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Hyponatremia in the 2009 161-km Western States Endurance Run

Abstract

Purpose: To determine the incidence of exercise-associated hyponatremia (EAH), the associated biochemical measurements and risk factors for EAH, and whether there is an association between postrace blood sodium concentration ($[Na^+]$) and changes in body mass among participants in the 2009 Western States Endurance Run, a 161-km mountain trail run.

Methods: Change in body mass, postrace $[Na^+]$, and blood creatine phosphokinase (CPK) concentration, and selected runner characteristics were evaluated among consenting competitors.

Results: Of the 47 study participants, 14 (30%) had EAH as defined by a postrace $[Na^+]$ /L. Postrace $[Na^+]$ and percent change in body mass were directly related ($r = .30, P = .044$), and 50% of those with EAH had body mass losses of 3–6%. EAH was unrelated to age, sex, finish time, or use of nonsteroidal anti-inflammatory drugs during the run, but those with EAH had completed a smaller ($P = .03$) number of 161-km ultramarathons. The relationship of CPK levels to postrace $[Na^+]$ did not reach statistical significance ($r = -.25, P = .097$).

Conclusions: EAH was common (30%) among finishers of this 161-km ultramarathon and it was not unusual for those with EAH to be dehydrated. As such, changes in body mass should not be relied upon in the assessment for EAH during 161-km ultramarathons.

Keywords

dehydration, endurance exercise, exercise, sodium, water-electrolyte imbalance, electrolytes, hyponatremia

Disciplines

International and Community Nutrition | Other Medicine and Health Sciences | Other Nutrition | Sports Sciences

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Purpose: To determine the incidence of exercise-associated hyponatremia (EAH), the associated biochemical measurements and risk factors for EAH, and whether there is an association between postrace blood sodium concentration ($[Na^+]$) and changes in body mass among participants in the 2009 Western States Endurance Run, a 161-km mountain trail run. **Methods:** Change in body mass, postrace $[Na^+]$, and blood creatine phosphokinase (CPK) concentration, and selected runner characteristics were evaluated among consenting competitors. **Results:** Of the 47 study participants, 14 (30%) had EAH as defined by a postrace $[Na^+] < 135$ mmol/L. Postrace $[Na^+]$ and percent change in body mass were directly related ($r = .30$, $P = .044$), and 50% of those with EAH had body mass losses of 3–6%. EAH was unrelated to age, sex, finish time, or use of nonsteroidal anti-inflammatory drugs during the run, but those with EAH had completed a smaller ($P = .03$) number of 161-km ultramarathons. The relationship of CPK levels to postrace $[Na^+]$ did not reach statistical significance ($r = -.25$, $P = .097$). **Conclusions:** EAH was common (30%) among finishers of this 161-km ultramarathon and it was not unusual for those with EAH to be dehydrated. As such, changes in body mass should not be relied upon in the assessment for EAH during 161-km ultramarathons.

Keywords: dehydration, endurance exercise, exercise, sodium, water–electrolyte imbalance

The development of exercise-associated hyponatremia (EAH; blood sodium concentration of < 135 mmol/L) appeared to be an unusual consequence of endurance exercise when first described in non-peer-reviewed magazines in the early 1980s^{1,2} and the scientific literature in 1985.³ Since these initial descriptions, at least nine deaths from EAH have been confirmed and this potentially fatal medical condition has been found to be common in a variety of endurance activities.⁴ For instance, the incidence of EAH has been observed to be as high as 18% in Ironman triathlons,⁵ 13% in standard marathons (42 km),^{6,7} and 12% in an endurance cycling event,⁸ but as low as 0–0.6% in other ultraendurance events.^{9–11}

In our recent work, we saw an alarmingly high EAH incidence of 51% during a 161-km trail ultramarathon

that was run under hot conditions.¹² That study also noted that 57% (12 of 21) of the hyponatremic finishers had lost $> 3\%$ of their body mass. This was in contrast to previous studies, mostly from shorter duration competitions, in which EAH was relatively rare in subjects who had lost that much body mass.^{6,13–16} Further work to clarify the incidence of hyponatremia with such levels of weight loss is of critical importance because runners presenting for medical attention with $> 3\%$ body mass loss would typically be treated with aggressive rehydration. Such treatment in the presence of hyponatremia could be fatal.

The present study examined the incidence of hyponatremia in a 161-km ultramarathon run to determine if the high incidence of EAH we previously observed in such an event¹² would be replicated. We also wished to determine whether change in body mass was related to EAH occurrence, and whether EAH would again prove to be common among those with weight loss.¹² Finally, we sought to discover whether select runner characteristics were related to the development of EAH. Since previously reported risk factors for EAH have included female sex, slow performance, event inexperience, and use of nonsteroidal anti-inflammatory drugs (NSAIDs) during the event,¹⁷ we examined these variables. Because there has been suggestion of an association between EAH and rhabdomyolysis,^{18–21} we also examined postrace blood creatine phosphokinase (CPK) concentrations.

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Methods

The study was performed at the 2009 Western States 100-Mile (161-km) Endurance Run (WSER), in Northern California. The course is almost entirely on single track mountain trails with 5500 m of cumulative climb and 7000 m of cumulative descent. Twenty-four aid stations were stocked with various fluids, nutritional items, and salt capsules. Body mass was measured at nine of these stations during the race, and race policy was to force rest and rehydration for competitors whose body mass loss was >5% compared with their registration mass. Participants had 30 h to complete the race. Nearby ambient temperature ranged from 4°C to 37°C.

Institutional Review Board approval was granted for this study and subjects provided informed consent. Subjects were recruited via preregistration emails and during race registration the day before the race. Body mass was measured with a weight scale (WW42D impedance scale, Weight Watchers, New York City, NY) positioned on a solid level surface during race registration with the subjects barefoot and wearing lightweight running clothes. Within a few minutes of finishing the race, barefoot body mass was measured with the same scale on a solid level surface, and blood was drawn from an antecubital vein for onsite analysis of sodium concentration ($[Na^+]$) using an I-Stat portable analyzer (Abbott, Princeton, NJ). The I-Stat has been reported to have a coefficient of variation of $\leq 0.01\%$ in the measurement of $[Na^+]$ and to yield a strong correlation ($r = .95$) with the Ciba Corning 288 blood gas analyzer for $[Na^+]$.²² Blood was also sent to the local hospital laboratory for determination of blood CPK concentration. Creatine phosphokinase values over 40,000 U/L were not diluted for further analysis and were reported as >40,000.

Each subject was sent an e-mail the day following the race with an invitation to complete an electronic survey. Details of the methods and findings of the survey

are provided elsewhere.^{23,24} Select questions used in the present study were those determining whether nausea had affected race performance, if NSAIDs were used during the race, the greatest distance run in one week during the 3 mo before the race, and the number of prior 161-km running races completed. The latter was determined from our records^{25,26} for those runners not completing the postrace survey. Finish times and subject ages were obtained from official race results.

Unpaired *t* tests and Fisher's exact tests were used to compare hyponatremic and normonatremic runners in interval and categorical variables, respectively. A non-parametric Mann-Whitney test was used to analyze the number of previously completed 161-km races because these data were not normally distributed. Pearson correlation analyses were used to examine the associations between interval variables after verifying the values approximated Gaussian distributions. A value of 40,000 was used in the analyses for CPK values >40,000. Statistical significance was set at $P < .05$.

Results

A total of 104 of the 399 race starters consented to participate in the study. Of those, 47 finished the race and provided blood samples upon completing the race, and 14 (30%) of this group had a $[Na^+]$ below 135 mmol/L. All were fully oriented and without evidence of altered mental status.

Table 1 compares characteristics of normonatremic and hyponatremic groups. The two groups were not statistically different with regard to age, sex, finish time, highest training distance week, presence of nausea during the race, and use of NSAIDs during the race. Those who were normonatremic had a greater ($P = .03$) number of previous 161-km race finishes. Normonatremics also had trends ($P = .06$) toward less weight loss and lower postrace CPK concentrations than hyponatremic runners.

Table 1 Comparison of normonatremic and hyponatremic groups. Data are presented as mean \pm SD except where indicated. The last three items are based upon survey responses from 35 of the 47 study participants.

Item	Normonatremic	Hyponatremic	<i>P</i> value
Postrace $[Na^+]$ (mmol/L)	138.3 \pm 2.4	131.5 \pm 2.2	<0.0001
Age (years)	44.6 \pm 7.0	42.0 \pm 9.6	0.30
Women (%)	18.2	28.6	0.46
Finish Time (hours)	25.84 \pm 2.73	26.29 \pm 2.80	0.61
Change (postrace minus registration) in Body Mass (%)	-1.0 \pm 2.6	-2.6 \pm 2.5	0.06
Postrace CPK (U/L)	19,018 \pm 13,467	27,518 \pm 13,571	0.06
161-km Races Previously Completed (Median and Range)	3 (range 0-56)	1 (range 0-43)	0.03
Highest Running Distance in 1 Week (km)	139 \pm 39	134 \pm 44	0.73
Presence of Nausea (%)	43.5	58.3	0.49
Use of NSAIDs (%)	56.5	58.3	1.00

Postrace $[Na^+]$ was positively and significantly related to percent change in body mass (Figure 1). Also notably evident from this figure is that 7 of the 14 hyponatremic runners lost 3–6% of body mass.

Finish time was unrelated to percent change in body mass (Figure 2). The greatest decrease in body mass (6.3%) was seen for the fastest study participant.

Creatine phosphokinase concentrations were unrelated to percent change in body mass ($r = .08$, $P = .60$) or to postrace $[Na^+]$ (Figure 3).

Discussion

The most important findings of this study are that there was a direct relationship between postrace $[Na^+]$ and percent change in body mass, and that half of the hyponatremic runners had lost >3% body mass during the race. These findings contrast with previous studies in which postrace $[Na^+]$ has universally been found to be inversely related to change in body mass.^{6,9,13–16,27} Furthermore, a compilation of data from 2,135 finishes of different endurance events that were mostly shorter in duration than that of the present study demonstrated that the incidence of EAH was only 4% among those who lost >3% body mass.¹⁵ This contrasts with the present study in which EAH was present among 54% of those with >3% body mass loss. Yet, the current findings are consistent with those of Lebus et al¹² and confirm that EAH can be relatively common during a 161-km ultramarathon when body mass loss is >3%. Furthermore, even if we were to consider a mass loss of 4% as the minimum required for dehydration, which would be appropriate based upon our energy balance data from this event,²⁸ we would conclude that 50% (5 of 10) of the runners with such mass loss had EAH.

Another important finding of the present study was the high incidence of EAH. The 30% incidence of EAH in the present study was considerably higher than the 7% incidence in the compilation study.¹⁵ Furthermore, a comparable study of 13 finishers of a hot (21–38°C) 160-km ultramarathon run reported that none were hyponatremic at the finish.⁹ Yet interestingly, our current findings are consistent with the high incidence (51%) reported by Lebus et al¹² during a 161-km ultramarathon, which, like the ultramarathon of the present study, took place under hot conditions in northern California on a challenging trail course. The reason for the apparently unique findings from these 161-km ultramarathons in northern California is not clear and invites further exploration.

Overhydration from excessive fluid intake is considered to be an important etiology of EAH.^{15,17} Considering overhydration as a lack of mass loss,¹⁵ 34% of the present sample was found to be overhydrated. Yet, of those who overhydrated, only two (13%) developed biochemical EAH, and none developed clinically significant EAH. This differs considerably from the compilation study in which overhydration was present among only 11% of subjects, of which 11% and 19% developed biochemical EAH and clinically significant EAH respectively, for a total of 30% of those who were overhydrated developing

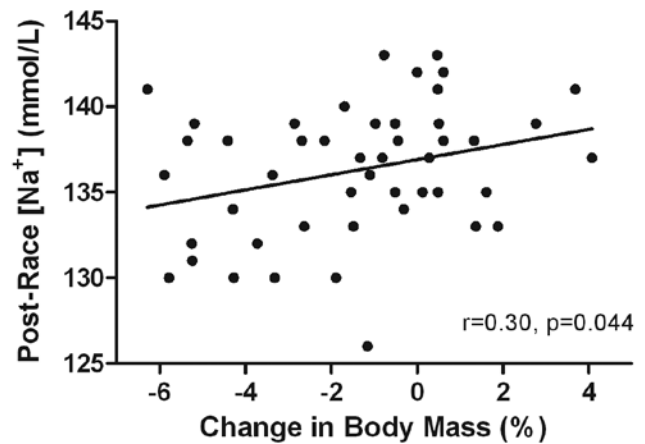


Figure 1 — Relationship of postrace $[Na^+]$ to percent change (postrace minus registration) in body mass.

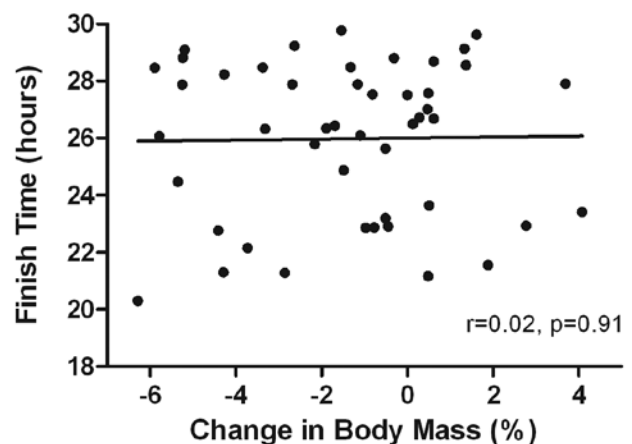


Figure 2 — Relationships of finish time to percent change (postrace minus registration) in body mass.

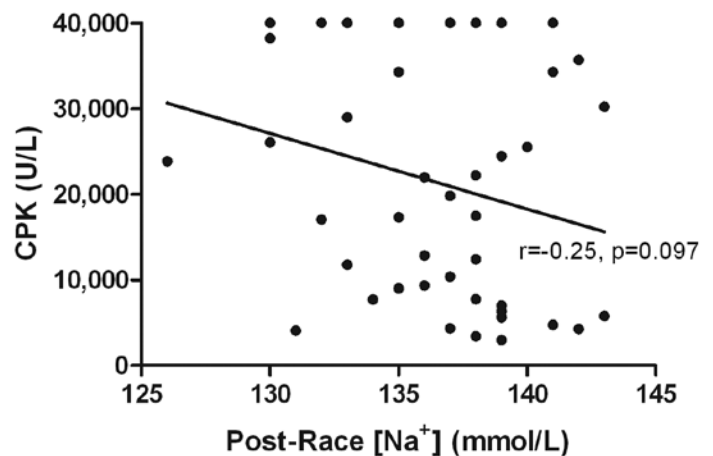


Figure 3 — Relationships of CPK to postrace $[Na^+]$.

EAH. Furthermore, 45% of those with EAH were overhydrated in the compilation study, whereas only 14% of those with EAH were overhydrated in the present study.

Thus, overhydration was more common in the present study, yet was not as likely to be associated with EAH as in the compilation study.

The high incidence of an increase in body mass in the present event probably relates to the emphasis on avoidance of significant mass loss. Race policy at the time of the event required that runners be stopped and rehydrated for mass loss of >5%. The high incidence of an increase in body mass may also relate to a great emphasis in the United States on the use of sodium supplementation during endurance competitions. In fact, the *WSER Participant's Guide* recommends sodium intake, and sodium capsules were provided at aid stations. Excessive sodium intake is likely to result in plasma volume expansion and gain in body mass.^{12,29} But, it is interesting that overhydration was less likely to be associated with EAH in this study compared with others. The fact that most of the runners in the overhydrated group were normonatremic is consistent with the premise that excessive fluid intake was accompanied by aggressive sodium intake.

Previous work has concluded that sodium supplementation via salt capsules is unnecessary to maintain $[Na^+]$ during prolonged endurance exercise.^{14,29} However, the high incidence of EAH in 161-km ultramarathons observed in the present study and that of Lebus and colleagues,¹² along with the finding that EAH can commonly be associated with dehydration suggests that sodium loss may be an etiological factor in the development of EAH during these longer duration events under high ambient temperatures. Alternatively, the osmotic inactivation of circulating sodium or failure to adequately mobilize osmotically inactive sodium from internal stores have been suggested as underlying etiologies of EAH.¹⁵ Further work is required to fully illuminate the relative role of these potential mechanisms in EAH under the conditions of this event.

The present study examined other risk factors for EAH. Previously reported risk factors for EAH include female sex, slow performance, event inexperience, and use of NSAIDs during the event.¹⁷ The present work found no statistical evidence for EAH being related to age, sex, race performance, or use of NSAIDs. However, this work does support higher risk associated with fewer previous event completions.

Nonosmotic secretion of arginine vasopressin is felt to be an exacerbating factor in most cases of EAH.¹⁷ Nausea is a potential nonosmotic stimulus for arginine vasopressin secretion. While about half the subjects reported nausea, there was no difference in the frequency of nausea between those who presented with EAH and those who did not.

Recent case reports have suggested a link between rhabdomyolysis and EAH.¹⁸⁻²¹ Theoretically, EAH could promote rhabdomyolysis through changes in intracellular potassium or calcium concentrations, or through hypotonic extracellular fluid being drawn into the muscle cell, each of which could reduce the stability of the cell membrane and allow for easier breakdown. Conversely, the breakdown of muscle cells could lead to third spacing of fluids, which could stimulate arginine vasopressin secretion leading to EAH. The present study examined CPK levels, and although statistical differences between hyponatremic

and normonatremic groups were not found ($P = .06$), the data suggest that closer examination with a larger subject sample is warranted.

This study examined only postrace $[Na^+]$ rather than prerace to postrace change in $[Na^+]$, as has been the case for some studies.^{9,27,30} We believe that postrace $[Na^+]$ is the more relevant measure and likely to be more reflective of any change in $[Na^+]$ from normal baseline under the conditions of this study. The WSER weighing policy has engendered a culture in which runners try to weigh-in light at registration. They typically weigh-in in the morning before eating on the day before the race so that the value is lower than it would be the next morning after aggressively hydrating during the remaining hours before the race. This practice may cause a transient lowering of prerace $[Na^+]$ that would not accurately reflect the normal baseline value for these individuals. For the same reason, we believe that our use of body mass measurements at registration the day before the event is more reflective of a normal baseline than measurements made immediately prerace. Previous work has demonstrated that body mass immediately before the start of an endurance competition is 0.6–1.05 kg higher than at race registration^{11,31-33} supporting the premise that such measurements may reflect an overhydrated state rather than a normal baseline. As such, our use of body mass measurements from registration would tend to underestimate mass loss during the event relative to what would be determined based upon prerace measurements. This means that the proportion of runners with EAH who had >3% mass loss would likely have been even higher than 50% had we used body mass measurements from immediately before the race. Regardless, the reality is that the registration weight is the value used by race officials, and so this value has the greatest practical importance in this race.

The lowest postrace $[Na^+]$ we observed was 126 mmol/L. In fact, only this one case was in the “clinically significant” range (<128.9 mmol/L) as defined by Noakes et al.¹⁵ Furthermore, none of our cases displayed evidence of hyponatremic encephalopathy. Yet, asymptomatic EAH can rapidly progress to severe illness.¹⁷ Thus, further investigation into the underlying mechanism and the explanation for the high incidence of EAH in 161-km ultramarathons is warranted.

The high incidence of EAH we have observed during hot weather 161-km ultramarathons makes it imperative that on-site and local emergency medical personnel be cognizant that some runners seeking medical attention are likely to be hyponatremic. In addition, EAH can occur with considerable loss in body mass during a hot 161-km ultramarathon indicating that changes in body mass cannot be relied upon in the clinical assessment for EAH. As such, the need for onsite $[Na^+]$ analysis, as recommended by the EAH Consensus Group,¹⁷ is justified. Furthermore, encouragement of oral hypotonic fluids or the administration of intravenous hypotonic fluids, even in athletes who present as fluid depleted, is risky without knowing the $[Na^+]$.

In summary, this work demonstrates that EAH can be common (30% incidence) among finishers at a hot 161-km run and that it may not be unusual for those with EAH to

be dehydrated. The development of EAH was unrelated to age, sex, finish time, or use of NSAIDs during the run, but those developing EAH had completed fewer 161-km ultramarathons.

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