

Exercise-Induced Hyponatremia in Ultradistance Triathletes Is Caused By Inappropriate Fluid Retention

*Dale B. Speedy, MBChB, MSc, †Ian R. Rogers, MBBS, ‡Timothy D. Noakes, MBChB, MD,
§Susan Wright, MBChB, §John M. D. Thompson, PhD, ¶Robert Campbell, MBChB,
¶¶Ien Helleman, MSc, ¶¶Nicholas E. Kimber, MSc, **D. Ross Boswell, MBChB, PhD,
††Jonathan A. Kuttner, MBChB, and ‡‡Shameem Safih, MBChB

*Department of General Practice and Primary Care, University of Auckland, Auckland, New Zealand; †Emergency Department, Sir Charles Gairdner Hospital, Nedlands, Perth, Western Australia, Australia; ‡Sports Science Institute of South Africa, University of Capetown, Capetown, South Africa; §University of Auckland, Auckland; ¶Sportsmed, Christchurch; ¶¶Lincoln University, Lincoln; **Diagnostic Laboratory, Auckland; ††Waiuku Medical Practice, Waiuku; and ‡‡Auckland Hospital Emergency Department, Auckland, New Zealand

Objective: To study fluid and sodium balance during overnight recovery following an ultradistance triathlon in hyponatremic athletes compared with normonatremic controls.

Case Control Study: Prospective descriptive study.

Setting: 1997 New Zealand Ironman Triathlon (3.8 Km swim, 180 Km cycle, 42.2 Km run).

Participants: Seven athletes ("subjects") hospitalized with hyponatremia (median sodium [Na] = 128 mmol L⁻¹). Data were compared with measurements from 11 normonatremic race finishers ("controls") (median sodium = 141 mmol L⁻¹).

Interventions: None.

Main Outcome Measures: Athletes were weighed prior to, immediately after, and on the morning after, the race. Blood was drawn for sodium, hemoglobin, and hematocrit immediately after the race and the following morning. Plasma concentrations of arginine-vasopressin (AVP) were also measured post race.

Results: Subjects were significantly smaller than controls

(62.5 vs. 72.0 Kg) and lost less weight during the race than controls (median -0.5% vs. -3.9%, $p = 0.002$) but more weight than controls during recovery (-4.4% vs. -0.8%, $p = 0.002$). Subjects excreted a median fluid excess during recovery (1,346 ml); controls had a median fluid deficit (521 ml) ($p = 0.009$). Estimated median sodium deficit was the same in subjects and controls (88 vs. 38 mmol L⁻¹, $p = 0.25$). Median AVP was significantly lower in subjects than in controls. Plasma volume fell during recovery in subjects (-5.9%, $p = 0.016$) but rose in controls (0.76%, $p = \text{NS}$).

Conclusions: Triathletes with symptomatic hyponatremia following very prolonged exercise have abnormal fluid retention including an increased extracellular volume, but without evidence for large sodium losses. Such fluid retention is not associated with elevated plasma AVP concentrations.

Key Words: Ultra-distance—Triathlon—Arginine-vasopressin—Ironman triathlon—Sodium.

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INTRODUCTION

Hyponatremia (plasma sodium concentration <135 mmol L⁻¹) is a relatively recently described complication of ultradistance running and triathlon events. First described in 1985¹ and 1986,² the condition may occur more frequently than previously appreciated. Thus hyponatremia of varying degrees of severity has been reported to occur in up to 29% of athletes in the Hawaiian Ironman Triathlon,^{3,4} in 18% of finishers in the New Zealand Ironman Triathlon,⁵ and in 0.3–10% of competitors in ultradistance running events.^{6–8}

Despite the frequency of this condition, its pathogen-

esis remains uncertain. The bulk of current evidence suggests that athletes with this condition ingest fluid at unusually high rates during prolonged exercise and retain fluid inappropriately.^{1,9–13} However most of the evidence for this hypothesis comes from retrospective studies involving relatively small subject numbers.^{1,2,9,12–16} Indeed there have been only three prospective studies of the relationship between exercise-related changes in plasma sodium concentrations and in body weight, the latter an indirect measure of fluid balance, in competitive ultradistance sporting events.^{5,11,12} All have found an inverse relationship between changes in body weight and in plasma sodium concentrations after Ironman triathlons, further suggesting that the hyponatremia of exercise is caused by inappropriate fluid retention. In contrast, dehydration causes hyper-, not hyponatremia, as incorrectly proposed by other researchers.¹⁷

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Address correspondence and reprint requests to Dale Speedy, MBChB, 179A Hill Rd., Manurewa, New Zealand. E-mail: dalespeedy@e3.net.nz

An alternative method for determining the postrace fluid status of athletes with exercise-induced hyponatremia is to measure fluid and electrolyte balance during recovery. Using this technique, Irving et al.¹⁰ studied eight subjects who developed severe symptomatic hyponatremia during the 90 Km Comrades Marathon footrace. These data were compared with historical results measured in athletes who had completed similar ultramarathon footraces while maintaining normal serum sodium concentrations. As the hyponatremic athletes excreted a fluid excess during recovery, while their plasma volumes increased, those authors concluded that the hyponatremia was caused by retention of the excess fluid in a site other than the extracellular compartment, perhaps in a physiological third space, most likely as a volume of unabsorbed fluid in the intestinal lumen.^{18–20} Osmotic movement of sodium ions from the extracellular fluid into the unabsorbed fluid in the intestinal lumen would cause the hyponatremia.

However there are two concerns with this hypothesis. First, the volumes of unabsorbed fluid in the intestine required to produce hyponatremia according to this model appear to be unrealistically large. Thus Noakes¹⁹ has calculated that up to 4 L of unabsorbed fluid in the intestine would be necessary to produce the low serum sodium concentrations reported by Irving et al.¹⁰ It seems unlikely that so much fluid could be present in either the small or large bowels without causing significant gastrointestinal symptoms especially in athletes participating in competitive sporting events. Second, we have recently shown a linear relationship between the postrace plasma sodium concentration and the hematocrit in a large group of competitors in the New Zealand Ironman Triathlon.⁵ This indicates that the hyponatremia is caused, at least in part, by an expansion of the extracellular space, causing a dilutional hyponatremia. In addition, two athletes who developed severe hyponatremia in the same race had very large (16–25%) postrace increases in plasma volume.¹⁵

Accordingly, the aim of this study was to evaluate fluid and electrolyte balance during recovery in a group of hyponatremic athletes admitted to hospital after the 1997 New Zealand Ironman ultradistance triathlon, an event in which there is a high incidence of hyponatremia.^{5,12,13,15} Results from these athletes were compared with those from a group of normonatremic athletes competing in the same race and who were studied prospectively. We reasoned that a control group from the same race, studied prospectively, would be preferable to the use of controls from other races, as in the only similar previous study.¹⁰ In addition, careful attention was paid to detect the likely location of any fluid imbalance and any possible role of the plasma hormones known to affect fluid and electrolyte balance: arginine-vasopressin (AVP) and aldosterone.

METHODS

Ethical approval for this study was obtained from the North Health Ethics Committee, and written informed

consent was obtained from subjects and controls prior to participation in the study.

The study was conducted on seven athletes admitted to the Auckland Hospital Emergency Department for management of symptomatic exercise-associated hyponatremia after the 1997 New Zealand Ironman Triathlon. Their data were compared with measurements from 11 normonatremic athletes (“controls”) who completed the same race and who participated in a prospective study of fluid balance and plasma sodium concentrations in a cohort of 600 athletes participating in that race, as described previously.⁵ The New Zealand Ironman Triathlon involved a 3.8 Km sea swim, 180 Km cycle, and 42.2 Km run. Ambient air temperature at 1200 hours on race day was 21°C, with a relative humidity of 91%. Water temperature was 20.7°C.

All the participants in the trial were weighed at race registration 2 days before the race, and then immediately after completing the race. Those (“subjects”) who developed symptomatic hyponatremia during the race and who were admitted to the Emergency Department were weighed again on discharge from hospital at 0700 hours on the morning after the race. The controls, who completed the same race with normal plasma sodium concentrations, were weighed approximately 1 hour before the race, immediately after completing the race, and on the following morning at 0800 hours. All weights were measured on a hard level surface with calibrated Seca scales (Hamburg, Germany).

On finishing the race, all subjects had a blood sample drawn by venipuncture from the antecubital fossa while reclining in the supine position. Sampling was performed in the medical tent on admission of the athlete to the facility and was repeated on discharge from the Emergency Department at 0700 hours the following morning. Sampling was again performed in the supine position after the subjects had lain in bed overnight.

Subjects admitted to the Emergency Department were treated according to a predetermined protocol. In particular, intravenous fluid was withheld unless there was clear clinical evidence of dehydration or hypovolemia. A record of fluid and sodium balance was begun when subjects arrived at the medical tent and continued in the Emergency Department until 0700 hours the following morning. Oral and intravenous fluid intake was measured. Urine volume was measured on a fluid balance sheet in the Emergency Department and was measured again electronically in the laboratory; the greater of the two volumes was used as the measure of urine output. Volume, but not the sodium content, of vomitus was measured. All urine collected was analyzed for sodium concentration.

Control athletes consisted of eight male and three female athletes who were part of a separate investigation to determine fluid intake during the same Ironman triathlon. They were recruited from all race entrants on the basis that selected athletes lived in Auckland where the race was held. Controls estimated their expected race finishing time to be between 10.5 and 13 hours. Of the 20 athletes initially in the control group, 2 male and 3 fe-

male athletes developed hyponatremia during the race, requiring exclusion from the control group. Accurate dietary data were not available on a further four control athletes who were accordingly also excluded.

Controls were issued with the appropriate vessels and instructed to collect all their urine from the time they finished the race until 0700 hours the next morning. Volume and sodium concentrations were measured for all urine collections, using routine methods. Fluid ingested from immediately after the race until the next morning was also recorded. Controls were given preprepared food to eat after the race and for breakfast the next morning—the sodium and water contents of which were known. Any additional food consumed by the controls was also recorded. Reported food intake during this period was analyzed using food composition tables.¹⁸ Controls had blood drawn by venipuncture in the sitting position on completion of the race and at 0800 hours the following morning.

Immediate postrace measurement of plasma sodium concentrations in the subjects was performed in the medical tent at the end of the race using a Nova Ion Selective Electrode analyzer (Waltham, MA, U.S.A.) on lithium-heparin anticoagulated samples. Postrace and recovery plasma sodium assays on controls were carried out the day after the race with Hitachi 747 or 737 analyzers (Boehringer Mannheim, Mannheim, Germany), using standard methods and the manufacturer's reagents, on plasma that had been collected and stored at 4°C after centrifugation within 1 hour of collection. Routine hematological assays for hemoglobin (Hb) and hematocrit (Hct) were performed the following day with Technicon H1, H2, or H3 analyzers (Technicon Corporation, Tarrytown, NY, U.S.A.) on EDTA-anticoagulated samples that had been stored at 4°C. Relative plasma volume changes were calculated using the formula derived from the equations of Dill and Costill.^{19,20} For specimens collected during hospital admission, routine assays were performed by the same methods on fresh blood specimens.

Subjects and controls also had specimens collected by venipuncture after the race for measurement of plasma AVP and aldosterone concentrations. These venipunctures were performed on completion of the race on the controls, and following diagnosis of hyponatremia in the subjects when they attended for medical treatment. These assays were performed on EDTA-anticoagulated plasma that was obtained by centrifugation, frozen on dry ice within 1 hour of venipuncture, and stored frozen at -20°C or below until thawed for assay. Specimens were assayed for AVP concentration [AVP] according to the method described by Inder et al.²¹ Assays for aldosterone concentration [Aldo] were performed according to the method described by Lun et al.²² Reference ranges (resting) for [AVP] were 0–9 pmol L⁻¹, and for [Aldo] are 100–800 pmol L⁻¹. Reference ranges for these hormones immediately after competing in an ultradistance triathlon are not known.

The fluid deficit or excess was calculated by subtracting the total volume of urine output and of vomitus from

the total fluid input (oral and intravenous) during the recovery period. An unexpected finding was that the subjects were discharged from hospital clinically recovered but before most had restored normal plasma [Na]. This invalidated attempts to accurately measure the sodium deficits in these subjects. Accordingly, in calculating the sodium balance during recovery in subjects, we assumed that any remaining reduction in the plasma [Na] was due to a remaining sodium deficit in the extracellular space. This assumption appeared valid as fluid balance appeared to be equally altered in both subjects and controls at the termination of the trial; both groups had reduced their prerace weights by approximately 3 Kg at the completion of the study, suggesting that any remaining fluid imbalance was equal in both groups. The volume of the extracellular space was estimated at 0.183 L Kg⁻¹ body weight (e.g., 11 L in a 60 Kg subject). The estimated sodium deficit/excess (column 11, Table 2) was therefore calculated as the remaining sodium deficit/excess required to return the serum [Na] concentration to the median recovery value measured in control subjects; namely, 139.5 mmol L⁻¹ minus the measured [Na] on discharge, all multiplied by the assumed extracellular fluid volume, estimated as described above (column 10, Table 2). To this was added the measured sodium balance during recovery and which comprised sodium losses in urine (column 9, Table 2) and sodium ingested or infused (column 8, in Table 2). The sodium content of vomitus was not measured. The same calculations were done for the controls.

Statistical analysis between the groups was performed with nonparametric Wilcoxon sign rank tests. Changes in Hb and Hct within the group were tested by means of a paired Student *t*-test.

RESULTS

The control subjects had a median age of 36 years (range, 23–49 years) and median body weight of 72.0 Kg (range, 63–93.5 Kg). All controls finished the race, with a median race time of 12.01 hours (range, 10.87–14.37 hours). The median duration of the recovery period studied in the control group was 12.95 hours (range, 9.63–14.13 hours). Median time from race finish to postrace venipuncture for the control group was 13.5 minutes (range, 7–22 minutes).

Fourteen athletes were transferred to the local hospital with a diagnosis of hyponatremia of exercise. Two of these athletes were admitted directly to the intensive care unit and were excluded from the analysis. One was profoundly hyponatremic (Na = 116 mmol L⁻¹) with grand mal seizures.¹³ His recovery required 4 days—too long for the overnight period of this investigation. The other athlete had mild hyponatremia (Na = 132 mmol L⁻¹) and low output cardiac failure of uncertain etiology requiring inotropic support. The remaining 12 athletes were admitted to the Emergency Department. Four of these athletes were only mildly affected and were discharged within several hours of arrival, and were therefore not included in this study. An accurate dietary rec-

ord of food and fluid intake during the immediate postrace period was not available on one athlete admitted to the Emergency Department overnight, and he was therefore excluded from the study. The median weight change of the seven athletes who were excluded from the trial for the above reasons was -1.8 Kg (range, -3.8 to $+3.2$ Kg), with a median postrace serum sodium concentration of 132 mmol L⁻¹ (range, 116 – 134 mmol L⁻¹).

Data from the remaining seven hyponatremic athletes were therefore analyzed. Two subjects were male and five were female with a median age of 36 years (range, 30–47 years) and a median body weight of 62.5 Kg (range, 54.0–68.5 Kg). Subjects were significantly lighter than controls (62.5 vs. 72.0 Kg, $p = 0.016$). Five subjects completed the triathlon with a median race time of 12.25 hours (range, 11–13.3 hours); two subjects discontinued the race after the bike leg (race times of 9.8 and 7.7 hours), as they were too ill to complete the run. Median time from race finish to subjects arrival in the medical tent was 6 minutes (range, 3–80 minutes). Median time from race finish to the first postrace venipuncture in the medical tent was 20 minutes (range, 12–100 minutes). The median time from race finish to venipuncture for AVP assay was 46 minutes (range, 40–90 minutes). The median duration of the recovery period studied in the subject group was 11.58 hours (range, 10.17–13.00 hours).

Common presenting symptoms included light-headedness, altered mental status, nausea and vomiting, exhaustion, and muscle cramps. Subjects 3 and 4 (Tables 1 and 2) were considered by the attending physicians to be dehydrated and were treated initially with intravenous fluids, receiving 2.90 L and 0.94 L of 0.9% saline, respectively.

All subjects were discharged from hospital the morn-

ing after the race, clinically well and asymptomatic, although four subjects were still mildly hyponatremic with [Na] of 128–131 mmol L⁻¹.

Data on absolute and relative weight changes during the study period for both subjects and controls are provided in Table 1. Compared with controls, hyponatremic subjects lost significantly less absolute weight (median, -0.3 vs. -3.0 Kg; $p = 0.003$) and relative weight (median, -0.5% vs. -3.9% ; $p = 0.002$) during the race. Conversely, hyponatremic subjects lost significantly more weight in the immediate postrace recovery period (median, -2.7 Kg or -4.4% body weight vs. -0.5 Kg or -0.8% body weight, $p = 0.002$). However there was no significant difference in total weight loss from pretrace to recovery in subjects (median, -2.5 Kg or -4.6% body weight) compared with controls (median, -3.0 Kg or -4.2% body weight).

Further data on fluid and electrolyte balance during recovery in the two groups are shown in Table 2. Median postrace plasma [Na] was significantly lower in the hyponatremic subjects (128 mmol L⁻¹ vs. 141 mmol L⁻¹, $p = 0.0005$). Plasma [Na] in the hyponatremic subjects rose significantly from a mean of 127.4 ± 1.8 to 132.1 ± 1.5 mmol L⁻¹ during recovery ($p = 0.009$). In contrast, the plasma [Na] of the controls fell slightly from 140.9 ± 0.6 to 139.9 ± 0.7 mmol L⁻¹ during recovery ($p = \text{NS}$).

Median total fluid intake in the subjects was 500 ml, with a median urine output of 2,231 ml. Two subjects vomited during recovery (510 and 1,200 ml for Subjects 3 and 4, respectively). Median total fluid intake in the controls was 1,646 ml, with a median urine output of 1,150 ml. As a result, the hyponatremic subjects excreted a median fluid excess of 1,346 ml during recovery, whereas controls had a median fluid deficit of 521 ml

TABLE 1. Relative body weight changes during the race and in recovery in subjects and controls

	Prerace weight (Kg)	Postrace weight (Kg)	Recovery weight (Kg)	Weight change: pre- to postrace (Kg)	% Weight change: pre- to postrace	Weight change: postrace to recovery (Kg)	% Weight change: postrace to recovery	Weight change: prerace to recovery (Kg)	% Weight change: prerace to recovery
Hyponatremic subjects									
1	54.0	54.2	51.5	0.2	0.4	-2.7	-5.0	-2.5	-4.6
2	58.0	57.7	53.0	-0.3	-0.5	-4.7	-8.1	-5.0	-8.6
3	64.0	66.2	65.0	2.2	3.5	-1.2	-1.9	1.0	1.6
4	68.5	67.2	66.0	-1.3	-1.9	-1.2	-1.8	-2.5	-3.6
5	54.0	52.7	49.5	-1.3	-2.4	-3.2	-6.0	-4.5	-8.3
6	70.0	67.7	66.0	-2.3	-3.3	-1.7	-2.5	-4.0	-5.7
7	62.5	62.7	60.0	0.2	0.4	-2.7	-4.4	-2.5	-4.0
Median	62.5	62.7	60.0	-0.3	-0.5	-2.7	-4.4	-2.5	-4.6
Normonatremic controls									
1	77.0	73.5	73.5	-3.5	-4.5	0	0	-3.5	-4.5
2	77.0	74.0	74.5	-3.0	-3.9	0.5	0.6	-2.5	-3.2
3	83.0	80.0	81.0	-3.0	-3.6	1.0	1.2	-2.0	-2.4
4	77.0	73.5	74.5	-3.5	-4.5	1.0	1.3	-2.5	-3.2
5	65.0	63.0	62.0	-2.0	-3.1	-1.0	-1.5	-3.0	-4.6
6	93.5	90.0	89.0	-3.5	-3.7	-1.0	-1.1	-4.5	-4.8
7	66.0	62.0	61.0	-4.0	-6.1	-1.0	-1.5	-5.0	-7.6
8	64.0	61.5	60.0	-2.5	-3.9	-1.5	-2.3	-4.0	-6.3
9	63.0	61.0	60.5	-2.0	-3.2	-0.5	-0.8	-2.5	-4.0
10	72.0	69.0	69.0	-3.0	-4.2	0	0	-3.0	-4.2
11	66.0	65.0	63.5	-1.0	-1.5	-1.5	-2.3	-2.5	-3.8
Median	72.0	69.0	69.0	-3.0	-3.9	-0.5	-0.8	-3.0	-4.2

TABLE 2. Fluid and electrolyte balance during recovery

	Postrace [Na] (mmol L ⁻¹) 1	Recovery [Na] (mmol L ⁻¹) 2	Total fluid intake (ml) 3	Total urine output (ml) 4	Total vomit (ml) 5	Urine [Na] (mmol/L) 6	Fluid excess (ml) 7	Total Na intake (mmol) 8	Total urinary Na losses (mmol) 9	Calculated residual Na balance (mmol) 10	Estimated Na balance during recovery (mmol) 11
Hyponatremic subjects											
1	130	138	0	3,700	0	5	+3,700	0	15	-15	0
2	119	128	500	4,363	0	9	+3,863	5	39	-122	-88
3	124	131	3,450	2,288	510	10	-652	450	23	-100	-527
4	128	130	1,315	551	1,200	44	+436	148	24	-119	-243
5	128	135	60	2,231	0	18	+2,171	0	40	-44	-4
6	134	135	520	1,344	0	9	+824	5	12	-58	-51
7	129	128	150	1,496	0	26	+1,346	2	39	-132	-95
Median	128	131	500	2,231	855	10	+1,346	5	24	-100	-88
Normonatremic controls											
1	143	140	2,110	1,111		33	-999	60	37	7	-15
2	142	142	1,646	795		73	-851	25	58	35	69
3	140	136	1,467	448		31	-1,019	40	14	-53	-79
4	142	139	2,696	428		79	-2,268	132	34	-7	-105
5	141	139	1,561	2,379		5	818	68	12	-6	-62
6	143	142	2,767	2,749		-	-18	-	-	-	-
7	143	143	1,551	1,324		56	-227	40	74	42	76
8	138	139	1,202	1,930		-	728	-	-	-	-
9	141	-	2,558	1,500		-	-1,058	-	-	-	-
10	140	141	1,671	1,150		-	-521	-	-	-	-
11	137	138	558	1,127		13	569	35	15	-18	-38
Median	141	139.5	1,646	1,150		33	-521	40	34	-6	-38

during recovery; this difference was statistically significant ($p = 0.009$).

Median sodium intake in recovery for subjects was 5 mmol. Median urine sodium concentration was low in subjects (10 mmol L^{-1}), with median total urinary sodium losses of 24 mmol. Median sodium intake in recovery for controls was 40 mmol. Median urine sodium concentration in controls was 33 mmol L^{-1} , with median total urinary sodium losses of 34 mmol. The calculated residual sodium deficit in subjects was significantly greater in subjects than controls (100 mmol vs. 6 mmol, $p = 0.007$). As a result, hyponatremic subjects had a median estimated sodium deficit of 88 mmol compared with an estimated deficit of 38 mmol in controls; this difference was not statistically significant ($p = 0.25$).

Two hyponatremic subjects (Subjects 3 and 4) had apparently large sodium deficits (527 and 243 mmol, respectively). However these deficits likely developed during the postrace recovery period, as both athletes vomited during recovery and the sodium content of their vomitus was not recorded. Thus the measured sodium deficits in these two subjects probably overestimate the actual sodium deficits incurred during the race. None of the control subjects developed such a large sodium deficit.

Hematocrit rose significantly in the hyponatremic subjects from a mean (\pm SEM) of $39.7 \pm 1.5\%$ postrace to $42.3 \pm 1.6\%$ during recovery ($p = 0.004$). Hematocrit remained essentially unchanged at $42.4 \pm 0.7\%$ postrace and $42.3 \pm 0.5\%$ during recovery in the controls ($p = \text{NS}$). As a result hyponatremic subjects had a significant fall in calculated mean plasma volume during recovery ($5.9 \pm 1.87\%$, $p = 0.016$), whereas plasma volume rose by $0.76 \pm 1.68\%$ ($p = \text{NS}$) during recovery in the controls.

Plasma [AVP] and [Aldo] were measured in 5 subjects

and in all 11 controls after the race. The median [AVP] concentration in the subjects was 1.6 pmol L^{-1} (range, $1.2\text{--}3.8 \text{ pmol L}^{-1}$) and was significantly lower than in the controls (4.6 pmol L^{-1} ; range, $1.3\text{--}40.0 \text{ pmol L}^{-1}$) ($p = 0.02$). The median postrace [Aldo] in subjects was 1258 pmol L^{-1} (range, $416\text{--}2,225 \text{ pmol L}^{-1}$) and did not differ significantly from the controls ($1,593 \text{ pmol L}^{-1}$; range, $455\text{--}4,136 \text{ pmol L}^{-1}$).

DISCUSSION

Hyponatremia is currently both the most common^{3-8,11,17} and the most serious^{1,5,10,12-14,16} complication of very prolonged, competitive endurance events. Only one previous study has documented fluid balance during recovery in athletes admitted to hospital with severe, symptomatic hyponatremia following an ultradistance sporting event.¹⁰ That study found that the hyponatremia was due to excessive fluid retention, as symptomatic athletes excreted a median excess of 3.04 L during recovery. Their sodium deficits were, however, not greater than those estimated to occur in athletes who did not develop hyponatremia during other ultramarathon races. The latter interpretation is compatible with the finding of an inverse relationship between postrace plasma sodium concentrations and body weight changes during ultradistance endurance events.^{5,11,12}

Thus the first important finding of this study was that hyponatremic subjects lost significantly less weight during the race than did controls (-0.5% vs. -3.9% change in body weight). In addition, hyponatremic subjects also lost significantly more weight than controls during the recovery period (-4.4% vs. -0.8%). Thus, overall, the group of athletes who developed symptomatic hyponatremia finished the ultratriathlon with a median fluid excess of 1.35 L compared with a median fluid deficit of

0.52 L in the control group, who maintained their plasma sodium concentrations during prolonged exercise.

The hyponatremic subjects were released from hospital with only one having reached a normal plasma sodium concentration; the median for the complete group was only 131 mmol L⁻¹ on discharge. Hence, the degree of fluid overload or of sodium deficit may have been underestimated in the total group. However, weight changes from prerace to recovery were similar in both subjects and controls (-2.5 vs. -3.0 Kg) suggesting that fluid balance may have been achieved equally during recovery in both groups. The possible reasons why all athletes were approximately 3 Kg lighter the day after the race than they were before the race are addressed subsequently.

Thus these results agree with the findings of Irving et al.¹⁰ who also reported that eight subjects with severe symptomatic hyponatremia excreted a fluid excess of between 1.22 to 5.92 L during recovery. It should also be noted that subjects in that study¹⁰ were considerably more ill than the subjects in this study and had substantially lower median postrace plasma sodium concentrations (124 vs. 128 mmol L⁻¹). Thus, if fluid overload alone causes the hyponatremia of exercise, it is logical that the values for fluid overload in the subjects in this study would be less than in the more severely hyponatremic subjects studied by Irving et al.¹⁰ Furthermore subjects in that study¹⁰ were released from hospital only when their plasma sodium concentrations had normalized, so that any fluid excess was more likely to have been recorded completely.

The second important contribution of this study is to show that this abnormal fluid balance was not associated with elevated plasma [AVP] or [Aldo] in the postrace period in contrast to the suggestion of others that these hormones may play a role in the etiology of this condition.^{10,14,23} Indeed, plasma [AVP] were significantly lower in subjects than in the normonatremic controls. To the best of our knowledge, there are no published data on the response of AVP to ultradistance exercise. We acknowledge that the delay (median, 46 minutes) between race finish and sampling for AVP in our subjects could mean that the AVP concentrations we measured may not have been representative of concentrations during actual racing. (The half-life of AVP has been reported as 24 minutes by Baumann et al.²⁴). However, measurements during racing in hyponatremic athletes would be impossible in the field situation. Thus our data do not support the postulate that inappropriately high plasma [AVP] are necessary for the development of the hyponatremia of exercise.

Similarly, we found no evidence that plasma [Aldo] contributed to the condition as these concentrations, although elevated after the race, were no higher in subjects than in controls. It is therefore probable that other chemicals, as yet unidentified, must contribute to the abnormal fluid retention in these subjects whose renal function is otherwise completely intact.¹⁰

The third finding of this study was that the estimated sodium deficit that developed during the race was not

significantly different in subjects and controls even though very variable sodium deficits were estimated especially in the subjects. Hyponatremic subjects had an estimated median sodium deficit of 88 mmol, whereas the estimated median deficit in controls was 38 mmol. The large sodium deficits recorded in Subjects 3 and 4 did not take into account the sodium content of the vomitus, hence these measures overestimate the sodium deficit in these two subjects. Subject 3 was given 2.9 L of intravenous normal saline for a clinical diagnosis of dehydration, despite gaining 2.2 Kg during the race. The data on her fluid and sodium balance during recovery suggested that she was still fluid overloaded at discharge from hospital, as she was still 1 Kg heavier at discharge than before the race. Hence these calculations overestimate her total sodium deficit. It is notable that her delayed recovery may likely have been caused by the injudicious use of intravenous fluids in a condition of fluid overload.

Accordingly these data are similar to those of Irving et al.¹⁰ who reported that the mean sodium deficit in hyponatremic ultramarathon runners was 153 mmol, which was not different from a mean value of 182 mmol in ultramarathon runners who completed other ultramarathon races with normal serum sodium concentrations. The reason why the estimated sodium losses in the controls was substantially less than the values measured in the ultramarathon runners in the study of Irving et al.¹⁰ is especially surprising given the longer duration of this triathlon than of the ultramarathon. This suggests that the sodium intakes in the races may have been different or that the sweat and urine sodium losses are less in the triathletes than in the runners. Salt water ingestion during the sea swim is one factor unique to the triathlon, and may have attenuated the triathletes sodium deficits.

The fourth important finding was that the hyponatremic subjects in this study showed a significant rise in hematocrit and fall in plasma volume during recovery, whereas these parameters remained essentially unchanged in the controls. These changes indicate that the hyponatremic athletes had an elevated plasma volume when they finished the race, hence a portion of the excess fluid in these athletes was retained in the extracellular space.

This finding agrees with other case reports that have described increased plasma volumes or lower hematocrits in hyponatremic athletes,^{8,9,15} suggesting that the hyponatremia results from expansion of the extracellular space. In contrast, Irving et al. reported an apparent 24% decrease in plasma volume in athletes who developed hyponatremia during an 88 Km foot race,²¹ prompting the hypothesis that their hyponatremia was due to the effects of a physiological third space.²⁵⁻²⁷ It is possible that differences in gastrointestinal fluid absorption between ultradistance running as compared with ultradistance triathlon might account for this different response in plasma volume. Nevertheless, the results of the current prospective study strongly support extracellular space fluid retention in the hyponatremic athletes, which would account for their significantly smaller weight change

during the race, their postrace diuresis with sodium retention, and subsequent contraction of plasma volume. We postulate that there must also have been an increase in the intracellular volume in association with this increase in extracellular volume; this postulate is consistent with the changes in cerebral function that usually occur with severe and symptomatic hyponatremia.

Finally, our data also provide information regarding the extent of the body weight losses that are independent of the fluid changes due principally to sweating. Surprisingly, despite a minimum median fluid excess of 1.3 L in the hyponatremic runners, the median weight change over the race in the group was -0.3 Kg. Thus, in this group, it would appear that a minimum weight loss of 1.6 Kg ($1.3 + 0.3$ Kg) resulted from sources other than a pure fluid loss. Similarly the controls showed a median fluid deficit of only 0.52 L but a total weight change of -3.0 Kg during the race. Hence they apparently lost approximately 2.5 Kg of weight from sources other than fluid.

Furthermore, both hyponatremic subjects and normonatremic controls lost similar amounts of weight from prerace to recovery, further suggesting that a significant amount of weight is lost from sources other than fluid losses in sweat and urine during very prolonged exercise. Sources of such weight loss include loss of fat, glycogen, and water stored with glycogen.²⁸ The corollary of these observations is that a neutral weight change or a minimal weight loss over an event of the Ironman distance may actually indicate overhydration of up to 2–3 Kg.

In summary, this study confirms that subjects with symptomatic hyponatremia develop a fluid excess during exercise that is associated with a measured increase in the plasma volume and a presumed increase in both extracellular and intracellular volumes; the latter assumption is based on the observed changes in cerebral function characteristic of the condition. The hormonal basis for this positive fluid balance was not established, but there was no evidence of elevated plasma [AVP] or [Aldo] measured after the race. Our data also demonstrate that 1.5–2.5 Kg of weight loss at an Ironman triathlon is independent of fluid loss and is likely due to metabolic fuel use and to changes in whole body water content associated with glycogen storage.

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