

The Incidence, Risk Factors, and Clinical Manifestations of Hyponatremia in Marathon Runners

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Objective: To report on the incidence, identify the risk factors, and clarify the clinical manifestations of acute hyponatremia in marathon runners.

Design: An observational and retrospective case-controlled series.

Setting: The medical care area of the 2000 Houston Marathon.

Patients: Marathon finishers treated in medical area receiving intravenous fluids (N=55), including a more detailed analysis of 39 runners completing a retrospective questionnaire.

Main Outcome Measures: Vital signs, serum electrolytes, and finish time were analyzed via ANOVA studies between all non-hyponatremic (NH: N=34) and hyponatremic (H: N=21) runners. Fluid intake, training variables, NSAID use, and Symptomatology were further analyzed to delineate all significant differences between groups.

Results: There were no significant differences in vital signs, training variables, or NSAID use between H and NH groups, although there was a trend towards the less experienced runners presenting with lower post-race sodium levels. H runners had

lower potassium [K] ($p=.04$), chloride [Cl] ($p<.001$), and blood urea nitrogen [BUN] ($p=.004$) levels than NH runners. There was a significant inverse linear relationship between both finish time versus [Na] ($r^2=.51$) and total amount of fluid ingested versus [Na] ($r^2=.39$). The total cups of water ($p=.004$), electrolyte/carbohydrate solution ($p=.005$) and total amount of fluid ingested ($p<.001$) were significantly higher in H compared to NH runners and the degree of hyponatremia was related in a dose dependant manner. Vomiting was observed more frequently in H than NH runners ($p=.03$).

Conclusion: 21 runners presented to the medical area of the Houston Marathon with hyponatremia (.31% of entrants). Excessive fluid consumption and longer finishing times were the primary risk factors for developing this condition. Vomiting was the only clinical sign differentiating hyponatremia from other conditions that induce exercise-associated collapse.

Key Words: Hyponatremia—Marathon runner—Hydration—Electrolyte imbalance.

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INTRODUCTION

The incidence of hyponatremia in endurance athletes participating in ultradistance running events and triathlons has been well documented over the past fifteen years.^{1–5} Prior to the year 2000, there had only been one reported case of hyponatremia in a marathon runner.⁶ Reports of this rare condition have been increasing over the past decade in standard 42K marathon runners and three deaths have been confirmed (Table 1).^{7–9}

The Houston Marathon medical team became alerted to the development of hyponatremia in marathon runners after four participants in the 1999 race presented to the medical area with hyponatremia: each requiring critical care and hospitalization. Being more attuned to this entity, serum electrolytes were subsequently measured in runners presenting to the major medical area of the medical tent in the year 2000. The purpose of this report is to present the incidence of acute hyponatremia in the 2000 Houston Marathon, and to further identify risk factors

and clinical signs that aid in the prevention and early detection of this life-threatening illness.

METHODS

For the observational portion of the study, data were collected on 73 runners treated in the major medical facility of the 2000 Houston Marathon. 55 of these 73 runners were unable to tolerate oral fluids, had unstable vital signs and/or altered mental status and were considered candidates for IV fluid resuscitation. All athletes who were given IV access had their serum electrolytes immediately checked using a portable I-STAT™ electrolyte measuring device and were monitored closely by a nurse (1:2 ratio) and a physician (1:4 ratio) until the runner either stabilized or a decision was made by the medical director to transport the participant to a local hospital.

To further determine the risk factors for hyponatremia, a retrospective analysis was subsequently performed. A post-race questionnaire, approved by the Baylor College of Medicine Institutional Review Board, was mailed out within ten days after completion of the marathon to 68 of

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TABLE 1. Documented cases of hyponatremia in marathon runners

Documented cases of hyponatremia in marathons (+)	
Big Sur	1993—1 death with [Na] under 117 mmol/L
Chicago	1998—1 death
Dallas	1996—1 case
Honolulu	1998—1 case with Na 118 mmol/L
Houston	1999—4 cases requiring critical care
Boston	1999—2 cases in slower runners, 1 death 2002
Twin Cities	1997—1 case
*San Diego	1998/1999—26 cases
Questionable Hyponatremia	
Walt Disney/Orlando	2000—1 hospitalized overnight with an “electrolyte imbalance”
San Francisco	1999—1 hospitalized “in a coma” for 1½ days

* The San Diego Marathon data was reported from their recent publication (8).

+ Data obtained from direct communication with marathon directors via questionnaires and telephone conversation.

the 73 runners treated in the major medical facility. Addresses were not available for five of the 73 marathoners. The questionnaire focused on fluid intake, training preparation for the marathon, symptomatology during and after completion of the event and NSAID use. 17 of 21 runners classified as hyponatremic (serum sodium levels at or below 135mmol/L) returned the questionnaire, while 22 of the 47 participants classified as non-hyponatremic “controls” completed the medical questionnaire. Non-responders in both the control and hyponatremia populations were not significantly different with reference to gender, age or vital signs from those who completed the questionnaire.

The hyponatremic group was further divided into subgroups for more detailed statistical analysis: Group A represented those runners presenting with mild hyponatremia (Na 135–130mmol/L). Group B included those with moderate hyponatremia (Na 129–121mmol/L). Group C represented those runners presenting with critical hyponatremia (Na 120mmol/L and below).

All continuous data were analyzed using one-sample t-tests to assess possible significance between the hyponatremic and non-hyponatremic groups. One-way analysis of variance (ANOVA) tests were further utilized to look for significant variations and possible trends between the controls and the mild, moderate and critical hyponatremic subsets.

All ordinal data pertaining to symptomatology and NSAID use were analyzed via Pearson chi-squared tests. Results were obtained comparing both the control versus the hyponatremic group as well as between hyponatremic subsets.

Medical complaints such as dizziness, lightheadedness, nausea, vomiting, headache, and puffiness that were observed and recorded by medical personnel in the medical tent of the 2000 Houston Marathon were also analyzed via chi-squared equations.

RESULTS

6660 runners entered the 2000 Houston Marathon. 5082 participants completed the course within the “official” time limit of five and one-half hours (5:30) and 34.4% of these finishers were women. Mean finishing times were 4:24 for males and 4:45 for females. The conditions for the marathon were unseasonably warm and humid: identical conditions to those experienced in the 1999 Houston Marathon. The race began at 8:00am with a temperature of 16.6°C (62°F) and 93% humidity. Peak temperature of 25°C (77°F) with 44% humidity was reached at noon. Water and Powerade™ were available to runners at aid stations located at each mile along the race course.

237 marathon participants were seen in the medical area. 73 of these were treated in the major medical tent with 21 diagnosed with symptomatic hyponatremia. Of the 55 runners who received intravenous fluids, 34 runners had serum sodium levels above 135mmol/L (controls), eight runners had serum sodium levels between 130-135mmol/L (mild hyponatremia), eleven runners were documented with serum sodium levels between 120-129mmol/L (moderate hyponatremia) and two female runners had serum sodium levels below

TABLE 2. Vital signs for hyponatremia and non-hyponatremic (control) runners

Groups	Systolic blood pressure (mmHg)	Diastolic blood pressure (mmHg)	Heart rate (bpm)	Temperature °F
Control (Na >135 mmol/L)	108 ± 19	69 ± 11	96 ± 21	97 ± 2
Mild (Na 130–135 mmol/L)	108 ± 19	69 ± 12	93 ± 18	96 ± 1
Moderate (Na 120–129 mmol/L)	120 ± 10	70 ± 9	81 ± 12	96 ± 2
Critical (Na <120 mmol/L)	134 ± 8	90 ± 0	85 ± 24	97 ± 0
p-value (ANOVA)	0.06	0.32	0.16	0.61

TABLE 3. Serum electrolytes for hyponatremic and non-hyponatremic (control) runners

Groups	Na mmol/L	K mmol/L	Cl mmol/L	BUN mg/dL	Glucose mg/dL
Control (Na >135 mmol/L)	140 ± 2	4.3 ± .4	102 ± 3	19 ± 4	110 ± 24
Mild (Na 130–135 mmol/L)	133 ± 2	4.4 ± .4	94 ± 3	17 ± 5	119 ± 23
Moderate (Na 120–129 mmol/L)	127 ± 2	3.9 ± .6	86 ± 3	14 ± 2	92 ± 16
Critical (Na <120 mmol/L)	119 ± 1	3.7 ± .5	79 ± 1	12 ± 3	126 ± 40
p-value (ANOVA)	<0.001	0.035	<0.001	0.004	0.05

120mmol/L (critical hyponatremia). The incidence of all total males and females who finished the race and were diagnosed in the medical tent with hyponatremia were 3.6 and 5.1 cases per thousand runners, respectively.

There were no significant differences in vital signs noted between the hyponatremic and non-hyponatremic (control) runners. However there was a trend toward those hyponatremic runners with the lowest serum sodium levels to present with higher initial systolic blood pressures (Table 2).

Sodium [Na], potassium [K], chloride [Cl], and blood urea nitrogen [BUN] levels were all significantly lower in the hyponatremic versus the non-hyponatremic runners. The progressive decline in these serum values were directly related to the severity of the hyponatremia (Table 3).

Hyponatremic athletes had longer finishing times than non-hyponatremic runners. Those runners who spent the longest time on the race course presented to the medical area with lower serum sodium levels ($p < .001$) (Table 4). There was a significant inverse linear correlation between serum sodium levels and finishing times ($r^2 = .51$) (Figure 1). Those runners finishing faster than 4:20 had a mean [Na] of 140.4mmol/L (average male [Na] 140.1mmol/L and average female [Na] 140.8mmol/L). Those runners who ran slower than 4:20 had a mean [Na] of 133.6mmol/L (average male [Na] 135.3mmol/L and average female [Na] 131.8mmol/L).

The 39 respondents to the medical questionnaire were evenly matched in size, age, and gender (Table 5). Hyponatremic subgroups suggest that hyponatremia occurs in equal frequency between men and women, with the two most serious cases developing in females (Table 6).

The hyponatremic group drank significantly more total cups of water ($p = .004$), electrolyte/carbohydrate solution ($p = .005$), and total amount of fluid ($p < .001$) than the control group. This was further exemplified via the ANOVA studies which showed significant differences noted between the mild, moderate, and critical classifi-

cations of the hyponatremic marathon runners in the number of cups of water ($p = .008$), electrolyte/carbohydrate beverage ($p = .011$) and total fluid intake ($p = .002$) documented by each subject. The total cups of fluid consumed and the severity of hyponatremia was dose dependant (Figure 2). There was a significant inverse linear relationship between the amount of fluid ingested and serum sodium levels ($r^2 = .39$) (Figure 3).

There was little difference between gender and fluid consumption that would suggest a behavioral difference in the drinking patterns of men versus women (Table 7).

There were no statistically significant differences noted between the hyponatremic and control groups in relationship to training histories as illustrated in Table 8. There was however, a notable trend suggesting that the more inexperienced runners were more prone toward developing hyponatremia. Fifty-three percent of the hyponatremic runners trained with one large (1000+) training program that catered to beginning runners while only 27% of the nonhyponatremic runners trained with this particular group ($p = .03$).

NSAIDS were utilized in equal frequency between the hyponatremic (29%) and non-hyponatremic control group (23%) during the race ($p = .36$). In the preceding two weeks before the marathon, 47% of the hyponatremic and 32% of the controls consumed NSAID products ($p = .14$).

Symptoms documented by medical personnel in the medical area revealed a significant difference in the incidence of vomiting between groups. The hyponatremic runners presented with vomiting more frequently than non-hyponatremic runners ($p = .03$) and those with moderate to critical serum sodium levels vomited signifi-

TABLE 4. Finish times for hyponatremic and non-hyponatremic (control) runners

Groups	Average finishing times (hours:minutes)
Control (Na >135 mmol/L)	4:10 ± 41
Mild (Na 130–135 mmol/L)	4:56 ± 35
Moderate (Na 120–129 mmol/L)	5:05 ± 23
Critical (Na <120 mmol/L)	5:43 ± 5
p-value (ANOVA)	<0.001

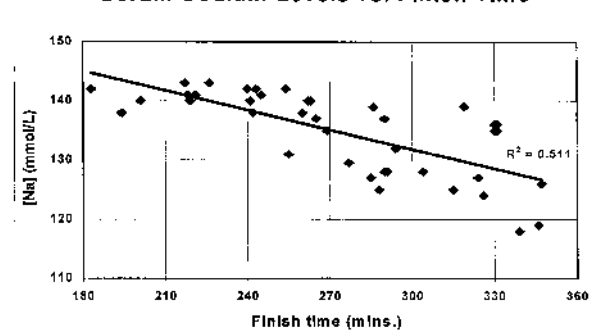
Serum Sodium Levels vs. Finish Time**FIG. 1.** Linear relationship between post race serum sodium levels and finish time.

TABLE 5. Respondent demographics in the hyponatremic and non-hyponatremic groups

	Hyponatremic		Non-hyponatremic (Control)	
	Male	Female	Male	Female
Subject	8	9	12	10
Age	35	37	43	38

cantly more than those with mild hyponatremia ($p = .03$) (Table 9).

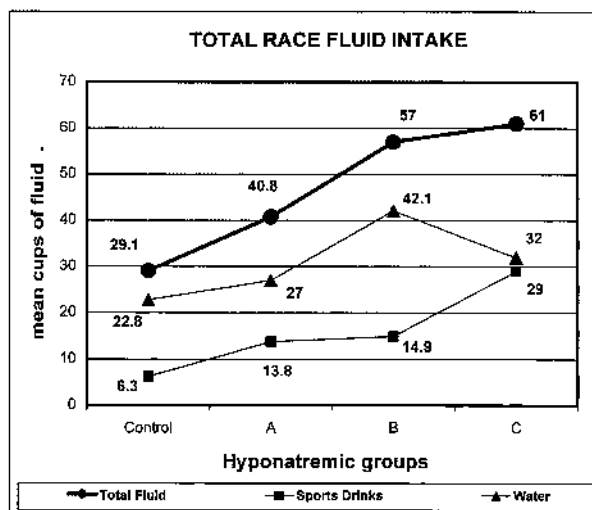
DISCUSSION

Twenty-one runners participating in the 2000 Houston Marathon were diagnosed with hyponatremia within the medical tent. This represents 9% of the total number of runners seeking medical care after the race with an incidence of 0.31% of all marathon entrants. This is the highest reported incidence of hyponatremia to date in a single 42K marathon, as cases documenting this serious illness are being recognized at an alarming rate. Davis et al reported on 26 cases of hyponatremia that presented to the emergency room from the San Diego Marathon in 1998 and 1999. They documented 21 cases out of 19,978 runners, an incidence of 0.11%, in 1998 alone.⁸ Hsieh et al. reported an incidence of hyponatremia in 5.6% of all athletes seeking medical attention after the 2000 Pittsburgh Marathon.⁹ Before these studies, hyponatremia was considered to be a rare and singular occurrence in marathon runners and clinical suspicion had been low. The high incidence of hyponatremia found in Houston Marathon runners may be attributed to the fact that this entity was suspected and electrolytes were rapidly measured in runners not responding to supportive management. Conversely, hyponatremia was not suspected in runners after the San Diego marathon and these very ill runners presented directly to the emergency room long after the race.

The past decade has revealed a new fitness phenomenon aptly referred to as the "second running boom". Previously sedentary individuals are targeting marathons as their ultimate fitness challenge and beginning marathon programs are enthusiastically accommodating this new trend. The new generation of slower, older, and previously unfit individuals are often being guided by fellow novice runner and rapidly changing the face of

TABLE 6. Respondent sub-classification of hyponatremic groups

Groups	Hyponatremic classification	N (male)	N (female)
A	Mild hyponatremia (Na 130–135 mmol/L)	3	1
B	Moderate hyponatremia (Na 120–129 mmol/L)	5	6
C	Severe hyponatremia (Na <120 mmol/L)	0	2

**FIG. 2.** Average fluid intake of hyponatremic and non-hyponatremic (control) groups.

marathon running while bringing a new generation of medical concerns into focus.

The hyponatremic runners in this study were largely a product of this new trend as 53% of these runners trained with a beginning marathon program. Hyponatremic athletes were slower than non-hyponatremic runners and there was an inverse linear relationship found between lower serum sodium levels and longer finishing times. When hyponatremic sub-groups were further evaluated, the runners who spent the longest time on the course presented to the medical tent with the lowest serum sodium levels. Time of presentation to the emergency room and the severity of hyponatremia were also significantly related in the Davis study, as those runners who presented to the hospital later in the day also had lower serum sodium levels.⁸

On further analysis, the highest incidence of hyponatremia was seen in runners finishing after 4:20. The

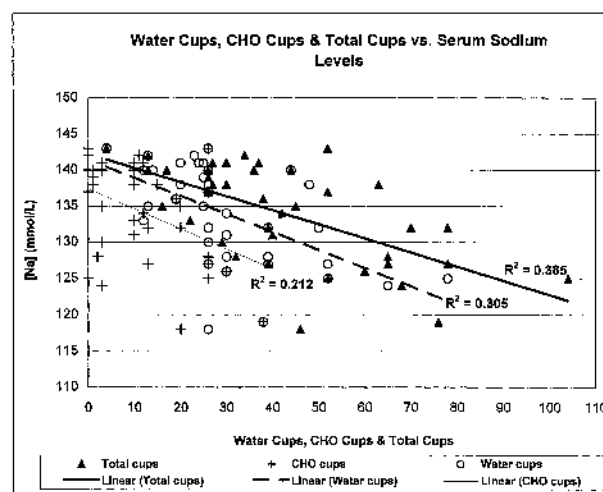
**FIG. 3.** Linear relationships between post-race serum sodium concentrations and amount of fluid consumed during the race.

TABLE 7. Respondent reported fluid intake in the hyponatremic and non-hyponatremic males and females

	Hyponatremic		Non-hyponatremic (Control)	
	Male	Female	Male	Female
Total fluid (cups)	49	57	28	31
Water (Cups)	36	38	22	24
Electrolyte (cups)	13	19	6	7

average serum sodium level for athletes presenting to the major medical area of the medical tent with finishing times beyond 260 minutes was 133.6mmol/L. Male finishers have a mean [Na] of 135.3, just beyond the clinically defined cut-off value for hyponatremia of 135mmol/L, whereas female finishers beyond 4:20 have a mean [Na] of 131.8. Although not statistically significant this data would tend to indicate that females might have a tendency toward more severe hyponatremia than males. The average finishing times for 2000 Houston Marathon finishers were over 4:20 for both male (4:24) and female (4:45) athletes, however, stressing the need for increased vigilance for the clinical suspicion of hyponatremia in slower runners.

Barr and Costill stated in 1986 that "symptomatic hyponatremia is clearly unexpected in events of marathon distance" based on precise calculations predicting hourly fluid and sodium losses through energy expenditure and heat of water vaporization at differing running speeds and body weights. They concluded that the amount of fluid necessary to dilute plasma serum sodium concentrations to produce clinical hyponatremia were "inconceivable" in a 26.2 mile event.¹⁰ The data presented in this study challenges this notion and is representative of other reports on ultraendurance runners, triathletes, military personnel, hikers, and most recently in marathon runners; all of which strongly suggest that fluid overload is the main determinant for the development of hyponatremia.^{1-5,8,11-17}

Our data shows a direct inverse relationship between the total amount of fluid consumed and post race serum sodium concentration. The athletes who drank the most fluid along the marathon course presented to the medical area with the lowest serum sodium levels. The hyponatremic runners consumed almost twice as much fluid as the non-hyponatremic control group and when categorized into groups delineating the severity of hyponatremia, the amount of total fluids ingested and the degree of hyponatremia were related in a dose dependent manner.

In striking contrast, eight well-conditioned runners showed increases in post race serum sodium concentrations with total fluid intakes of less than 500ml during the Boston Marathon.¹⁸ Thus, when fluid intake is voluntarily restricted over the marathon distance, hyponatremia does not occur.^{2,11}

The progressive fall in the serum values of potassium and chloride in conjunction with the fall in serum sodium levels can also be attributed to a dilutional effect on these electrolytes, further supporting the theory of fluid overload in the development of hyponatremia due to the expansion of the extracellular fluid volume (ECF). Blood urea nitrogen levels also declined with the progressive fall in serum sodium values, also suggestive of a dose dependency between hydration status and degree of hyponatremia.

There was a trend in those runners with the lowest serum sodium levels to show slight increases in systolic blood pressure, but it was not statistically significant in the ANOVA studies. This trend also supports the theory of fluid overload as increases in plasma volume as a direct relationship to whole body fluid overload can present with subtle increases in blood pressure. The Cushing reaction of increased arterial pressure as a direct response to cerebral edema and central nervous system ischemia may also explain this steady rise in blood pressure, which would also be secondary to a fluid overloaded state.

Training practices of those runners developing hyponatremia versus those athletes treated in the medical area for associated causes of exercise-associated collapse were not significantly different. There was a trend noted however, indicating that those runners who were less experienced in marathon running had an increased susceptibility for becoming hyponatremic even though it was not statistically significant.

There is widespread interest in the possible role that nonsteroidal anti-inflammatory agents play in inducing chronic hyponatremia. This theory is largely based on a few case studies. Since prostaglandins are natriuretic – antagonizing arginine vasopressin to result in water diuresis–inhibition via anti-inflammatory agents may potentiate anti-diuretic hormone and decrease free water excretion. Cases of this occurrence have been documented in two populations distinct from marathoners: neonates treated with indomethacin for patent ductus arteriosus and elderly adults with other diseases that might impair urinary dilution.¹⁹⁻²² It is generally thought that "hyponatremia is a rare complication of nonsteroidal

TABLE 8. Respondent training histories of the hyponatremic and non-hyponatremic control groups

	Hyponatremic	Non-hyponatremic	P-values (t-test)
Number of years running?	8 ± 6	12 ± 10	0.13
Number of months training for the marathon?	4 ± 2	6 ± 5	0.14
Number of marathons completed?	3 ± 2	5 ± 6	0.19
Longest run prior to the marathon (miles)?	16 ± 7	20 ± 9	0.13
Number of weeks long run preceded the marathon?	3 ± 3	5 ± 6	0.24

TABLE 9. Documented symptoms of hyponatremic and non-hyponatremic runners

Symptoms	Hyponatremic N (%)	Non-hyponatremic N (%)	P-value
Headache	4 (24%)	3 (14%)	0.35
Puffiness	4 (24%)	2 (9%)	0.21
Nausea	10 (59%)	11 (50%)	0.33
Dizziness	7 (41%)	12 (55%)	0.37
Vomiting	10 (59%)	6 (27%)	0.03
Lightheadedness	6 (35%)	4 (18%)	0.17

anti-inflammatory drugs in adults, with additional factors necessary to develop water intoxication.”¹⁹

Our data does not support a relationship between hyponatremia and NSAID use in marathon runners, despite contrary findings by Ayus et al.⁷ and Davis et al.⁸ Hyponatremic and non-hyponatremic controls consumed NSAID products during the race with equal frequency (29% vs. 23%). Further inquiry into the type and amount of NSAIDs consumed by both groups also revealed that even if anti-inflammatories were consumed during or prior to the marathon, more than half of the dosages consumed were less than a regular prescription dosage and not likely to cause significant renal alterations.

On analysis of documented clinical signs taken from the actual medical records of the runners treated in the medical tent, the incidence of vomiting was significantly higher in the hyponatremic runners versus the runners in the control population. The vomiting of clear fluids would be expected in cases of fluid overload and not likely to be found in runners who are dehydrated. When the body is overloaded with fluid, unabsorbed fluid is likely to be sequestered into the gut and any opportunity the body has to expel this unnecessary fluid—where intake has exceeded both sweat rate as well as maximum rate of urine production—would be considered a natural defense mechanism and protective physiological response. The vomiting may be a reflex action in response to the increasing distention of the large and unnecessary amount of fluid within the gastrointestinal tract. Or, this vomiting may be triggered by the central nervous system in response to the hyponatremic encephalopathy that concomitantly induces seizures and altered mental status: both of which are also pathognomonic signs for severe hyponatremia, in the absence of high rectal temperatures or hypoglycemia.^{1,4-6,8,11,12,14,15}

Other factors that are hypothesized to play a role in the development of hyponatremia in marathon runners are high ambient temperatures and gender.

High ambient temperatures have been repeatedly documented in cases where hyponatremia has been reported.^{4,8,14-16} Conversely, personal communication with medical directors from the 1999 Chicago Marathon (27K participants) and 2000 Boston Marathon (17K participants) revealed no documented symptomatic cases of hyponatremia with temperatures at 20°–40°F throughout both races. Clearly, heat may play a role in the development of hyponatremia in endurance athletes, hikers, and military personnel. The most plausible and simple

explanation for this phenomenon is that all marathon runners slow down significantly in the heat. The perception of this slowing down as “fatigue” can be misjudged by our runners as “dehydration”, thus encouraging these slower runners to actively rehydrate to combat their apparent decrease in performance. Hence, there is increased tendency to overhydrate as a result of the heat due to this misinterpretation. If the heat alone induced physiological changes within the body, there would expect to be abnormal drinking responses and hyponatremia more uniformly spread throughout all participants and the degree of severity of this disorder would not be volume dependent. Slower athletes can better tolerate oral fluids because they are working at lower metabolic rates than their faster, more fit, counterparts. As a result, these slower runners have better opportunity and inclination to drink.

Hyponatremia presents with equal incidence in both men and women in our study, as an absolute value. Given that 34.4% of the race finishers were women (an incidence of 5 per 1000 females runners versus 4 per 1000 male runners developing hyponatremia), there appears to be a small but increased tendency for women to develop this condition. Furthermore, our two most serious cases arose in female runners. Ayus et al. who on analysis of post-operative patients concluded that “men and women are equally likely to develop hyponatremia . . . however, when hyponatremic encephalopathy develops, menstruant women are about 25 times more likely to die or have permanent brain damage.”²³ This finding would explain why a majority of case studies on hyponatremia report on a disproportionate number of symptomatic females,^{2,7,8,14} although low serum sodium levels have been widely reported in both genders, suggesting a possible hormonal bias. The smaller size of women following recommended fluid guidelines designed for 70kg men, the likelihood that women will comply with these guidelines, and the fact that women generally run slower than men also may explain why there is an increased susceptibility for females to develop hyponatremia during marathon races.

In closing, explanations as to why an alarming number of marathon runners in the 2000 Houston Marathon overdrank and developed hyponatremia can be traced to the social circumstances and environment in which the runners trained. The statistically significant number of runners who developed hyponatremia trained with one large beginning training program which promoted aggressive hydration practices: regularly advocating to “drink until your urine is clear” and to “do not wait until you are thirsty to drink” in response to this particular group’s misguided fears of dehydration. Advertisements promoting the benefits of “adequate” hydration via sports drinks as well as distorted information from the lay press—that is handed down to inexperienced coaches by word of mouth only—also influenced these runners into thinking that “if a little is good, than more must be better”. Blame should not be placed upon any single program or individual for the mass preaching of overzealous hydration, but rather an appropriate educational and marketing

overhaul must be undertaken to inform individuals to listen to their own bodies and achieve the appropriate balance of fluids based on scientific fact instead of popular fiction.

CONCLUSION

Longer finishing times and overingestion of fluids are the main risk factors for the development of hyponatremia in marathon runners. There is an inverse linear relationship between serum sodium levels versus finishing times and also between the amounts of fluid ingested versus post-race serum sodium concentration. Those athletes with the lowest serum sodium concentrations were the slowest marathon runners and reported the highest volumes of fluid intake.

Vomiting is the only clinical sign measured occurring significantly more in hyponatremic versus non-hyponatremic runners. Because hyponatremia developed as a consequence of fluid overload, vomiting is an expected physiological response of the body to expel the fluid excess. This is in contrast to dehydrated states where the body would aim to conserve fluid.

Accordingly, we suggest that marathon athletes restrict their fluid intake along the course and not intake fluids in excess of individual sweat and urine production rates. Failure to restrict fluid intake will continue to put marathon participants, female and novice runners in particular, at significant risk for developing hyponatremia.²⁴⁻²⁷

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