)	48 (19)	93 (52)	2 (23)	−78 (47)	6 (39)	−23 (65)	8 (24)
Respiratory water loss ($g \cdot m^{-2} \cdot hr^{-1}$)	12 (1)	12 (1)	12 (1)	12 (1)	10 (1)	10 (1)	11 (0.4)
Insensible water loss ($g \cdot m^{-2} \cdot hr^{-1}$)	18	18	18	18	18	18	18
Sweat loss ($g \cdot m^{-2} \cdot hr^{-1}$)	77 (19)	123 (52)	31 (22)	−48 (47)	34 (39)	5 (65)	35 (24)
			Exercise phase				
Water balance ($g \cdot m^{-2} \cdot hr^{-1}$)	−198 (42)	−315 (73)	−126 (34)	−151 (31)	−238 (73)	−221 (62)	−208 (27)
Respiratory water loss ($g \cdot m^{-2} \cdot hr^{-1}$)	49 (2)	50 (3)	52 (2)	49 (1)	50 (4)	49 (3)	50 (0.5)
Insensible water loss ($g \cdot m^{-2} \cdot hr^{-1}$)	18	18	18	18	18	18	18
Sweat loss ($g \cdot m^{-2} \cdot hr^{-1}$)	130 (40)	247 (72)	55 (34)	84 (30)	169 (71)	154 (59)	140 (28)

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 $ml \cdot min^{-1}$) and exercise (0.8 ± 0.3 $ml \cdot min^{-1}$) urinary excretion rates, and low rest (−1.01 ± 0.29 $ml \cdot min^{-1}$) and exercise (−0.91 ± 0.26 $ml \cdot min^{-1}$) urinary free water clearances.

0/0: Second lowest increase in %ΔPV at rest (by 1.2%), lowest exercise plasma glucose concentration (86 ± 3 $mg \cdot dl^{-1}$) and reduction in content (Δ = −31%), low rest (1.8 ± 0.6 $ml \cdot min^{-1}$) and exercise (0.8 ± 0.2 $ml \cdot min^{-1}$) urinary excretion rates, and low rest (−1.38 ± 0.76 $ml \cdot min^{-1}$) and exercise (−1.12 ± 0.21 $ml \cdot min^{-1}$) 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 $ml \cdot kg^{-1}$ were ingested within 5 min at the beginning of the 70-min rest period. In the present study the drinks were ingested at 10 $ml \cdot kg^{-1}$ 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 $ml \cdot kg^{-1}$) isotonic NaCl (0.9%, 315 $mOsm \cdot kg^{-1}$) and hypertonic NaCl (1.5%, 493 $mOsm \cdot kg^{-1}$) 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 rehydration 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 $ml \cdot kg^{-1}$ 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 dehydration.

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 $mOsm \cdot kg^{-1}$ was so much greater than that of AA (253 $mOsm \cdot kg^{-1}$), 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 stimuli 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 effect the ΔPV from that of P2 without glycerol. While consumption of glycerol plus water induces total body hyperhydration 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 hypervolemic effect than Na^+-containing compounds which distribute mainly into the extracellular fluid space.

Citric 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 hypervolemia 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 $ml \cdot h^{-1}$), 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 $ml \cdot min^{-1}$). 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 T_{RE} and sweat rate during exercise (15). Changes in T_{RE} 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 $g \cdot m^{-2} \cdot h^{-1}$, respectively. Nor was there any regular relationship between ΔT_{RE} and equilibrium (70 min) levels of plasma Na^+ or Osm; factors influencing thermoregulation (10,22) which varied within their respective normal ranges: 146–149 $mEq \cdot L^{-1}$ and 292–296 $mOsm \cdot kg^{-1}$, respectively. Essentially similar ΔT_{RE} (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 ΔT_{RE} 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-

drated 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" hypohydration becomes functional dehydration during reentry as spacecraft deceleration and accompanying increased +Gz (head-to-foot) gravitational force on the astronauts reestablishes their hypohydrated state. This hypohydration can contribute to their general reentry syndrome (GRS) characterized by adverse pre-syncopal signs and symptoms including fatigue, gastrointestinal discomfort, postural disequilibrium, vertigo, occasional emesis and syncope, and general debilitation that often occur during reentry and immediately after landing (8). Performance 1 and AstroAde would seem to be efficacious beverages to "hyperhydrate" astronauts prior to reentry because both formulations induce effective hypervolemia in resting, "euhydrated" subjects. Of the 18 male subjects tested in the present and prior (17) series of fluid intake studies, only one subject in the present study exhibited mild gastrointestinal distress. Astronauts don somewhat cumbersome reentry suits, fitted with inflatable bladders for acceleration and orthostatic protection, a few hours before reentry when their drinking (hyperhydration) countermeasure occurs. The inflated abdominal bladder can cause discomfort with a full stomach, and urinating into the suit is also upleasant; hence the reluctance of some astronauts to hyperhydrate. In our subjects the resting urinary excretion was lowest without drinking (1.2–1.8 $ml \cdot min^{-1}$), was somewhat lower with AstroAde (2.3 $ml \cdot min^{-1}$) than with Performance 1 (2.9 $ml \cdot min^{-1}$), and was highest with Performance 2/Glycerol (3.2 $ml \cdot min^{-1}$). After hyperhydrating with AstroAde (10 $ml \cdot kg^{-1}$ body wt), a resting urinary output of 414 $ml \cdot 3\ h^{-1}$ may be retained comfortably by astronauts during preparation, reentry, and landing if they urinated prior to donning their reentry suits.

Astronauts: extravehicular activity (EVA): Fluid balance (intake minus outgo) perturbations during EVA would depend on the astronauts rest/exercise ratio and total time of exposure. Hyperhydration before EVA, similar to that discussed above, might be useful and comfortable for 3–4 h of EVA with low to mild metabolic (exercise) intensity. Because the mode of exercise during EVA appears to be mainly isometric or isokinetic, data from the cycle isotonic exercise performed in the present study may not be applicable. Fluid balance data from use of various fluid formulations are needed from studies utilizing isometric and isokinetic exercise in a simulated EVA suit environment to enhance EVA countermeasures.

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