Exertional hyponatremia is a plasma sodium concentration less than 135 mmol/L (normal is 135–146 mmol/L). The condition most commonly occurs in low- to moderate-intensity events lasting 4 hr or more in a hot environment. Also known as water intoxication, exertional hyponatremia is associated with a low-sodium diet or insufficient consumption of sodium or rehydrating with an inappropriate type or volume of fluids. These etiological factors alone or in combination might result in sodium dilution that can detrimentally affect fluid balance. Excess water in the extracellular space or large deficits in extracellular sodium concentrations result in neurological disturbances that are often confused with signs and symptoms of dehydration or heat illness. Unrecognized or untreated exertional hyponatremia can result in central-nervous-system dysfunction, convulsions, cerebral edema, pulmonary edema, and death. The purpose of this article is to describe exertional hyponatremia, identify athletes who might be at risk, and provide certified athletic trainers with useful information for preventing, recognizing, evaluating, and managing this potentially life-threatening condition.

Key Points

- Awareness of hyponatremia as a disorder affecting physically active individuals will increase the probability of early recognition, appropriate treatment, and effective prevention by members of the sports-medicine team.
- Recognition of the potential for developing exertional hyponatremia, its consequences, and its differences from exertional heat illness are essential for preventing, evaluating, and treating the condition.
- Individualized rehydration protocols can reduce the occurrence of life-threatening complications associated with exertional hyponatremia.
- Key Words: water intoxication, prolonged exercise, electrolyte replacement

Populations at Risk

Excessive fluid intake in athletes, hikers, and soldiers can be attributed to a recent heightened emphasis on water consumption for the prevention of heat illness and widespread ignorance of the potential for exertional hyponatremia. Increasing awareness of hydration as a preventive measure for heat illness, as well as the increased popularity of endurance events, might have lead to the recent increase in the incidence of exertional hyponatremia. Development of exertional hyponatremia depends on the type and duration of the event, environmental conditions, and body type and size, as well as hydration and nutritional status. In ultradistance events, asymptomatic exertional hyponatremia is relatively common, and symptomatic hyponatremia is a less common but potentially serious condition. Accurate estimation of its prevalence depends on recognition of symptoms, which affect 1–29% of finishers of such events. In general, at-risk individuals consume and retain large quantities of water while performing long-duration, low-intensity exercise, which might be intermittent in nature (i.e., stopping for rest or at rehydration stations).

Hyponatremia unrelated to exertion has been reported in the medical literature and is most often associated with psychogenic polydipsia (compulsive water drinking), excessive water intake to alter the results of drug tests, infants fed excessive amounts of water when infant formula was unavailable, or a syndrome of inappropriate antidiuretic-hormone secretion. Case studies of exercise-induced or exertional hyponatremia were first reported in 1985 among athletes.
competing in endurance races. Military recruits, hikers and backpackers, ultraendurance athletes, and adventure athletes have succumbed to overhydration and the resultant exercise-related hyponatremia. Hyponatremia might be occurring more often than initially realized and has been reported to occur during relatively short-distance events like marathons.

Individuals at risk for exertional hyponatremia are typically physically active people who might be aware of standard recommendations for fluid replacement but are unable to accurately estimate sweat losses in relation to fluid and electrolyte ingestion. Athletes are often advised to drink as much as possible, and some athletes drink so much water during prolonged exercise that potentially fatal consequences result. Severe hyponatremia has been induced by water intake of as little as 1 L/hr. Experienced male runners who collapsed during or after an 88-K race were found to be hyponatremic (average plasma sodium concentration of 122.4 ± 2.2 mmol/L) as a result of consuming fluid at a rate of 0.8 to 1.3 L/hr, compared with the maximum intake rate of 0.6 L/hr in normonatremic runners. Low-intensity, long-duration events that progress at a very slow pace with access to ample water-consumption opportunities could put athletes at risk for exertional hyponatremia. If athletes drink beyond their fluid needs during such events, intestinal absorption of fluid causes excess fluid to enter the extracellular space.

Fluid-replacement recommendations must take into account body size, sweat rate, exercise duration and intensity, acclimatization status, environmental factors, and individual preferences in a manner consistent with the recommendations of the National Athletic Trainers’ Association (NATA). Individualized rehydration addresses those with low sweat rates that might dangerously overhydrate and might present risk for exertional hyponatremia, as well as high sweat rates that might present risk for dehydration and exertional heat illness. Female athletes are at a significantly greater risk of exertional hyponatremia, perhaps because of smaller body size, lower sweat rate, lesser fluid requirements, or longer time for completion of races and possible antidiuretic or ovarian-hormone influences. Hyponatremic marathoners are more likely to be women, users of nonsteroidal anti-inflammatory drugs, and slower race finishers than are normonatremic runners. Speedy et al. reported that women are at significantly greater risk than men for exertional hyponatremia because of fluid overload in ultradistance triathlons. Greater risk among slow runners might be caused by greater opportunity for overhydration, greater absorption of fluid in the digestive tract, lack of sodium consumption during an event, and lesser fluid requirements (as a result of lower activity intensity). Rehydration protocols at the low end of the American College of Sports Medicine guidelines (fluid replacement of 0.6–1.2 L/hr of activity) should be followed, but individual fluid needs must be considered to avoid fluid overconsumption or sodium deficits among athletes who are competing at low intensity for long durations or among small athletes who have low sweat rates and low metabolic rates.

**Recognition and Evaluation**

Symptoms of exertional hyponatremia appear to be associated with the degree or magnitude of plasma sodium deficit. Plasma sodium concentrations < 125 mmol/L are reported to produce more severe symptoms (e.g., altered consciousness, coma, convulsions, altered cognitive functioning), whereas most athletes with plasma sodium concentrations > 130 mmol/L have mild symptoms or no symptoms. Mild exertional hyponatremia (plasma sodium concentration of 130–134 mmol/L) is often asymptomatic or associated with mild symptoms of headache, nausea, or dizziness. More severe hyponatremia (< 130 mmol/L) results in more severe symptoms such as central-nervous-system dysfunction, which might erroneously be attributed to dehydration or heat stroke (Table 1). It is difficult to distinguish signs and symptoms of hyponatremia from dehydration or heat stroke in the field, because there is considerable overlap in identifying characteristics and symptoms (Table 1). An accurate assessment and differential diagnosis are essential for initiation of an appropriate treatment protocol. Severe symptoms of hyponatremia are similar to the symptoms of heat stroke, which involve central-nervous-system manifestations that might be related to cerebral edema associated with osmotic swelling of brain tissue. These severe symptoms constitute a medical emergency and can result in seizures, loss of consciousness, pulmonary edema, respiratory arrest, brain-stem herniation from increased intracranial pressure, coma, brain damage, and death. Hyponatremic individuals often experience an increase in the number and intensity of symptoms 0.1–6.0 hr