RECURRENT HYponATREMIA IN THE ENDURANCE ATHLETE:

A CASE STUDY

By

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ABSTRACT

Hyponatremia of exercise (HAE) is discussed using a case study of a 42-year-old amateur triathlete who experienced HAE four times within a 6-month period. A review of literature was performed to determine the etiology, diagnosis, and treatment of HAE. Current theories on the development of HAE are discussed, with the probable cause being increased ingestion of hypotonic fluids, but this has not been scientifically proven. The current body of knowledge in these areas is presented to guide primary care providers in the treatment of athletes with emphasis on fluid restriction, necessary laboratory tests, monitored use of hypertonic intravenous solutions, and recommendations for training and race strategies for endurance athletes who experience HAE.
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RECURRENT HYponATREMIA IN THE ENDURANCE ATHLETE:

A CASE STUDY

Hyponatremia is the most common electrolyte disorder encountered in clinical medicine (Rutecki & Whittier, 1994). Hyponatremia is associated with many clinical syndromes and has numerous etiologies. Symptomatic hyponatremia is a medical emergency. Untreated, it can lead to life-threatening complications such as generalized seizures, pulmonary edema, respiratory arrest, and death. The incidence and prevalence of hyponatremia is 1% and 2.5%, respectively, with a mortality rate of 40% (Anderson, Chung, Rudiger, & Schrier, 1985). Acute symptomatic hyponatremia is most often seen in four distinct populations: (a) psychogenic polydipsia, (b) inpatients with impaired water excretion who are treated intravenously with hypotonic fluids, (c) inpatients who systemically absorb electrolyte free irrigating solutions during prostate surgery, and (d) endurance athletes (Surgenor & Uphold, 1994).

Hyponatremia associated with exercise (HAE) has been recognized with increasing frequency in recent years. HAE has been diagnosed in people involved in moderate exercise such as hikes, college football, and marches (Herfel, Stone, Koury, & Blake, 1998; Zelingher et al., 1996), but the largest group experiencing HAE has been endurance athletes participating in triathlons or marathons. Endurance athletes are defined as athletes who participate in events such as triathlons (a combination of
swimming, biking, and running), lasting greater than 4 hr (Hiller, 1989) or a multisport event lasting 7 hours or more (Noakes, Irving, & Buck, 1990).

The number of participants in endurance events continues to grow yearly. In the United States alone, over 200,000 athletes participate in nearly 700 sanctioned events across the country (USA Triathlon, 1999). The Ironman triathlon is held as the gold standard of triathlons, consisting of a 2.4-mile swim, 112-mile bike, and a 26.2-mile run and taking from 8.5 to 16.5 hours to complete (Speedy, Faris, Hamlin, Gallagher, & Campbell, 1997b). There are numerous reports of HAE during triathlons and marathons (Herfel et al., 1998; Irving et al., 1991; Noakes, Goodwin, Rayner, Branken, & Taylor, 1985; Surgenor & Uphold, 1994). More importantly, two deaths have been attributed to ingestion of more than 15 L of hypotonic fluids while exercising (Garigan & Ristedt, 1999; Zorn, 1999). Up to 29% of triathlon starters may develop HAE (Hiller, O'Toole, Massimino, Hiller, & Laird, 1985; Noakes et al., 1990a; Noakes et al., 1990b). While the exact time needed to develop HAE is unknown, symptoms of HAE have been documented following, as few as 4 hours of moderate exercise.

HAE is neither a significant financial issue of health care, nor a burden to society as the acute symptoms of HAE are generally reversible and without sequelae. However, the emotional costs to the athlete are significant. The endurance athlete is dedicated to physical training, investing both thousands of hours and dollars in preparation for one race. In developing HAE, the body of which the athlete is so proud has failed and medical science cannot provide the athlete with an explanation.

The etiology and treatment of HAE remain elusive. As HAE becomes more frequent in the athletic, and more recently general population, the primary care provider
must learn to recognize symptoms of HAE in presenting patients and differentiate HAE from a similar process presenting with like symptoms. The primary care provider must also become familiar with diagnostic criteria, etiologies, treatment alternatives, and patient education, which will ultimately allow the athlete to continue the exercise which he/she loves. The purpose of this article is to present a case study of a triathlete (who on several occasions experienced HAE), review the literature, discuss the definition, etiology, diagnosis, and treatment of HAE, and make recommendations for endurance athletes who experience HAE.

Case Study

C. N. is a 45-year-old amateur triathlete who experienced three and possibly four episodes of hyponatremia associated with exercise over a 4-month period of time. He has a past medical history of mild asthma controlled by zafirlukast (Accolate) 20 mg BID, salmeterol xinafoate (Serevent) 2 puffs BID, and albuterol sulfate (Ventolin) PRN. He has been active in mountain climbing, cross country skiing, and triathlons throughout his adult life. He has competed successfully in several short triathlons and one-half Ironman triathlon without any major sequela.

In August of 1996 he began a year of intense training for the Canadian Ironman held in August 1997. He biked, ran, swam, or lifted weights for an average of 12 hours per week. As part of this training, in May of 1997 he competed in the Wildflower Half Ironman Triathlon, a 1.2-mile swim, 56-mile bike, and 13.1-mile run, but disqualified himself 6½ hours into the race due to fatigue, abdominal cramping and vomiting, and “a funny headache.” He later developed severe shortness of breath, vertigo, uncoordinated gait, and a feeling of eminent doom. He drank approximately 5 L of fluid during the race
and urinated a small amount of concentrated urine 4.5 hours into the race (Table 1). He quit sweating 30 minutes prior to his disqualification. In the medical tent at the race site, his temperature was 37 °C and his serum sodium was 119 mg/dl. He was given 3 L of intravenous (IV) normal saline (NS) and transported to the hospital via ambulance. At the hospital his oxygen saturation (O₂ sat) was 99%, chest x-ray was negative, and telemetry monitor showed normal sinus rhythm. His lab values (see Table 2) were serum sodium 122 mmol/L, potassium 4.6 mmol/L, chloride 91 mmol/L, bicarbonate 21 mmol/L, BUN 13 mg/dl, creatinine 0.8 mg/dl, glucose 200 mg/dl, CPK 422 IU/L, and CPK MB 11%. Urinalysis was negative for blood but positive for small to moderate ketones (Table 2). He received a fourth bolus of normal saline, which was followed by D5 ½ NS with 40 mEq potassium at 75 cc/hr. He also received prochlorperazine (Compazine) 5 mg and metoclopramide hydrochloride (Reglan) 10 mg for nausea, and 5 mg of albuterol sulfate with 0.5 mg ipratropium bromide (Atrovent) via nebulizer for shortness of breath. He was admitted to the hospital overnight and released the following morning. His physician stated that his diagnosis was “status post heat exhaustion from participating in a triathlon where the temperature was very hot. This is somewhat surprising since he did give a history that he was taking plenty of fluids during the race.” Discharge instructions encouraged more fluids during races.

Approximately 2½ months later, on July 28, 1997, C. N. was admitted to the emergency room following an 8-hour strenuous bike ride in the heat. He consumed 10.5 L of fluid, half consisting of a commercial electrolyte solution (All Sport) and the remainder being tap water. During the ride he voided twice (Table 1). Upon completion of the ride he felt well, but symptoms of fatigue, nausea, dyspnea at rest, pallor, and a
feeling of eminent doom began within 45 minutes after completion of the event. Two
puffs of albuterol were used hourly in an attempt to alleviate the dyspnea without relief.
The symptoms of fatigue, nausea, dyspnea at rest, pallor, and a feeling of eminent doom
intensified and finally cumulated in a fainting episode 3 hours following the completion
of the ride. He was then taken to the emergency room for evaluation and treatment.
Orthostatic blood pressure was 118/72 lying, 128/80 sitting, and 120/60 standing. EKG
showed normal sinus rhythm. His mini-profile showed a glucose of 107, sodium 126,
chloride 91, anion gap 14.8, troponin-I was normal at 0.4, WBC 10,600, hemoglobin 14,
and platelets 235,000 (Table 2). At the emergency room he was given 3 L (Table 1) of
Lactated Ringer’s solution IV and discharged in satisfactory condition with the diagnosis
of moderate dehydration, overexertion, and hyponatremia. His discharge instructions
specified rehydration orally. Race strategies included the need to use more electrolyte
solutions in place of water and to continue with increased hydration.

C. N. may have experienced another episode of HAE on August 6, following a
4-hour bike ride in moderate heat. His total fluid intake at that time was 18 cups over 4
hours (see Table 1). He was fatigued, nauseated, and slightly dyspneic at the completion
of the ride but attempted to abort the hyponatremic attack at home. He consumed 2½
cups of chicken broth in 2½ hours. Blood pressure remained stable with the low of
100/80 and high of 114/78. He voided concentrated urine twice during the ride and three
more times following the end of the ride, with the urine slowly becoming straw colored.
His weight at the beginning of the ride was 157 lb and on return 158 lb.

On August 25, 1997, he attempted the Ironman Canada (IMC). He completed the
swim and the bike portions of the triathlon in 9 hours and stopped by the medical tent to
obtain a prearranged serum sodium level. His serum sodium level was 127, and he voluntarily disqualified himself from the race. During this race he had ingested 36 cups of balanced electrolyte replacement beverages and also ingested a 1,000 g sodium/chloride capsule on an hourly basis. Shortly after stopping the race, he began to experience nausea, fatigue, dyspnea, and a feeling of eminent doom. He developed generalized edema and pallor. He was encouraged to drink three to four cups of chicken broth. Three hours following the termination of the race, C. N. was admitted to the medical tent proper with a serum sodium of 123 mEq/L for which he received 3 L of 5% dextrose with NS (D5NS) over the ensuing 3 hours. A fourth IV was hung, but he developed basilar crackles and the IV was discontinued. A final serum sodium of 125 mEq/L was obtained, and he was discharged.

Following the third documented episode of hyponatremia, C. N. became depressed and lost his desire for endurance exercise. He attempted a mountain summit during the winter of 1998, but aborted the attempt when he began to experience fatigue, dyspnea, and “a funny headache” symptoms which had previously led to episodes of HAE. After this experience C. N. stopped exercising completely. Although C. N. was never diagnosed with HAE, in retrospect his symptoms highly resemble those associated with HAE.

Definition of Hyponatremia Associated with Exercise

Hyponatremia is not in itself a disease entity but rather a clinical manifestation of a more basic underlying process. All true hyponatremia is dilutional. By definition, the defect in all hyponatremic patients is too much water for body volume (Rutecki & Whittier, 1994).
Clinical symptoms of HAE include weakness, increased fatigability, muscle cramps, decreased mentation and confusion, and may proceed to disorientation, generalized seizure, coma, increased intracranial pressure, pulmonary edema, respiratory arrest, and death (Clark & Gennair, 1993; Garigan & Ristedt, 1999; Maclean, Champion, & Trash, 1976; Zorn, 1999). Endurance athletes present with symptoms of light-headedness, nausea, vomiting, malaise, exhaustion, altered mental states, tonic clonic seizures, and headache (see Table 3; Speedy et al., 1997a; Speedy et al., 1997b).

Serologic manifestations of HAE include a concentration of sodium in plasma that is below the normal range of 135-145 mEq/L. Mild hyponatremia is defined as a plasma concentration of 130-134 mEq/L. Severe hyponatremia is defined as a plasma sodium concentration below 130 mEq/L (Speedy et al., 1999). Neurologic manifestations of acute hyponatremia generally present at sodium levels of less than 125 mEq/L (Arieff, Llach, & Massry, 1976). Not all athletes with chemically diagnosed hyponatremia present for medical care; this condition has been termed “asymptomatic hyponatremia” and may account for more than 50% of HAE cases. Athletes having both serologic and physical manifestations of HAE are classified as symptomatic and account for 23% of athletes presenting for health care during a triathlon (O’Toole, Douglas, Laird, & Hiller, 1995). Female premenopausal athletes are more likely to present with more severe symptoms at higher levels (104-130 mEq/L) of serum sodium (Fraser & Arieff, 1997; Speedy et al., 1999).

The differential diagnosis of HAE includes both nonexercise and exercise-related disorders of collapsed athletes (see Table 4). Nonexercise disorders are rare and will not be covered in this article. Exercise-related disorders of collapse include dehydration, heat
stroke, heat exhaustion, symptomatic hyponatremia, hypoglycemia, and exercise-associated collapse (see Table 5). The three most serious emergencies likely to be encountered are heatstroke, symptomatic hyponatremia, and profound hypoglycemia (Holtzhausen & Noakes, 1997; Noakes, 1992). Heat stroke can be differentiated from heat exhaustion by a rectal temperature > 40 °C and a significant alteration in mental status. Hyponatremia (serum sodium < 135 mEq/L) and hypoglycemia (serum glucose < 60 mg/dl) can be differentiated by their abnormal lab values. Athletes may also present with symptoms of dehydration, which include elevated hemoglobin and hematocrit, inability to spit, decreased skin turgor, and sunken eyeballs (Holtzhausen & Noakes, 1997). Exercise-associated collapse can be differentiated from other disorders in that onset closely follows cessation of exercise (Holtzhausen et al., 1994; Roberts, 1996).

Pathophysiology of Hyponatremia

Fluid and electrolyte homeostasis in the body is regulated by a complex system. Under normal conditions fluid and sodium balance are well regulated by aldosterone and antidiuretic hormone (ADH). Water balance is regulated by hypothalamic control over ADH release from the posterior pituitary. ADH is released in response to increased osmolality of the blood, a decrease in plasma volume, or decreased left atrial pressure and production of angiotensin II (Barr, Costill, & Fink, 1991).

In the kidney, the principal site of action of ADH, with respect to water balance, is on the renal cortical and medullary collecting tubules and ducts. The osmolality of the urine excreted is determined by the effect of ADH on the permeability to water of the distal tubule and collecting ducts. In the absence of ADH, the luminal cells of the distal tubule and collecting ducts are impermeable to water, and dilute urine is formed. In the
presence of ADH, fluid is reabsorbed from the lumen and concentrated urine is excreted. The renal role in regulation of the tonicity of body fluids depends on the ability of the kidney to excrete either dilute or concentrated urine in response to extrarenal fluid balance. This is accomplished by separating water from solute in the glomerular filtrate (extracellular fluid) and recombining these for excretion in properties responsive to total body fluid requirements and to the maintenance of normal body fluid tonicity (Hellerstein, 1993).

Regulation of sodium is closely tied to that of water. Sodium is the major contributor to serum osmolality, which is a regulator of aldosterone secretion. Aldosterone acts to increase sodium reabsorption in the distal tubules of the kidney, as well as in the intestine and the lumen, of sweat and salivary glands. When sodium is added to or removed from the body, renal regulation of water balance results in retention or excretion of water to maintain body fluid osmolality within a very narrow range. The efficiency of this system in regulating water and sodium balance is indicated by the body’s capacity to maintain normal sodium balance. In the absence of significant sweating, this can be accomplished with oral intakes ranging from as little as 230 mg (~10 mmol) to well in excess of 5,000 mg/day (217 mmol/day).

Similarly, water balance can be maintained with, as little as 1.5 L/day with maximal ADH stimulation, or as much as 20 L/day at which level the kidney’s ability to produce urine (13 ml/min) is exceeded. The maximal rate of urine formation is similar to the maximal rate at which fluid empties from the stomach for absorption from the gastrointestinal tract. This suggests that under normal conditions, the capacity to ingest fluid is well matched to the capacity to excrete it (Barr et al., 1991).
As the athlete begins to exercise, water is rapidly shifted from the plasma into the exercising muscles and interstitium, causing a drop in plasma volume. The drop in plasma volume (a) stimulates ADH release, causing water reabsorption; and (b) decreases renal blood flow and pressure (Barr et al., 1991). Decreased renal perfusion, in association with release of catecholamines, stimulates renin release from the kidney, resulting in aldosterone secretion and sodium reabsorption. An increase in serum potassium also stimulates aldosterone secretion. Thus, even before significant sweat losses have occurred, mechanisms for conservation of sodium and water have been activated. As exercise continues, water loss from sweating becomes more important, as the production and evaporation of sweat are major means of dissipating heat generated by muscle activity. Because sweat is hyponatremic compared to body fluids, water losses generally exceed sodium losses. As a result, the osmolality of the blood increases, further stimulating ADH release.

At a normal plasma osmolality of approximately 285 mOsm/kg water, circulating plasma ADH level is approximately 2 pg/mL, which is the level needed to produce a half maximal urine concentration of approximately 600 mOsm/kg. With dehydration, thirst is first experienced only when plasma osmolarity reaches approximately 294 mOsm/kg water. At this level of plasma osmolarity, ADH is maximally stimulated (usually above 5 pg/mL) and is sufficient to achieve a maximal concentrated urine (Fraser & Arieff, 1997).

When plasma osmolarity falls as a result of hyponatremia, water immediately starts to move into cells to achieve osmotic equilibrium. This equilibrium between
cellular compartments is maintained by either extrusion of intracellular solutes or by the 
dilution of intracellular solutes by the influx of water from the extracellular space.

Regardless of the mechanism by which the organism perceives changes in the 
ECF volume, the kidneys must control body sodium excretion to regulate sodium 
balance. Almost every factor affecting glomerular filtration, tubular reabsorption, and 
tubular secretion affects sodium excretion (Hellerstein, 1993).

Etiology of Hyponatremia

The mechanism of action which leads to hyponatremia in endurance athletes has 
not been identified. Researchers have postulated theories, often based on observations of 
athletes symptomatic with HAE, but none have been proven. Of these theories, increased 
sweating, increased ingestion of hypotonic solutions, third spacing of fluids, 
inappropriate renal function, inappropriate endocrine function, or any combination of the 
above have all been implicated.

Theories of Etiology of Hyponatremia Associated with Exercise

Increased Sweating

Researchers have looked at the role of sweat loss in causing hyponatremia, but 
results have not adequately explained the decreased sodium. Adolph’s (1947) classic 
study on dehydration concluded that acclimatized individuals have an increased sweat 
volume and lower sweating thresholds because of the sweat gland’s ability to concentrate 
sweat sodium while increasing sweat rate. Therefore, acclimatized individuals have 
higher, not lower water requirements. Hiller (1989) concurred with Adolph, suggesting 
that hyponatremia associated with exercise is caused by massive unreplaced sodium 
losses in the sweat associated with partial replacement of fluid, but not sodium.
This postulate has not been supported by further studies. Irving et al. (1991) have shown that sodium losses of runners who developed hyponatremia in a 90-km ultramarathon were no greater than were the losses of runners who maintained normal serum sodium concentrations.

Armstrong et al. (1993) supported Irving’s findings when researching the effects of dietary sodium consumption on heat acclimation and physical performance on 10 subjects. Sweat rate was determined by measuring the difference between the pre and post exercising rates. This calculation of sweat rate was corrected for water intake, urine output, fecal excretion, and food consumption. Urine volume and urinary sodium were measured in all samples produced during testing. Sweat electrolytes were measured by rinsing the entire body with a known volume of distilled, deionized water at the end of exercise; subjects showered prior to testing, wore ion-free clothing, and blotted their skin with ion-free towels to ensure that sweat did not drip onto the floor. During the study one subject unexpectedly became hyponatremic. The hyponatremic subject’s sweat sodium was compared with the normonatremic subjects’ sweat sodium and found to be identical. Sweat sodium concentrations of unacclimatized subjects were found to be 22.2 and 28.4 mEq/L (Armstrong et al., 1993).

This is substantially different (2.8 to 31.6 mEq/L) from the 25-60 mEq/L Noakes et al. (1990b) found in acclimatized subjects. Using calculations, if an unacclimatized 68-kg athlete with a prerace serum sodium of 140 mEq/L sweat at a rate of 500-750 ml per hour (Barr & Costill, 1989) when competing in a 10-hour race (total fluid loss 5 to 7.5 L), the athlete should loose between 142 and 213 mmol of sodium chloride using the high sweat sodium (28.4 mEq/L) noted above. This loss of sodium would drop the serum
sodium chloride concentrations to 135 mEq/L if the extracellular volume were to be maintained. This result is at the low end of normal for serum sodium. It would take a total loss of 21.2 L of sodium or 2.1 L/hr to drop the serum sodium to 120 mEq/L. The same athlete, if he were acclimatized, would lose between 300-450 mmol of sodium using the high rate for acclimatized athletes noted above. This would drop the serum sodium to 129 mEq/L, if the extracellular volume were to be maintained. However, acclimatized athletes are less likely to develop hyponatremia (Thein, 1995).

Vrijens and Rehrer (1999) studied the sweat rates and plasma sodium levels of 10 subjects during a 3-hour exercise trial in a hot, humid environment to determine if there was a correlation between sweat rate and plasma sodium concentration. The mean rate of sweat loss was 1.27 ± 0.22 L/hr. Plasma volume was calculated according to the formula of Van Beaumont (1973): % change PV = (100/00 - Hctpre) x 100 (Hctpre - Hctpost) / Hctpost. The results showed no significant correlation between sweat rate and rate of change in plasma sodium concentration.

Although sodium is lost through sweat, neither the sodium concentration of sweat nor the total sweat loss indicates that sweat sodium loss is a critical factor in the development of HAE. However, it may be a contributing factor.

**Increased Ingestion of Hypotonic Solutions**

There is great controversy over the amount of fluid an athlete needs to maintain proper hydration when exercising. The emphasis, by some researchers, is placed on the need to drink increased fluids during exercise in hot climates to avoid dehydration and heat stroke (Hiller, O’Toole, & Laird, 1986; Hiller, 1989). Other researchers emphasize the need to limit the intake of fluids to avoid fluid volume overload and ensuing
complications of cerebral and pulmonary edema, respiratory arrest, and death (Noakes et al., 1985; Barr et al., 1991; Montain, Latzka, & Swaka, 1999).

Irving et al. (1991) studied 8 runners who developed severe, symptomatic hyponatremia during or after the 1988, 88-km Comrades Marathon. Subjects were hospitalized, and their renal function, including fluid and electrolyte balance, was studied in detail until their serum sodium concentrations had normalized, usually within 12 to 36 hours after hospital admission. The critical finding of this study was that the hyponatremic runners excreted a mean excess of 3 L of fluid during recovery.

Nonhypernatremic runners usually conserve water and gain weight during recovery from marathon and ultramarathon races (Irving et al., 1990; Irving et al., 1991). It was found that the estimated mean fluid rate of intake of hypernatremic runners during exercise (1.3 L/hr) greatly exceeded the rates found in runners who maintained normal serum sodium concentrations during prolonged exercise (± 0.5 L/hr; Armstrong et al., 1993; Garigan & Ristedt, 1999; Irving et al., 1990; Noakes et al., 1990b). Accordingly, it was concluded that the symptomatic hyponatremia associated with exercise results from abnormal fluid retention in runners who ingest excessive fluid volumes during prolonged exercise.

Speedy et al. (1997b) attempted to show a correlation between weight and serum sodium concentrations. Athletes (N = 264) were weighed within 1 hour of the start of the race and at the finish line. All athletes presenting themselves for medical care were also weighed. The mean weight loss over the course of the race in athletes not seeking medical care was 2.1 kg (2.9% loss of body weight). There was a mean weight loss of 1.9 kg (2.5% loss of body weight) in those athletes seeking medical care. Only 1 of 8
athletes presenting with hyponatremia had been weighed prior to the race. He showed a weight gain of 2.5 kg. His estimated fluid intake was 2 L before the start of the race and 16 L over the course of the race. This is an average of ~1.5 L/hr over the 11 hours of racing. The fluid retention of the hyponatremic athlete compared with the weight loss of the normonatremic athletes suggests the fluid retention hypothesis as the cause of hyponatremia in this athlete. Post-race serum sodium ranged from 130 to 148 mEq/L. An inverse relationship between post-race sodium concentrations and percent change in body weight was observed, where subjects who decreased in body weight had the higher post-race sodium concentrations. These results correlate with two previous studies (Irving et al., 1991; O’Toole et al., 1995).

Speedy et al. (1997a) also studied the correlation between weight and serum sodium levels following a multisport triathlon (N = 48). A multisport triathlon differs from a triathlon in that a multisport triathlon typically involves kayaking, cycling, and running (there is no swimming). Athletes were weighed 30 minutes and 10 minutes prior to the start of the race. A 5-ml blood sample was also collected 10 minutes prior to the race. Within 10 minutes of completion of the race, athletes were weighed again in clothes similar to those worn during the prerace weight and another 5-ml blood sample was collected. The mean weight change over the course of the race was a loss of 2.5 kg or a mean percentage loss of body weight of 3.1%. No athletes sought medical attention after the race. The mean serum sodium concentration at the end of the race was 139.3 mEq/L. One athlete was found to be hyponatremic (serum sodium = 134 mEq/L). This athlete maintained his weight over the course of the race and did not seek medical attention. There was a weak, but statistically significant (r = 0.30; p = 0.04) correlation
between serum sodium concentrations and weight loss. Athletes who experience greater
weight loss present with higher serum sodium levels. One of the factors contributing to
the low incidence of hyponatremia in this study may be that the athletes had less access to
fluid (athletes provided their own support teams) during the race compared with races
that provide organized aid stations with freely available fluids. Noakes (1992) notes that
hyponatremia associated with exercise did not present at triathlons until the addition of
water at aid stations. There is documentation showing that athletes who have previously
presented with HAE have been able to successfully complete a triathlon of the duration
previously attempted by decreasing the amount of fluid ingested (Noakes et al., 1985).

In the previously cited study, Speedy et al. (1997a) described a mean percentage
body weight loss of 3.1%, or an estimated degree of dehydration of 1.9% in the athletes;
however, all athletes were able to complete the race. Prior studies have shown that
exercise-induced levels of dehydration of 1.8% loss of body weight are associated with a
decrease in exercise performance (Walsh, Noakes, Hawley, & Dennis, 1994).
Dehydration has also been associated with increased heart rates, elevated rectal
temperatures, and lower skin blood flow (Coyle & Montain, 1992; Noakes, 1995).
However, whether the levels of dehydration present in endurance athletes (1-4% body
weight) pose any major health risks has recently been questioned (Noakes, 1995).

In light of a history which includes ingestion of large amounts of fluid, with
resultant weight gain, and diuresis following exercise in hyponatremic athletes, it appears
that ingestion of increased fluids is a major contributing factor to the development of
HAE during exercise. What remains unclear is the pathology of this fluid retention.
Decreased Gastric Emptying and Intestinal Fluid Absorption

Gastric emptying and intestinal absorption rates of liquids are also implicated in affecting both dehydration and electrolyte status. Gastric emptying of liquids follows an exponential time course with marked variability among subjects (Gisolfi & Duchamn, 1992). Gastric volume plays a major role in regulating the rate of gastric emptying (Minami & McCallum, 1984; Noakes, Reher, & Maughan, 1991; Rehrer, 1994). The importance of maintaining a large gastric volume was shown by Ryan, Bleiler, Carter, and Gisolfi (1989), who had subjects drink 350 ml of fluid every 20 minutes during 3 hours of cycle exercise in the heat (33 °C). Following this regime, subjects emptied between 95% and 99% of the drink ingested. Lambert et al. (1996) noted that exercising subjects who maintained relatively constant stomach volumes over a prolonged period of time produced relatively constant gastric emptying rates.

The addition of a carbohydrate to fluids has been found to decrease the gastric emptying rate. Costill, Krammer, and Fisher (1970) found that the rate of gastric emptying is slowed proportionately with increasing glucose concentration above 8%. However, when gastric fluid volume is maintained at 600 ml or more, most individuals can still empty more than 1,000 ml per hour, when the fluids contain a 4% to 8% carbohydrate concentration (Coyle & Montain, 1992; Noakes et al., 1991).

Once the gastric contents are emptied into the small intestine, this solution must be absorbed before any benefit of hydration is realized. Glucose, salt, and water are transported to cells by transcellular-mediated transport and “solution drag” between brush border cells (Gisolfi & Duchamn, 1992). Gisolfi, Summers, Lambert, and Xia (1998) compared the intestinal fluid absorption of a 6% carbohydrate-electrolyte beverage to
water and found no difference between the two in fluid absorption from the duodenojejunum during exercise. The most effective ratio of glucose to sodium promoting water absorption is 2:1 (Lifshitz & Wapnir, 1984; Modigliani & Bernier, 1971). It has also been shown that a sodium level below 90 mEq/L in the intestinal lumen can impair sodium and water absorption (Spiller, Jones, & Silk, 1987). Moderate exercise appears to have little or no effect on gastric emptying, while heavy exercise at intensities greater than 70-75% VO₂max may slow gastric emptying (Costill & Saltin, 1974).

Ryan et al. (1998) studied the effects of hypohydration on gastric emptying and intestinal absorption during exercise. They concluded that hypohydration to approximately 3% of body weight does not impair gastric emptying or fluid absorption during moderate exercise when ingesting a water placebo and that hyperosmolality (> 400 mosmol) reduced water flux in the small intestine.

Splanchnic ischemia, which may impair enteral absorption, can occur during exercise (Knochel, 1990). Kielblock, Strydom, Burger, Pretorius, and Manjoo (1982) discussed the vascular changes needed to compensate for blood flow changes and demands necessary for exercise. These changes include peripheral vasodilatation and decreased total peripheral vascular resistance, with compensatory splanchnic vasoconstriction. It is possible, therefore, that part of the water ingested by athletes is not absorbed while exercising, but is rather sequestered in the gut, causing third spacing of fluids. With cessation of exercise and reperfusion of the splanchnic bed, this sequestered water could be absorbed rapidly, expanding body fluid volume and contributing to hyponatremia (Clark & Gennair, 1993).
The athlete must take into account the rate of gastric emptying and intestinal absorption when calculating fluid intake to avoid sequestered fluid in the gut. A third space effect would be the likely cause of HAE if hyponatremia developed, regardless of the volume or type of fluid ingested, and the gut would be the most likely site for this movement if the hyponatremia was exacerbated by water ingestion.

**Inappropriate Renal Function**

Another theory of HAE is inappropriate renal function. A normal kidney can excrete 15-20 L of water per day, as long as antidiuretic hormone (ADH) is suppressed (Galun et al., 1991). Water intoxication occurs when free water intake is increased due to the ingestion of large amounts of hypotonic fluids so that even normally functioning kidneys cannot excrete it rapidly enough. This is the equivalent of psychogenic polydipsia (Wolfson, 1995). Speedy et al. (1999) tested post-race serum sodium concentrations in 373 athletes and found the plasma sodium level to be significantly related to hematocrit. Athletes with the lower plasma sodium concentrations had the lower hematocrit, indicating that the hyponatremia was a result of an expansion of the extracellular space. These data support others (Armstrong et al., 1993; Clark & Gennari, 1993; Noakes, 1995), suggesting that HAE is probably a dilutional hyponatremia. In virtually all clinical circumstances in which dilutional hyponatremia develops, there exists a decreased effective renal blood flow (Krumlovsky, 1975).

Irving et al. (1991) provide data to support the theory that inappropriate renal function leads to fluid retention in the vascular space, causing a hypervolemic hyponatremia. They compared data on 8 athletes treated for hyponatremia following the completion of a 90-km ultra-marathon with data of 18 normonatremic athletes who took
part in similar races. The fluid ingestion of the hyponatremic subjects (0.8 to 1.3 L/hr) was greater than the normonatremic subjects (0.6 L/hr) by as much as 7 L over the 10-hour race. The hyponatremic subjects excreted a fluid excess of 2.95 ± 0.56 L during the ± 15.6 hours of recovery, indicating a fluid overload. Renal response during the recovery phase was normal as indicated by the elevated urine output and creatinine clearance. An elevated serum aldosterone concentration was found in all hyponatremic subjects, and increased renin activity was found in 2 subjects. The authors conclude that renal function during the race was clearly inappropriate, but it was unlikely that exercise-induced renal damage was the cause.

Galun et al. (1991) studied the effect of prolonged physical stress on the development of hyponatremia and renal function in 17 trained male subjects during a 24-hour march but were unable to link hyponatremia to renal changes. During the 24-hour march, marchers had five stops of 5-10 minutes every 20 km. All urine was saved throughout the march and during 64 hours of recovery. Serum was collected for analysis at 9 and 16 hours of marching, upon termination, and at 16, 40, and 64 hours of recovery. Creatinine clearance was significantly depressed and negatively correlated with plasma sodium levels. Therefore, marchers with the greatest decrease in creatinine clearance exhibited the highest plasma sodium levels. This inverse correlation indicates that the fall in glomerular filtration rate (GFR) did not account for the hyponatremia. Salt wasting (a negative sodium balance at a normal dietary sodium) could contribute to the development of HAE. However, a significant correlation was found between the amount of salt excreted in the urine and plasma sodium levels, suggesting that the kidney responded normally to changes in plasma. The increase in urine output, the fall in urine
osmolality, and the increase in free water clearance suggest that the renal dilution mechanism was intact and operated in a physiological fashion. Of note, urine osmolarity failed to reach a minimum concentration (urine osmolarity below 100 mOsm/L), indicating that the kidney had an inadequate response to water load leading to the development of hyponatremia. These electrolyte and fluid changes are speculated to be related to high vasopressin levels.

GFR can also be affected by the use of nonsteroidal anti-inflammatory agents (NSAIDS), particularly in people who are dependent on prostaglandin synthesis to maintain renal profusion. Additionally, NSAIDs, by their inhibition of prostaglandin synthesis, potentiate the renal effect of vasopressin (Wen, 1997). Although the kidney plays a major role in maintaining fluid equilibrium in the body, there is no evidence to prove that permanent kidney damage is the causative factor of HAE. However, there is speculation that the stress of endurance exercise can cause transient renal malfunction or reveal an underlying renal deficit.

Inappropriate Endocrine Function

Inappropriately high arginine vasopressin (AVP) levels resulting in the retention of fluids in endurance athletes during prolonged exercise has also been postulated in the development of HAE (Clark & Gennare, 1993; Irving et al., 1991; Zelingher et al., 1996). Armstrong et al. (1993) reported a 460% increase in AVP levels in a hyponatremic runner despite fluid overloads. This AVP increase developed despite a plasma osmolality of 253 mOsm/kg, suggesting that stimulation of this large AVP release was nonosmotic. Nonosmotic causes of AVP secretion include increased angiotensin II, decreased blood
volume, hypotension, increased core body temperature, emotional stress, nausea, and pain (Armstrong et al., 1993; Krumlovsky, 1975; Fried & Palevsky, 1997).

Nelson, Robinson, Kapoor, and Rinaldo (1988) and Noakes (1992) speculated that an expanded plasma volume (due to excess fluid intake) failed to suppress AVP, as occurs in the "syndrome of inappropriate antidiuretic hormone." Speedy et al. (1999) investigated the role of AVP in HAE in a perspective study that compared plasma AVP to post-race weight and plasma sodium concentration in endurance athletes. Of note, plasma AVP levels were not elevated in athletes presenting symptomatic HAE, with the exception of one athlete who presented with an AVP of 59 pmol/L (normal < 9 pmol/L). He was subsequently diagnosed with a nonfunctioning anterior pituitary microadenoma. These results agree with Holtzhausen et al. (1994) who found no differences in AVP levels between collapsed runners and normonatremic controls; however, the two groups had similar numbers of hyponatremic athletes. These data do not support the postulate that inappropriately high AVP concentrations are routinely involved in the etiology of hyponatremia; however, as noted above, some athletes can present with elevated AVP in response to a subclinical disease.

To summarize, the cause of HAE in endurance athletes is not presently known. Reduction of total body sodium through sweating does not seem to be a significant mechanism. A massive uptake of fluids after the completion of the race is also not the total explanation because the renal system should be able to handle such loads without difficulty. Also, data showing a relationship between a decreased hematocrit and decreased serum sodium suggest a dilutional hyponatremia, rather than a hyponatremia primarily from pooling of fluid in the gut. Increased antidiuretic hormone (ADH) release
was not found in a large prospective study, but occasional instances of increased ADH release has been documented in the literature. Therefore, the literature supports the theories of an increase in total body water secondary to ingestion of hypotonic fluids alone or in association with inappropriate renal function as the cause of HAE. These theories guide the current treatment for HAE.

Treatment of Hyponatremia Associated with Exercise

Although athletes present with varied complaints following a race, only treatment of hyponatremia will be discussed. As with most aspects of medicine, specific assessment and treatment protocols cannot be given for every possible combination of problems that might occur.

Treatment for HAE can initiate at any of these three sites, the medical tent at the race site, the emergency room at a hospital, or the primary care provider’s office. Treatment at each site is specific to the presenting symptoms. The history and physical exam should include training and race data noted in Table 6. Appropriate, individualized treatment should then be administered.

Medical Tent Treatment

Treatment for mild hyponatremia generally occurs in a medical tent at the race site and in the past has consisted of monitoring serum sodium levels and administering large amounts of intravenous normal saline (Hiller, 1989; Laird, 1989). Current recommendations for the conscious athletes with mild hyponatremia include assessing for dehydration, resting the athlete in the supine position with legs and hips elevated above the heart, restricting fluids, and checking serum sodium levels a minimum of every hour while waiting for spontaneous diuresis to commence (Shopes, 1997; Holtzhausen &
Noakes, 1997). It would seem prudent to insert a intravenous cannula with a saline lock. The unconscious hyponatremic athlete should be stabilized in the medical tent while alerting on-site paramedics of a high-priority transfer. The goal is to have the athlete transferred to a hospital in a maximum time of 30 min from admission to the medical tent. There is controversy as to when to transfer the athlete with mild hyponatremia (Laird, 1989; Holtzhausen & Noakes, 1997). Transfer should be arranged if the athlete’s serum sodium level continues to fall or if central nervous symptoms develop.

**In-hospital Treatment**

In-hospital treatment involves a rapid assessment of airway, breathing, circulation, mental status, and neurological examination with continuous monitoring of vital signs. A head CT scan, cardiac enzymes, and chest x-ray are ordered as indicated (Maclean et al., 1976; Surgenor & Uphold, 1994). Laboratory studies to be obtained focus on hydration, renal, electrolyte, and endocrine status (see Table 7; Michelis, Warms, & Davis, 1975; Surgenor & Uphold, 1994). An abnormal cortisol level would reveal pituitary or adrenal abnormalities, whereas an abnormal thyroid-stimulating hormone would point to a thyroid disorder. Athletes with acute hyponatremia should have muscle enzymes monitored routinely for at least the first 3-5 days after admission to rule out rhabdomyolysis (Korzets et al., 1996). During the acute phase of the hyponatremic episode, the body is at a critical point with respect to blood and urine chemistries. Electrolyte and hormonal laboratory values found in the hyponatremic state are impossible to duplicate in a euhydrionic state; therefore, it may be advantageous to draw two tubes of heparinized serum which can be frozen at -20 °C for up to 10 days without deterioration in the event further laboratory tests should be ordered.
Treatment for hyponatremia is dependent on the type of presenting hyponatremia. As cited previously in this paper, HAE is primarily a dilutional hyponatremia of rapid onset (< 24 hr). Vigorous routine treatment of athletes with large amounts of intravenous fluids for assumed dehydration can exacerbate encephalopathy and have fatal consequences (Noakes, 1999). Current recommendations for treatment of dilutional hyponatremia primarily consist of water restriction. A slow infusion of hypertonic saline can be initiated if serum sodium levels are low (< 120), but, more importantly, hypertonic normal saline should be started if neurological symptoms are present (Berry & Belsha, 1990; Culpepper, Clements, & Pence, 1994; Ellis, 1995; Fraser & Arieff, 1997; Sterns, 1994; Zarinetchi & Berl, 1996). To avoid potential complications from osmotic demyelination syndrome, a maximal correction of 1 to 2 mEq/L/hr using a 3% normal saline should be used (Arieff, 1990). During the interval that active correction of symptomatic hyponatremia is being carried out, frequent mental status examinations as well as monitoring of plasma electrolytes should be completed every 2 hours, with appropriate adjustments in the infusion rate until the patient becomes neurologically stable and has reached the desired therapeutic goal.

Therapy with hypertonic NaCl should be discontinued when (a) the patient becomes asymptomatic, (b) the patient’s plasma sodium has increased by 20 mmol/L, or (c) the plasma sodium reaches a value in the range of 120-125 mmol/L. These guidelines may be modified if patients are symptomatic at higher levels of plasma sodium (124-131 mmol/L; Fraser & Arieff, 1997; Rutecki & Whittier, 1994).
Clinic Treatment

Athletes present to their primary health care provider in clinic settings either as a follow-up or routine visit to address concerns. These patients are rarely symptomatic but are wanting answers to their questions concerning diagnosis, training, and fluid replacement regimen. The primary care provider must compile and review the athlete’s medical data and relate it to the hyponatremic episode. The physical exam should focus on any neurological, endocrine, or renal deficits. Labs should include CBC, serum electrolytes, creatinine, BUN, and serum osmolality. Thyroid-stimulating hormone and cortisol levels should also be drawn if not previously measured. A urinalysis, urine osmolality, and urine sodium should also be performed to screen or rule out other causes (Michelis et al., 1975).

Patient Teaching

Patient education is a mainstay of patient care. Upon discharge from the medical tent or hospital, there is little or no patient education concerning the cause and prevention of further hyponatremic episodes, and what education athletes receive may be erroneous. The primary care provider will need to inform the athlete of training options available, which can decrease the chance of incurring another episode of HAE.

Treatment plans for fluid replacement. Athletes should be taught that too much water is harmful. Noakes et al. (1985) theorized that slower runners demonstrate a decreased production of sweat, decreased nausea and less inhibition of gastric emptying, increased absorption from the gastrointestinal tract, and increased total running time allowing for increased ingestion of hypotonic fluids, all leading to overhydration. He
proposed a 500 ml/hr fluid replacement regimen during exercise for athletes at risk of hyponatremia (Noakes et al., 1991).

Barr et al. (1991) and Irving et al. (1991) suggest that the athlete should perform pre and postexercise weight checks and replace the amount of fluids lost on an hourly basis. Montain et al. (1999) has published data relating heat categories to work intensity, work-rest cycle, and water intake. They propose a minimum fluid replacement of 0.5 quarts/hr and maximum of 1.5 quarts/hr with a total daily fluid intake not to exceed 12 quarts. The athlete must be taught to replace only the fluids which are lost. In training the athlete should choose one of the previously mentioned methods of monitoring fluid intake and incorporate it into the training regimen.

**Use of a carbohydrate and electrolyte solution versus plain water.** A second controversy in fluid replacement is the use of a glucose/electrolyte solution versus plain water. In 1996, the American College of Sports Medicine printed a position statement on fluid replacement and exercise. It was proposed that athletes should consume adequate fluids during the 24-hour period before an event and 500 ml of fluid 2 hours before exercise. Athletes should then ingest 600-1,200 ml of a solution containing 4% to 8% and 0.5-0.7 g of sodium per liter of water in exercises lasting over 1 hour (Convertino et al., 1996). Barr et al. (1991) studied the heart rate, body temperature, plasma volume, plasma sodium, and aldosterone levels in 8 athletes who completed 6 hours of exercise in an environmental chamber maintained at 30 °C and 50% relative humidity. The subjects attempted three 6-hr trials, ingesting plain water (Trial W), a 25 mmol/L NaCl solution (Trial S), and no fluids (Trial NF). Subjects acted as their own controls. Only 1 subject
was able to complete the no-fluid trial. Results from the Group S and Group W showed no significant differences between fluid replacement with water or saline on all the variables, including plasma sodium. The results of this study

"indicate that a need for sodium replacement would be unexpected in exercise of less than six hours duration, assuming plasma sodium is normal at the start of exercise. Moreover, the amount of sodium in commercial beverages is inadequate to prevent a decrease in plasma sodium.” (Barr et al., 1991, p. 811)

A number of other studies have not been able to demonstrate an effect of beverage sodium content on plasma concentration (Brandenberger, Candas, Follenius, & Kahn, 1989; Cade, Spooner, Schlein, Pickering, & Dean, 1972; Greenleaf & Brock, 1980; Powers et al., 1990).

Vrijens and Rehrer (1999) studied the effect of distilled water (W) and a sodium-containing beverage (G), Gatorade, on serum sodium and glucose in 10 subjects in a controlled environment of 34 °C and 65% relative humidity. All subjects performed in all trials. Subjects were instructed to drink 1 L of Gatorade the evening before each trial and had unlimited access to water before the start of each trial. The length of each trial was 3 hours, and fluid was given every 15 minutes, equal to an individually predetermined fluid loss. The results demonstrated sodium concentrations were decreased to a greater extent with distilled water (W) than with a sports drink (G). Of note, 1 subject became hyponatremic on the W trial, but the same subject was also unable to finish the G trial due to nausea, which may have been an early symptom of hyponatremia. The authors provide the pretrial (plasma sodium 144 mmol/L) and
posttrial (plasma sodium 135 mmol/L) levels for the W trial, but do not provide the pre and posttrial plasma sodium levels for the G trial of the subject who developed hyponatremia.

Electrolyte/carbohydrate solutions have been shown to be of benefit in improving intestinal absorption of water, maintaining glucose concentration, and enhancing carbohydrate oxidation (Coggan & Coyle, 1991; Murray, Paul, Seifert, & Eddy, 1991; Schedl, Maughan, & Gisolfi, 1994). However, they have not been shown to decrease the possibility of hyponatremia in the athlete at risk for developing hyponatremia.

Implications for Practice

Hyponatremia associated with exercise is a serious complication of exercise. The cause remains unknown but is probably due to ingestion of large amounts of hypotonic fluids. Athletes should be taught to avoid HAE by learning to monitor their fluid needs during exercise. This can be done by monitoring weight losses during swimming, cycling, and running in training and replacing lost fluids on an hourly basis according to average weight loss during exercise. Athletes should also consider their maximal rate of intestinal fluid absorption and not simply rates of sweating, urination, or gastric emptying when making decisions concerning the amounts of fluids to ingest during exercise. It is important to realize that even sports drinks are hypotonic to plasma. Studies have been unable to show a difference on serum sodium with the ingestion of water and an electrolyte solution; however, the American College of Sports Medicine along with leading experts recommend the use of an electrolyte solution as a significant part of fluid replacement. A prerace meal which is high in sodium can also be beneficial in providing extra salt in the colon for use during the race. Acclimatization is necessary if
participating in a race in another geographical environment. Acclimatization transpires over a period of more than 4 to 9 days and is not complete for up to 30 days. The athlete would benefit from presenting to the race location a minimum of 4 or more days prior to the race day to become acclimatized.

If the athlete develops acute symptomatic HAE, medical personnel should realize that HAE can develop into a medical emergency. Athletes experiencing symptoms should be assessed for hydration status before intravenous treatment is initiated. If the athlete is well hydrated, it can be assumed that the hyponatremia is dilutional and the infusion of an intravenous solution can cause encephalopathy, respiratory arrest, and death. If the athlete is experiencing central nervous symptoms, an IV of a 3% hypertonic saline solution should be initiated at a slow rate, and the athlete should be transferred to a medical facility. Many athletes, if not presenting central nervous system symptoms, will recover with fluid restriction alone followed by a spontaneous diuresis between 4 to 6 hours following the onset of symptoms.

Based on the review of literature and recommended treatments, and with skillful follow through and patient teaching by the primary care provider, the athlete will be able to manage HAE, compete successfully, and complete the endurance activity of choice.

Conclusions

In the case presented here, a review of C. N.’s fluid ingestion (see Table 8) reveals a consistent excess fluid intake of up to 4-6 L. The one serum and urine osmolality comparison shows an increased urine osmolality (see Table 1), pointing to conservation of fluids in the kidney. Having an arginine vasopressin level drawn during a hyponatremic episode would be valuable in differentiating out the cause of HAE in C. N.
Together these findings point to increased ingestion of hypotonic fluids associated with questionable kidney dysfunction as the causative factor for C. N.’s episodes of HAE.

Currently C. N. has begun exercising at a low intensity again with the goal of attempting a second Ironman.
REFERENCES


after an ultradistance multisport triathlon. Clinical Journal of Sports Medicine, 7(2), 100-103.


APPENDIX
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## Table 2

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<td>1.35 mlU/L</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>T4</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>6.2 µg/dl</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Urine</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>582 mOsm/kg</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Osmolality</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Random Urine Creatinine</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>222 mg/dl</td>
<td></td>
<td>21 mEq/L</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Random Urine Sodium</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>21 mEq/L</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cortisol (am)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>16 µg/dl</td>
<td></td>
</tr>
</tbody>
</table>

<sup>a</sup>PCP = Primary Care Provider
<sup>b</sup>SMD = Sports Medicine Doctor
### Table 3

**Symptoms of Hyponatremia and Hypochloremia**

<table>
<thead>
<tr>
<th>Hyponatremia (serum sodium &lt; 135 mEq/L)</th>
<th>Hypochloremia (serum chloride &lt; 95 mEq/L)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anorexia</td>
<td>Weight gain</td>
</tr>
<tr>
<td>Nausea</td>
<td>Hypertension</td>
</tr>
<tr>
<td>Vomiting</td>
<td>Restlessness</td>
</tr>
<tr>
<td>Abdominal cramping</td>
<td>Confusion</td>
</tr>
<tr>
<td>Headache</td>
<td>Disorientation</td>
</tr>
<tr>
<td>Dizziness</td>
<td>Lethargy</td>
</tr>
<tr>
<td>Tachycardia</td>
<td>Seizures</td>
</tr>
<tr>
<td>Postural hypotension</td>
<td>Coma</td>
</tr>
</tbody>
</table>

*The same signs/symptoms of hyponatremia plus:*

- Muscle Weakness
- Twitching
- Tetany
- Slow, shallow respirations
- Respiratory arrest

### Table 4

**Differential Diagnosis of Collapsed Athlete**

<table>
<thead>
<tr>
<th>Nonexercise-Related Disorder</th>
<th>Exercise-Related Disorder</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cardiac Arrest</td>
<td>Heat Stroke</td>
</tr>
<tr>
<td>Grand Mal Epilepsy</td>
<td>Heat Exhaustion</td>
</tr>
<tr>
<td>Subarachnoid Hemorrhage</td>
<td>Hyponatremia</td>
</tr>
<tr>
<td>Diabetic Coma</td>
<td>Hypoglycemia</td>
</tr>
<tr>
<td></td>
<td>Dehydration</td>
</tr>
<tr>
<td></td>
<td>Exercise-Associated Collapse</td>
</tr>
</tbody>
</table>

### Table 5

**Signs and Symptoms Differentiating Exercise-Related Conditions of Collapse During Endurance Exercise**

<table>
<thead>
<tr>
<th>Condition</th>
<th>Temperature</th>
<th>Heart Rate</th>
<th>Blood Pressure</th>
<th>Mental Status</th>
<th>Serum Sodium</th>
<th>Serum Glucose</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dehydration</td>
<td>$&lt; 39.4 , ^\circ C$</td>
<td>↑</td>
<td>↓</td>
<td>Alert Disoriented</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>Heat Stroke</td>
<td>$&gt; 40 , ^\circ C$</td>
<td>↑</td>
<td>↓</td>
<td>Significant Alteration</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>Heat Exhaustion</td>
<td>$&lt; 39.4 , ^\circ C$</td>
<td>↑</td>
<td>↓</td>
<td>Intact</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>Hyponatremia</td>
<td>$&lt; 40 , ^\circ C$</td>
<td>–</td>
<td>–</td>
<td>Alert Disoriented Coma</td>
<td>$&lt; 135$ mEq/L</td>
<td>–</td>
</tr>
<tr>
<td>Hypoglycemia</td>
<td>–</td>
<td>↑</td>
<td>↓</td>
<td>Confusion Coma</td>
<td>–</td>
<td>$&lt; 60 , \text{mg/dl}$</td>
</tr>
<tr>
<td>Exercise-Associated Collapse</td>
<td>$&lt; 39 , ^\circ C$</td>
<td>↑</td>
<td>↓</td>
<td>–</td>
<td>–</td>
<td>–</td>
</tr>
</tbody>
</table>

**Note.** Compiled from Arnheim and Prentice (1997); Dambro and Griffith (1996), Ferri, Danakas, Masci, Mercier, and Olson (1999); Holthausen and Noakes (1977); and Thein (1995).

$\rightarrow$ = within normal limits

↑ = elevated

↓ = decreased
Table 6

**Information Required from Each Athlete Admitted With Collapse**

<table>
<thead>
<tr>
<th>Pertinent History</th>
<th>Adequate Examination</th>
</tr>
</thead>
<tbody>
<tr>
<td>Date, length, and time to finish race</td>
<td>Level of consciousness/mental state</td>
</tr>
<tr>
<td></td>
<td>State of hydration</td>
</tr>
<tr>
<td>Amount of fluid ingested during the race</td>
<td>Rectal temperature</td>
</tr>
<tr>
<td>Amount of urine passed during the race</td>
<td>Heart rate, supine and erect</td>
</tr>
<tr>
<td>Presence of vomiting and/or diarrhea before and during the race</td>
<td>Blood pressure, supine and erect</td>
</tr>
<tr>
<td>Amount of carbohydrate ingested before and during the race</td>
<td>Blood glucose concentration</td>
</tr>
<tr>
<td>Drugs taken during the race</td>
<td>Serum sodium concentration</td>
</tr>
<tr>
<td>Recent intercurrent illness</td>
<td></td>
</tr>
<tr>
<td>Race preparation: heat acclimatization, training schedule, distance training</td>
<td></td>
</tr>
</tbody>
</table>

### Laboratory Tests to Define HAE

<table>
<thead>
<tr>
<th>Status</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hydration</td>
<td>CBC</td>
</tr>
<tr>
<td></td>
<td>Hemoglobin</td>
</tr>
<tr>
<td></td>
<td>Hematocrit</td>
</tr>
<tr>
<td>Electrolyte</td>
<td>Serum Electrolytes</td>
</tr>
<tr>
<td></td>
<td>Arterial Blood Gases</td>
</tr>
<tr>
<td>Endocrine</td>
<td>Serum Glucose</td>
</tr>
<tr>
<td></td>
<td>Thyroid Stimulating Hormone</td>
</tr>
<tr>
<td></td>
<td>Arginine Vasopressin</td>
</tr>
<tr>
<td></td>
<td>Cortisol</td>
</tr>
<tr>
<td>Renal Function</td>
<td>BUN/Creatinine</td>
</tr>
<tr>
<td></td>
<td>Urine Osmolality</td>
</tr>
<tr>
<td></td>
<td>Urine Sodium</td>
</tr>
</tbody>
</table>
### Table 8

**Actual Versus Suggested Intakes for Duration of Exercise for Patient in Case Study**

<table>
<thead>
<tr>
<th>Location</th>
<th>Actual Intake</th>
<th>Suggested Intake Montain et al.</th>
<th>Difference Actual &amp; Suggested</th>
<th>Suggested Intake Noakes et al.</th>
<th>Difference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Wildflower</td>
<td>5,000 ml</td>
<td>4,875 ml</td>
<td>+ 125 ml</td>
<td>3,250 ml</td>
<td>+ 1,750 ml</td>
</tr>
<tr>
<td>Wallowa Lake</td>
<td>10,080 ml</td>
<td>6,000 ml</td>
<td>+ 4,080 ml</td>
<td>4,000 ml</td>
<td>+ 6,080 ml</td>
</tr>
<tr>
<td>4-hr Bike</td>
<td>4,320 ml</td>
<td>4,000 ml</td>
<td>+ 320 ml</td>
<td>2,000 ml</td>
<td>+ 2,320 ml</td>
</tr>
<tr>
<td>IMC</td>
<td>8,640 ml</td>
<td>6,750 ml</td>
<td>+ 1,890 ml</td>
<td>4,500 ml</td>
<td>+ 4,140 ml</td>
</tr>
</tbody>
</table>

**Notes.** Montain based on Wet Bulb Globe Temperature index and work intensity.

Noakes based on average intake of nonhydroneutremic athletes.