Sucrose Ingestion Following Exercise: Selected Cardiovascular, Hormonal, Renal, and Metabolic Effects

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Carbohydrates, frequently consumed following exercise for glycogen resynthesis, have been shown to have other systemic effects in resting men. We examined the effects of postexercise sucrose (a disaccharide carbohydrate) ingestion on the renal, cardiovascular, and sympathetic nervous systems. Eight men consumed 1 l of water (W) or 1 l of a 200 g sucrose solution (S) following 1 hour of bicycle exercise at 70% heart rate reserve. Measurements were made during 2 hours of recovery. Heart rate and systolic blood pressure were elevated following S as compared to W ($p < 0.009$, $p < 0.04$, respectively). Diastolic blood pressure was lower after S ($p < 0.04$) and mean blood pressure did not differ between beverages. Plasma and urinary catecholamines decreased similarly after exercise regardless of treatment. After S insulin ($p = 0.0019$) and glucose ($p = 0.0036$) were increased but serum aldosterone ($p = 0.0083$) and potassium ($p = 0.0285$) responses were lower. No differences were observed for plasma renin activity. Urine volume and kaliuresis were less after S ($p = 0.03$, $p = 0.03$). A 24% increase in metabolic rate ($p = 0.002$) and increased respiratory exchange ratio ($p = 0.02$) after S were observed. Systemic effects of sucrose ingestion following exercise include cardiovascular, renal, endocrine, and metabolic changes.

Abbreviations: BP = blood pressure, CHO = carbohydrate(s), DBP = diastolic blood pressure, FFA = free fatty acid(s), HR = heart rate, K = potassium, kcal = kilocalorie, Na = sodium, S = sucrose solution, SBP = systolic blood pressure, W = water

INTRODUCTION

The importance to athletes of a diet high in carbohydrates (CHO) is now well accepted. Ingestion of CHO-rich sources immediately following exercise and at 2-hour intervals following cessation of exercise has been shown to augment the replacement of muscle glycogen [1,2]. The effect of CHO ingestion on systems other than muscle glycogen resynthesis following exercise is less well known. Nevertheless, ample evidence exists to suggest that other systems that are recovering from the exercise stress are likely to be affected by CHO since they have been shown to be affected at rest. A single meal of glucose and sucrose were shown to acutely elevate systolic blood pressure (SBP) in young men while glucose, fructose, sucrose and lactose were also acutely antinatriuretic [3]. Furthermore, because of the effect of insulin on fat metabolism any feeding which elevates blood glucose suppresses free fatty acid (FFA) levels and inhibits the utilization of FFA as fuel [4,5].

Appreciating the heterogeneity of responses to the ingestion of CHO is of particular importance when considering the period of recovery following exercise. At the end of an exercise session levels of catecholamines, plasma renin activity and aldosterone are all elevated [6–8] reflecting the cardiovascular stress of exercise as well as the demands on the water-conserving and salt-conserving mechanisms of the kidney. Recovery SBP has been shown to be suppressed in both normotensive and hypertensive men [9]. At the end of nonexhaustive prolonged exercise, FFA levels are elevated [10] and CHO metabolism is depressed [11]. The use of CHO in this setting may well produce as yet unknown effects other than the desired enhancement of muscle glycogen replacement. The purpose of this study

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was to examine the effect of CHO ingestion on cardiovascular, hormonal, renal and metabolic recovery from exercise. Sucrose was chosen as the CHO because of its documented effects on blood glucose levels and antinatriuretic properties at rest.

METHODS

Subjects

Eight healthy, normotensive men (aged 29 ± 3 years) were tested. All participants gave verbal and written consent after the experimental procedures were explained. The study was approved by the Human Investigations Committee of Lenox Hill Hospital.

Determination of Maximal Oxygen Consumption

The volunteers reported to the laboratory on 3 nonconsecutive days over a 2–3 week period. During the first session percent body fat was estimated with tetrapolar bioelectrical impedance [12] (RJL Systems, Detroit, MI) and height and weight were measured without shoes. Subjects then performed an incremental test to exhaustion on a Fitron cycle ergometer (Lumex, Ronkonkoma, NY). Work was increased by 100 or 150 kpm each minute according to body size and athletic ability [13]. Oxygen consumption was measured using a SensorMedics 2900 metabolic cart (Sensor Medics, Yorba Linda, CA). This unit was calibrated before each test using a 1 l volumetric syringe with standardized gases. Corrections to gas volumes were made by an on-board computer for pressure and temperature. Heart rate (HR) was monitored throughout the test with 12 leads by a Sensormedics Horizon electrocardiogram (Sensor Medics, Yorba Linda, CA). Subjects were provided with an instruction sheet which listed foods to be avoided the 24 hours prior to the next 2 testing dates. This list included those foods containing caffeine, alcohol, and tyramine.

Testing Protocol

Subjects arrived at the laboratory for the second and third sessions at 8 a.m. after an overnight fast and were questioned to determine their adherence to the diet instructions. An 18 gauge Med Teflon intravenous cannula was inserted into the antecubital vein and was connected to a 500 ml solution of normal saline (0.9% NaCl). The volunteers then exercised for 1 hour on a bicycle ergometer at 70% HR reserve. This was calculated as:

$$\text{resting HR} + 0.7(\text{maximal HR} - \text{resting HR})$$

During both sessions HR was monitored continuously using a Uniq Pro Trainer, Model 8733 HR monitor (Computer Instruments, Hempstead, NY). Blood pressure (BP) was measured every 15 minutes using the standard auscultatory method.

Response to CHO Ingestion

Immediately following exercise subjects were given 1 l of water (W) or 1 l of water with 200 g of dissolved sucrose (S). Water contained 0.02 mEq/l potassium (K) and 0.2 mEq/l sodium (Na) (Puro Corp, Maspeth, NY). The order of beverage was alternated between subjects. Volunteers were instructed to void and were then seated in a reclining position. They were covered with a sheet during all measurements, and were instructed to remain as still as possible. The ambient temperature was maintained at 21–23°C during testing. No reading material, music or talking was permitted during metabolic measurements.

BP and HR measurements were made immediately after exercise and then every ½ hour for 2 hours during recovery. Blood samples were drawn at the end of exercise, and at ½, 1 and 2 hours postexercise by flushing the saline from the line with approximately 5–6 ml of blood which was then discarded. Urine was collected at the end of the 2-hour recovery period.

After calibrating with standardized gases, metabolic measurements were determined using the dilution method from 10 to 30, 40 to 60, and 100 to 120 minutes postexercise. Respiratory gas exchange and energy expenditure were measured continuously during the measurement periods with 60 second averaging using computerized open-circuit indirect calorimetry with a ventilated mask system (Sensor Medics 2900, SensorMedics, Yorba Linda, CA). Values more than two standard deviations from the mean were eliminated from the data for each measurement period. This was done to prevent the inclusion of measurements not representative of the resting condition, i.e., during sneezing or coughing.

Analytical Methods

Blood was distributed into tubes without anticoagulant for serum (serum separation tube, Bectin/Dickinson, Rutherford, NJ) and tubes treated with EDTA for plasma. After centrifuging for 10 minutes at 3000 rpm and separation, the samples were kept on ice until analysis could be performed. Serum glucose, K, and Na were analyzed using a Technicon SMAC II system. Serum insulin was analyzed with radioimmunoassay. Plasma, serum, and urine to be analyzed for renin, aldosterone and catecholamines were stored at −70°F until analysis could be completed. High-performance liquid chromatography with electrochemical detection was used to determine plasma and urinary catecholamine levels (Waters, Milford, MA). Renin was analyzed using the Renin-Angiotensin I method with single antibody technique (New England Nuclear,

Billerica, MA) and serum aldosterone determination was made using the solid phase antibody method (Coat-A-Count, Diagnostic Product Corp, Los Angeles, CA). Analysis of urinary nitrogen, K, Na, and creatinine was made with a Beckman Asta Eight Chemical Analyzer (Beckman, Los Angeles, CA).

Statistics

Comparisons between the two treatment conditions, S and W, were made by paired t-tests. Areas under the curve for all plasma and serum hormonal and electrolyte levels were computed by the method of trapezoids for the 2-hour recovery period and compared by paired t-tests. All values are displayed as mean ± SEM, and precise p values are indicated throughout in tables and graphs to facilitate interpretation of the data.

RESULTS

The eight men used in this study were 18–45 years old and both sedentary and active individuals as reflected by the range in maximal oxygen consumption (44–66 ml/kg/minute, Table 1). The subjects were either lean or of average body fatness (9–16%). None had any history of hypertension and all were normotensive at the time of the studies. The average caloric expenditure of the eight subjects during exercise was 702 ± 59 kcal, based on oxygen consumption at the steady state HR from the progressive exercise test.

Hemodynamic Response

All subjects successfully completed the hour of exercise and at the end of exercise demonstrated no differences in any of the hemodynamic variables measured [HR, SBP, diastolic blood pressure (DBP), mean BP and rate pressure product, Table 2]. HR was elevated throughout recovery following S ingestion. Mean BP was comparable between treatments during the 2 hours of recovery. This occurred by virtue of an increase in SBP (significantly during the first hour, $p < 0.04$) and a decrease in DBP (significantly during the last hour, $p < 0.04$) following sucrose ingestion. Rate pressure product following sucrose ingestion was elevated throughout the recovery period indicating increased myocardial oxygen demand. Average resting SBP before both exercise periods was 112 ± 1.4 mm Hg, and at the 2-hour recovery measurement during S and W treatments, 113 ± 3.3 and 106 ± 4.0, respectively. SBP at the end of recovery was significantly lower than SBP at rest for the W condition only, $p < 0.025$.

Table 1. Sample Descriptives

	Mean	SEM	Minimum	Maximum
Age	29.6	3.1	18	45
Height (cm)	178.0	1.5	173	187
Weight (kg)	74.5	2.7	69	93
% fat	12.9	0.9	9	16
LBM (kg)	64.9	2.2	59.6	79.0
VO_{2max} (O_2/kg/minute)	55.4	2.8	44	66

LBM = lean body mass.

Endocrine Response

Ingestion of sucrose following exercise resulted in dramatic increases in glucose and subsequent hyperinsulinemia (Fig. 1). The areas under the curves for the S and W treatments was significantly different for both glucose and insulin ($p = 0.0036$ and $p = 0.0019$, respectively).

The decline in plasma renin activity was nearly identical between treatments with a mean of 7.4 and 6.4 ng AI/ml/hour for S and W, respectively, at the end of exercise and 1.8 and 1.5 ng AI/ml/hour at 2 hours of recovery ($p = 0.83$ for areas under the curve). The decline in serum aldosterone paralleled the fall in serum K with more rapid declines in the case of S (Fig. 2, $p = 0.0083$ and $p = 0.0285$ for areas under the curve, respectively). Catecholamines declined at similar rates between treatments (Fig. 3, $p = 0.42$ and $p = 0.35$ for norepinephrine and epinephrine, respectively).

Renal Response

Sucrose ingestion was associated with an over 50% reduction in urine volume ($p = 0.03$) during the 2-hour period following exercise. Comparable sample collection is validated by noting similar creatinine excretion (Table 3, $p = 0.68$). K excretion was diminished by S ingestion ($p = 0.03$) and no significant differences were noted for Na excretion, although a trend ($p = 0.09$) was noted for increased Na excretion following S ingestion. No significant differences existed for catecholamine excretion, although norepinephrine tended to be greater following S ingestion ($p = 0.09$).

Metabolic Response

S ingestion was associated with an increased caloric expenditure at all 20-minute measurement intervals during recovery ($p = 0.002$ or less at all three intervals, Fig. 4). While significantly different, the ingestion of 800 kcal (200 g) in S resulted in a difference of no more than 40 kcal between the two treatments for the 2 hours of recovery.

The switch to primarily CHO metabolism following S ingestion is evident from the respiratory exchange ratio (Fig. 4). Respiratory exchange ratio continued to decline when W was ingested but was above or close to 0.9

Table 2. Hemodynamic Results

Heart rate

	End ex	0.5 hour	1 hour	1.5 hour	2 hour
Sucrose	144 ± 3	69 ± 3	69 ± 2	67 ± 2	65 ± 3
Water	145 ± 3	61 ± 2	59 ± 2	56 ± 2	55 ± 3
	p = 0.82	p = 0.003	p = 0.001	p = 0.004	p = 0.009

Mean blood pressure

	End ex	0.5 hour	1 hour	1.5 hour	2 hour
Sucrose	101.5 ± 2	85.3 ± 3	83.2 ± 2	82.4 ± 2	82.6 ± 2
Water	101.4 ± 2	82.1 ± 2	85.3 ± 2	83.5 ± 2	84.1 ± 2
	p = 0.916	p = 0.300	p = 0.172	p = 0.561	p = 0.407

Systolic blood pressure

	End ex	0.5 hour	1 hour	1.5 hour	2 hour
Sucrose	174 ± 4	115 ± 3	112 ± 3	112 ± 4	113 ± 3
Water	178 ± 4	105 ± 3	108 ± 2	106 ± 2	106 ± 2
	p = 0.33	p = 0.03	p = 0.04	p = 0.11	p = 0.06

Diastolic blood pressure

	End ex	0.5 hour	1 hour	1.5 hour	2 hour
Sucrose	66 ± 2	70 ± 4	68 ± 2	68 ± 2	68 ± 2
Water	63 ± 2	70 ± 3	74 ± 2	72 ± 3	73 ± 3
	p = 0.34	p = 0.86	p = 0.01	p = 0.03	p = 0.04

Rate pressure product (HR × SBP/100)

	End ex	0.5 hour	1 hour	1.5 hour	2 hour
Sucrose	251 ± 6	79 ± 3	78 ± 3	75 ± 3	73 ± 3
Water	256 ± 8	64 ± 2	64 ± 2	59 ± 2	59 ± 2
	p = 0.33	p = 0.001	p = 0.001	p = 0.000	p = 0.002

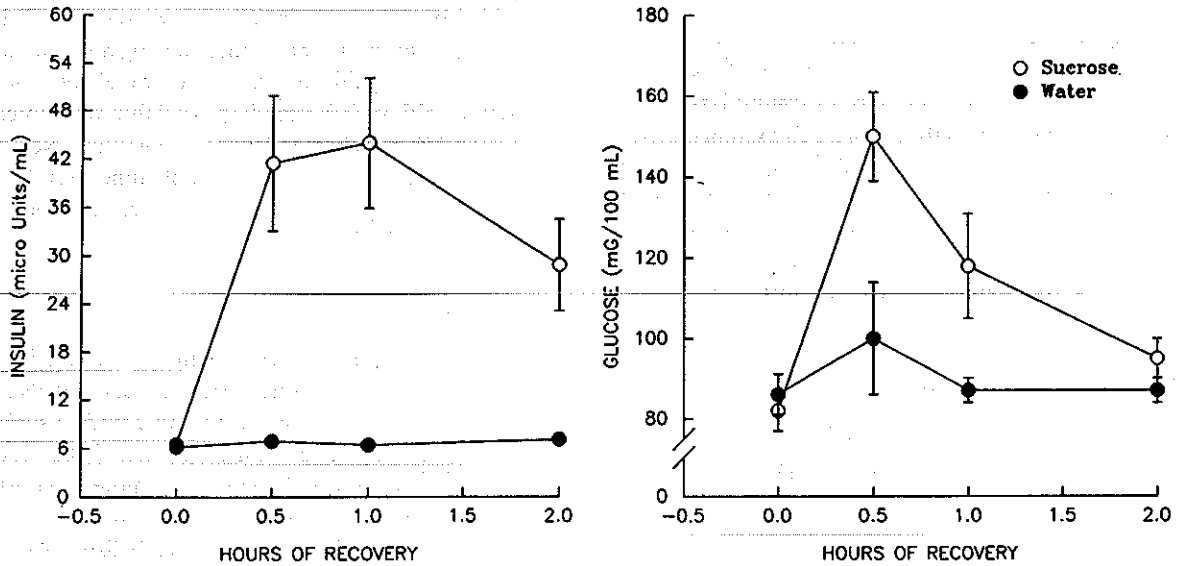

Fig. 1. Effect of sucrose or water ingestion on recovery levels of insulin and glucose. Areas under the curves for insulin and glucose were significantly greater in the sucrose condition, p = 0.0019 and p = 0.0036, respectively.

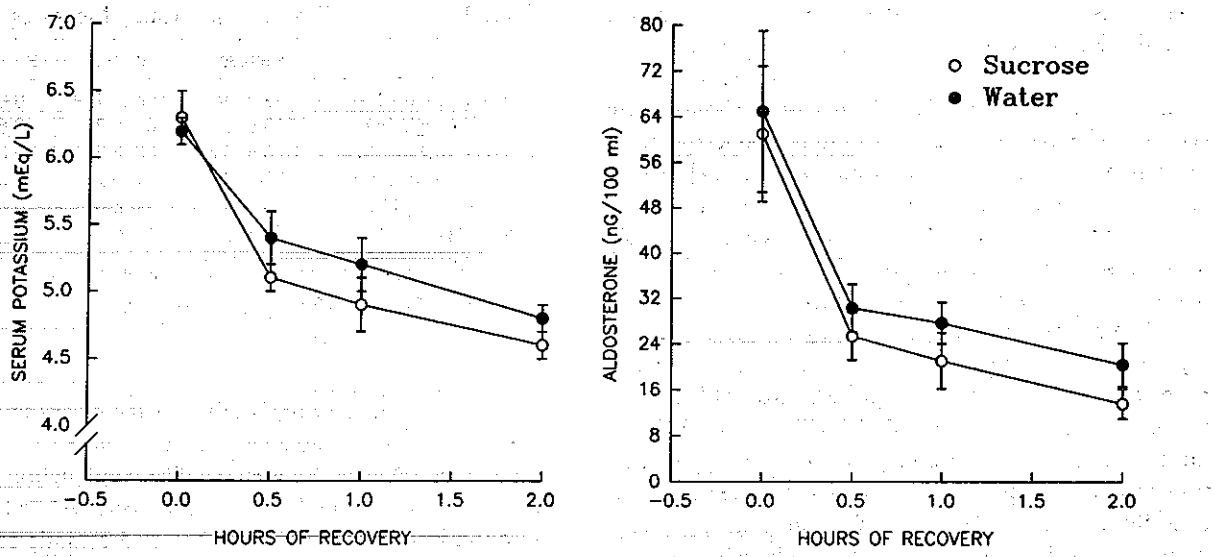

Fig. 2. Effect of sucrose or water ingestion on recovery levels of potassium and aldosterone. Areas under the curves for potassium and aldosterone were significantly less in the sucrose condition, $p = 0.0285$ and $p = 0.0083$, respectively.

following S ingestion, reflecting the decrease in fat metabolism.

DISCUSSION

Hemodynamic Response

Significantly higher HR were obtained during recovery following sucrose ingestion. The net effect on mean BP was not different, although SBP was higher and DBP lower following S ingestion. The elevation of SBP following S ingestion is similar to that obtained previously in fasted, resting normotensive male subjects following S or fructose ingestion [3]. The present observations might be explained by an S-induced fall in total peripheral resistance and an increase in cardiac output maintaining mean arterial pressure.

Ingestion of a mixed meal at rest increases cardiac output by up to 1.5 l/minute, with most of this flow directed to the splanchnic region [14]. In addition, glucose has been shown to stimulate hepatic blood flow within 30 minutes [15]. During exercise, digestion has the effect of increasing cardiac output to permit perfusion of the splanchnic circulation [16]. The effect we observed on indirect indices of cardiac output and total peripheral resistance is consistent with similar augmentation of flow to the splanchnic region following CHO ingestion during recovery from exercise. The effect was evident 30 minutes following ingestion and sustained for the 2-hour period of observation. SBP at 2 hours of recovery was significantly lower (6 mm Hg, $p < 0.025$) than resting SBP for the W condition but not the S condition. Consequently, the decline in SBP following exercise [9] appears to be attenuated during S ingestion.

Endocrine Response

It is well known that exercise of sufficient intensity or duration inhibits pancreatic insulin release [17], that insulin can remain low for periods following exercise [18], and that blood glucose levels can be elevated dramatically following exercise, presumably by an epinephrine response in highly trained athletes [19]. The low levels of insulin evident at the end of exercise in the present study remained low throughout the recovery period following W ingestion. A modest increase in glucose at 30 minutes of recovery was also noted. Not unexpectedly, the ingestion of 200 g of S dramatically elevated blood glucose and insulin levels (Fig. 1). The insulin levels were still elevated at 2 hours of recovery and blood glucose had returned to near end exercise levels. It is likely that not all of the S had been absorbed by the intestine. Previous studies of gastric emptying with glucose using 400 ml of solution predict that 2.1 kcal/min will be delivered regardless of solution concentration [20]. Davis et al demonstrated that absorption of glucose from the intestine decreased in solutions of concentrations >15% [21]. From these studies we estimate that <500 ml would have emptied from the stomach. Therefore, we would predict that insulin levels would remain elevated for periods well beyond 2 hours to match the rate of glucose absorption.

Elevations in aldosterone levels at the end of exercise have been noted previously [7] and these elevations are

likely to last for some time following exercise [8]. Multiple stimuli exist for aldosterone release by the adrenal cortex including elevations in the levels of angiotensin II and serum K [22,23]. The effect of increasing plasma renin activity has previously been shown to be dissociable from increasing aldosterone levels during exercise [24]. Serum K was clearly elevated at the end of exercise in our study (Fig. 2), but both K and aldosterone declined more rapidly in the case of S ingestion. No differences were noted in plasma renin activity between treatments.

K homeostasis is vital to normal physiological functioning and multiple regulatory mechanisms exist for this purpose. Adrenergic beta$_2$-stimulation promotes cellular uptake of K [25], an effect that has been shown to be important during and following exercise [26,27]. In addition, insulin and glucose are known to promote the uptake of K by muscle and liver [28]. We found no significant differences in the venous levels of norepinephrine or epinephrine. It is likely that the more rapid decline in K following S ingestion in our study was caused by the combined effects of insulin and glucose which subsequently resulted in the observed decline in serum aldosterone.

Renal Responses

Urine volume was over 50% less in the 2 hours following the ingestion of S, while creatinine excretion was comparable (Table 3). Renal K excretion was significantly less following S ingestion with a trend toward increased Na excretion. No measurement of plasma or urinary osmolality was made in this study, but the effects on urine volume may be explainable in part by increased plasma osmolality following S ingestion. The combination of increased levels of blood glucose and decreased water absorption in the gut due to the increased osmotic content (20% CHO) would have served to increase plasma osmolality in the case of S by comparison to W. Vasopressin levels are known to be elevated, promoting W reabsorption by the kidney, following exercise of sufficient intensity [29] and return to near baseline levels by 75 minutes postexercise [23]. Increases in plasma osmolality are associated with increases in the levels of vasopressin [34] and the normalization of plasma osmolality during recovery is associated with the decline in vasopressin. It might be postulated that S ingestion delays the recovery of plasma osmolality, and results in prolonged elevation of vasopressin levels but the exact mechanism for decreased urine volume following S ingestion awaits additional measures of osmolality and vasopressin.

Table 3. Two-Hour Postexercise Urinary Measures

	Sucrose	Water	P value
Volume (ml/2 hr)	259 ± 36	596 ± 119	0.03
$U_{Cr}V$ (mg/2 hr)	213 ± 26	206 ± 26	0.68
$U_{Na}V$ (mEq/2 hr)	18.6 ± 3.6	14.0 ± 2.7	0.09
U_KV (mEq/2 hr)	10.6 ± 2.1	18.1 ± 2.9	0.03
NE (ng/mg Cr)	58 ± 8	39 ± 6	0.08
Epi (ng/mg Cr)	27 ± 10	16 ± 2	0.40

$U_{Cr}V$ = creatinine excretion; $U_{Na}V$ = sodium excretion; U_KV = potassium excretion; NE = norepinephrine; Epi = epinephrine.

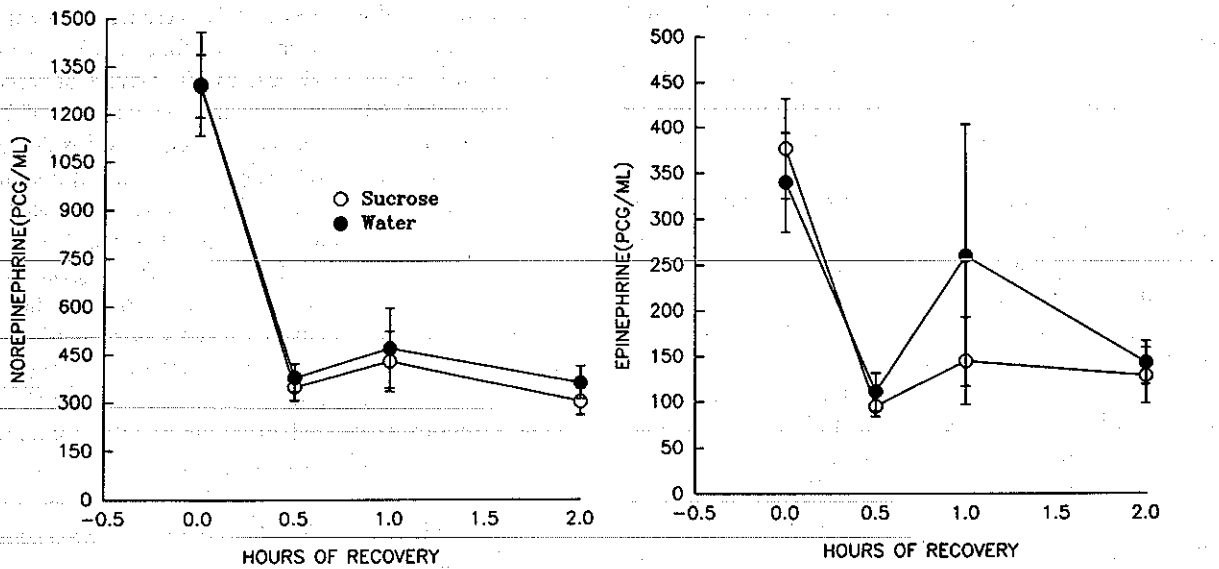

Fig. 3. Effect of sucrose or water ingestion on recovery levels of catecholamines. Areas under the curves for norepinephrine and epinephrine were not significantly different, $p = 0.42$ and $p = 0.35$, respectively.

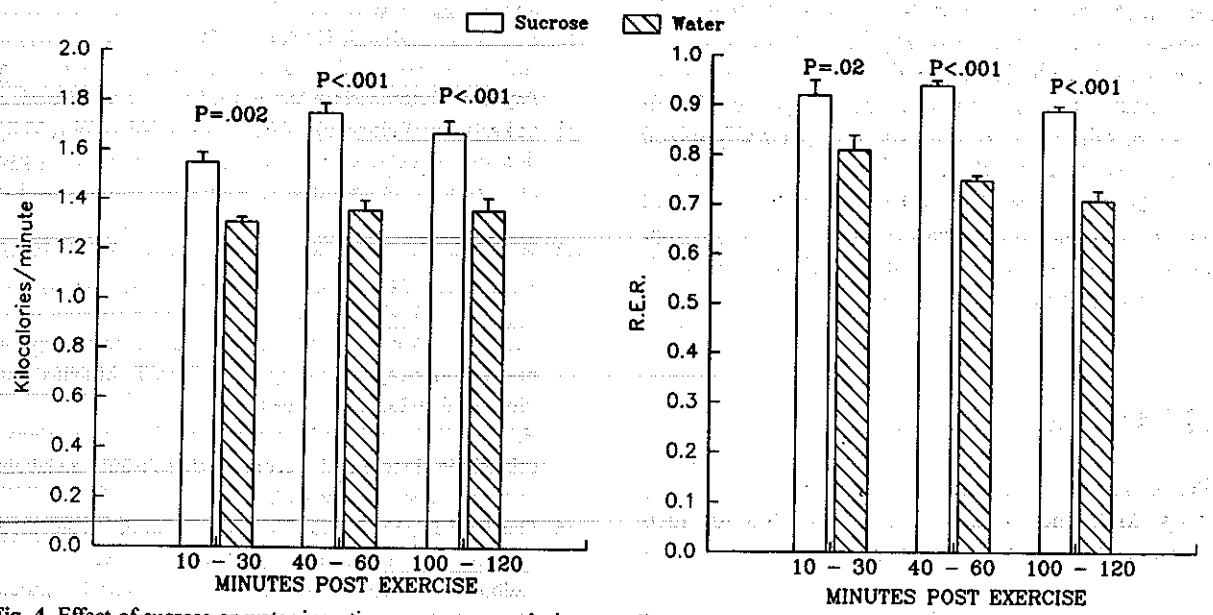

Fig. 4. Effect of sucrose or water ingestion on recovery calorie expenditure and respiratory exchange ratio. Individual bars represent 20-minute collection periods.

Increased K excretion is consistent with the known effects of aldosterone on the kidney [31]. We feel that the observed differences in excretion of K between the treatments is likely associated with the observed differences in the rate of decline in aldosterone. The more rapid decline in serum aldosterone following S could explain the significantly lower kaliuresis. Notably we did not see an antinatriuretic effect of S as reported previously in resting, fasted men [3], further suggesting that alternate mechanisms are at work during exercise recovery.

Unlike previous chronic studies in rats [33], we were unable to show that CHO ingestion acutely affects renal epinephrine and norepinephrine excretion in humans following exercise. The venous plasma results for norepinephrine and epinephrine were also not significantly different between treatments, suggesting that sympathetic nervous system recovery was comparable between treatments.

Metabolic Responses

It has previously been shown during recovery that hyperinsulinemia, with reported levels higher than those obtained in our study, enhances the metabolic rate [34]. Mikines et al also showed that lipid oxidation decreased at levels of hyperinsulinemia comparable to ours, resulting in elevations of the respiratory exchange ratio [34]. Our ability to detect increased metabolic rate at lower levels of hyperinsulinemia may have been due to the flow through ventilation system used.

Over the 2-hour recovery period, the increase in metabolic rate induced by S ingestion was about 24% higher than that observed with W ingestion. Previous investigations in resting man have demonstrated a 14% increase following 50 g of glucose ingestion [35] and 18% following a mixed meal of 755 kcal in trained men [36]. The increased metabolic rate induced by the ingestion of 800 kcal of S in the present study accounted for an additional caloric expenditure of about 40 kcal over W ingestion. The increased respiratory exchange ratio noted in our study also indicates a reversal of primarily fat metabolism to CHO metabolism.

CONCLUSIONS

Ingestion of large amounts of S as opposed to W immediately following exercise has diverse effects on many systems in the body. The present study indicates that HR and SBP remained elevated for longer periods of time, although sympathetic nervous system activity, as indicated from plasma and urinary catecholamine measures, appeared to recover at the same rate as purely W ingestion. No differences were noted for plasma renin activity, although serum K and aldosterone levels decreased more in the S condition. In addition, lower urine volume and K excretion were evident following S ingestion. Finally, in-

creased recovery caloric expenditure and decreased fat metabolism were evident following S ingestion.

It is unknown whether or not other forms of CHO have similar effects or whether there is a dose-response effect to S ingestion. Further, since ours was an athletically mixed and normotensive population of men, it is unknown whether or not fitness, hypertension or gender have an effect. A thorough understanding of the effects of food ingestion following exercise seems prudent and awaits further investigation.

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