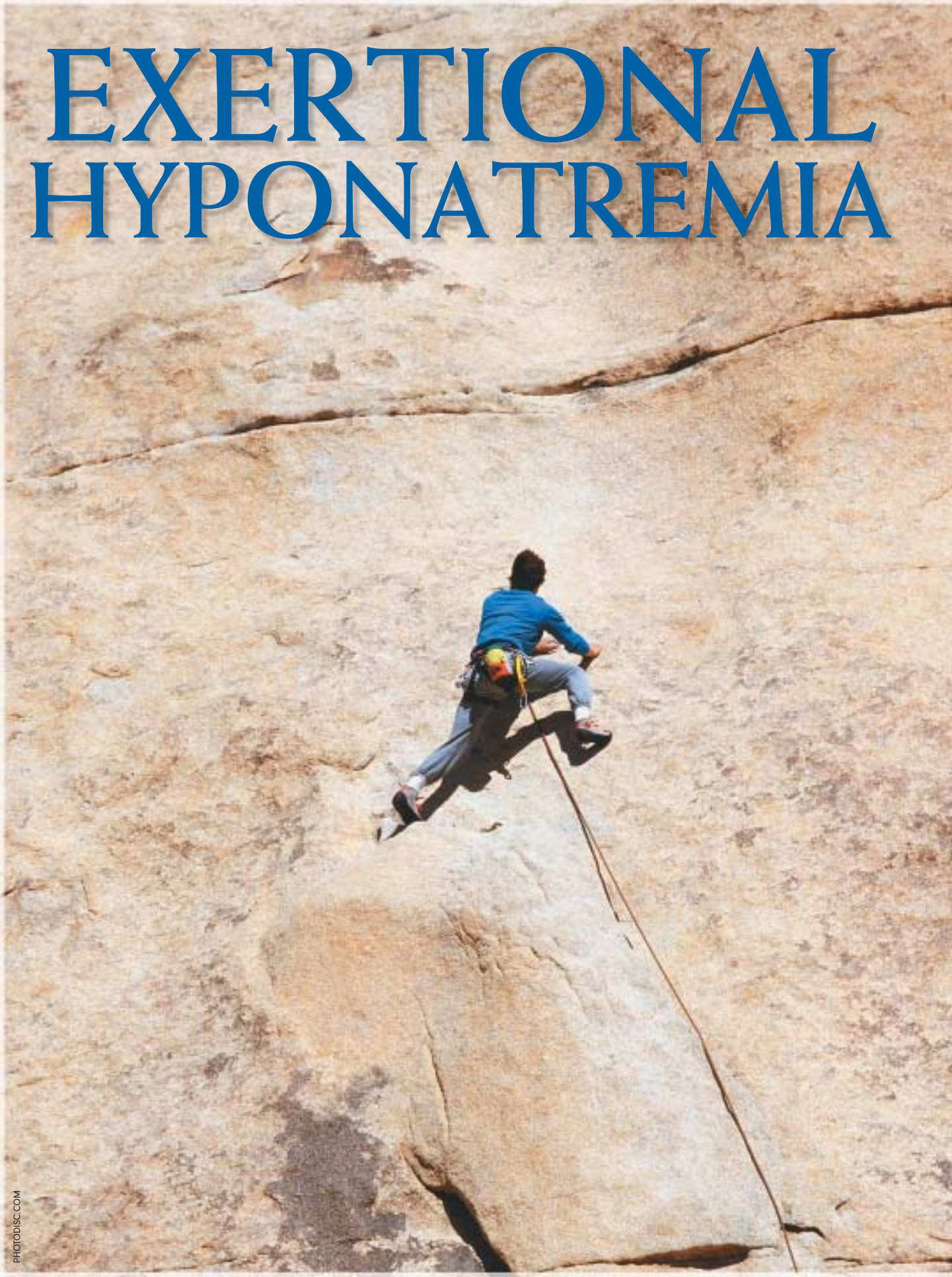
EXERTIONAL HYPONATREMIA



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The *other* heat-related emergency

**By Sherrie Collins, NREMT-P, WEMT,
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The setting: Grand Canyon National Park, July 7, 2003. It's 1400 HRS on a hot sunny afternoon. Rangers receive a report of a 35-year-old man collapsed on the trail, six miles below the rim. The temperature is 102° F with 22% humidity and light winds. Rangers from Phantom Ranch, two miles away, are the initial responders. The ETA for the park medical helicopter is 20 minutes.

The first-arriving ranger EMTs find a semiconscious male lying alongside the trail in the shade. The patient moans and flails all extremities to painful stimuli. *Vitals:* HR: 94; RR: 24; BP: 140/84. The patient's lungs are clear, and he has warm, pale, dry skin.

According to his hiking partner, the patient felt weak and tired for the last three hours of their hike. He has consumed 8 L of water since starting the descent into the canyon that morning, and eaten two apples, a bagel and some trail mix. He had been sitting at this location for the past two hours, complaining of nausea, fatigue and a feeling of impending doom, when he suddenly experienced what his hiking partner describes as seizure activity. Other hikers ran for help. The patient has not regained consciousness (30 minutes ago).

The EMT rangers place the patient on his side and administer oxygen to maintain his airway. A rectal temperature check indicates a core body temperature of 99° F, ruling out heatstroke.

Paramedics arrive via helicopter. They intubate the patient, establish an IV of normal saline and obtain an I-STAT blood chemistry: BGL = 86; Na = 125; K = 4.1; BUN = 15. Initial assessment is acute exertional hyponatremia.

The patient is packaged for helicopter transport. During loading, the patient becomes combative. The crew administers 2.5 mg Midazolam HCL via IVP. The patient arrives at a regional hospital 45 minutes later with his condition unchanged. He's admitted to the ICU, where his sodium levels are restored slowly over the next 36 hours. The patient awakens 48 hours later with retrograde amnesia, but no other neurological impairments.

Center for Emergency Medicine
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Objectives

- Describe the pathophysiology of exertional hyponatremia.
- Explain the mechanisms by which the body cools itself.
- List the treatment options for exertional hyponatremia.

Discussion

The effects of low sodium levels, or hyponatremia, on athletes and outdoor enthusiasts have been increasingly recognized as a source of illness and injury. Sometimes called *water intoxication* (due to neurological symptoms resembling alcohol intoxication), the mechanism is far more complex than simply drinking too much water.

Ultra-endurance events, including marathons, triathlons and even the popular "eco-challenges," are resulting in increased hyponatremia incidents. Last year, there were 32 documented cases of exertional hyponatremia in hikers at Grand Canyon National Park, seven of whom were admitted to the ICU with dangerously increased intracranial pressure (ICP).

Exertional hyponatremia is not limited to endurance events and can occur in any situation in which sodium levels become dangerously low. However, the incidents are most common in heat-stress environments. EMS providers must understand the physiology and mechanism of this heat-related illness that often mimics both heatstroke and heat exhaustion. We also need to promote public education about this life-threatening emergency and its prevention.

PHOTO BETSY HEIGHTMAN

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Systemic response to heat

Let's look at how our bodies respond to a heat-challenge situation. To understand dehydration and electrolytes in the context of exertional heat illness, you must understand the mech-

Distribution of Cardiac Output in Normal Human Subjects at Rest & after 10 Minutes of Strenuous Exercise[2]

Vascular System	Bed Rest	10 Minutes of Exercise
Skeletal muscle	1,200 mL/min. (21%)	12,500 mL/min. (71%)
Splanchnic	1,400 mL/min. (24%)	600 mL/min. (3%)
Renal	1,100 mL/min. (19%)	600 mL/min. (3%)
Cerebral	750 mL/min. (13%)	750 mL/min. (5%)
Coronary	250 mL/min. (4%)	750 mL/min. (5%)
Skin	500 mL/min. (9%)	900 mL/min. (11%)
Other organs	600 mL/min. (10%)	400 mL/min. (2%)
Cardiac output	5,800 mL/min. (100%)	16,500 mL/min. (100%)

anisms of hypovolemic shock and the sequence of events in multiple organ systems.

Severe dehydration is a form of hypovolemic shock, with widespread hypoperfusion of cells and tissues due to reduction in fluid volume. Dehydration is a progressive disorder that, if not halted, spirals downward into deeper levels of hemodynamic and metabolic deterioration. Exertion can further exacerbate the hypovolemia in the system. Moderate exercise in hot conditions may result in:

1. Large fluid shifts into the skin and muscle blood flow that increase core hypoperfusion;
2. Fluid loss through the respiratory tract and secretion of sweat;
3. Reduced plasma volume;
4. Displacement of large amounts of fluid from the gastrointestinal and other splanchnic areas; and
5. Antidiuretic hormone (**ADH**) release.

Blood flow

It is important to recognize that exertion in a hot environment results in various systems and organs vying for blood supply. The losers are the gut and kidneys. The gut can become ischemic, with slowed gastric emptying. In this situation, the Gatorade you've been drinking just sits there. As volume shock continues, the body's fluid, which usually moves from the core outward, will instead be shunted from distal sites to the core (see Table above left).

The long-term risk of this is increased core temperature (hyperthermia). Because the maximum rate of gastric emptying is much less than the maximum sweat rate (1.2 vs. 3.71 L per hour), rehydration cannot keep pace with sweat losses under those conditions. Rehydration and replacement of electrolytes lost in sweat via

Glossary

ADH: An antidiuretic hormone, also known as vasopressin.
AVPU scale: A system for rating level of consciousness. The scale indicates a patient's verbal, motor and pain response to stimulation. Usually coupled with a Glasgow Coma Scale rating.
Myelinolysis: A pathologic process that dissolves the myelin sheaths around certain nerve fibers, such as those of the pons in alcoholic or undernourished people who are afflicted with central pontine myelinolysis.
Osmosis: The movement of a pure solvent, such as water, through a semipermeable membrane from a solution that has a lower solute concentration to one that has a higher solute concentration.
Osmolarity: The osmotic concentration of ions in a solution, which is expressed as osmoles of solute per liter (L) of fluid.
Paresthesia: An abnormal sensation, such as numbness, burning, prickling or tingling.

oral intake may require 36 hours (with occasional urination) to complete.

Sweat is a hypotonic solution containing from 10–90 mEq/L of sodium. Other solutes lost in sweat include potassium and chloride. Well-conditioned athletes who have acclimated to hot environments are at a decreased risk of developing heat-related illness compared with their sedentary counterparts because their sweating starts earlier, and, as the sweat rate increases, the electrolyte content of the sweat decreases.[1]

Fluid and electrolyte losses with heavy sweating are associated with increased muscular fatigue and decreased performance. Large imbalances affect the nervous system and may result in bad judgment with associated injuries. Changes in cognitive function seem to occur before the development of the physical symptoms associated with heat stress and may be due specifically to hyponatremia.

Time distortion, memory impairment, deterioration in attention and decreased ability to calculate mathematical problems are frequent cognitive characteristics associated with heat stress and hyponatremia. In humid conditions, the rate of sweating is higher and the cooling rate is less.

It is therefore critical to understand mechanisms of heat loss and how to promote normal fluid volume when exerting oneself in a hot environment. The next section describes how the body corrects itself when in an overheated state.

Facilitation of heat loss

Radiation: Heat is exchanged between all objects through electromagnetic waves. Through these waves, heat is transferred from warm objects to colder objects. When the ambient temperature is lower than body temperature, heat is released from the body to the ambient environment. This exchange does not require molecular contact. However, if the objects in the environment are warmer than body temperatures, no net heat

loss from the body occurs. This explains why thermoregulation is more efficient in the shade than in direct sunlight. The sun warms objects through radiation, and the objects in the environment are cooler when shaded. If they are cooler than body temperature, heat can be released. Generally, 50% of body heat is released through radiation. This avenue is lost when the ambient temperature exceeds body temperature.[2]

Rehydration & replacement of electrolytes lost in sweat via oral intake may require 36 hours to complete.

Conduction: The direct transfer of heat from one object to another, or conduction, requires molecular interaction. One of the first responses to increases in body temperature is vasodilation. This enables the body to transfer most of the core heat to the surface rapidly. The heat is transferred from surface molecules on the skin directly to clothing or air molecules. Again, this transfer depends on the environmental objects being cooler than body temperature. This phenomenon explains the cooling benefit of a wet shirt or wet towel.[2]

Convection: The motion of fluids (gases or liquids) and their ability to carry molecules of higher temperature away from the body and replace them with cooler molecules is known as convection. This process is co-dependent on conduction for actual heat loss.

The air or water molecules immediately adjacent to the skin are warmed by conduction. The convective currents then move these molecules away and replace them with other molecules that can be heated. If these convective currents are not present, the warmed molecules of air or water remain adjacent to the warm body and actually provide a layer of insulation that impedes further heat loss. Together, conduction and convection account for 15% of heat loss.[2]

Vaporization/evaporation: Although evaporation accounts for only 30% of heat loss in normal situations, it is the principal physiologic defense mechanism against hyperthermia because it does not depend on the ambient temperature to have an effect on the body. As body temperature increases, the hypothalamus stimulates the cholinergic sympathetic fibers, which, in turn, stimulate the 3 million sweat glands on the human body. This stimulus causes the release of a hypotonic saline solution, which is then vaporized on the skin surface.

For every 1 gram of saline solution vaporized, approximately 0.6 kcals of heat are removed. Because this occurs on the skin surface, it is the skin that is cooled. The cool skin now cools the blood circulating in the skin's surface by convection and conduction.

At high ambient temperatures, radiation, conduction and convection all serve to *increase* body temperature because the objects around the body are as hot or hotter. In this situation, only evaporation will cool the body. Studies show that the rate of sweating

is directly proportional to the ambient temperature.

The major limitation of this evaporation is humidity—the amount of moisture in the air. For a given temperature, the air holds only so much water. As this limit is reached, relative humidity increases. As the vapor pressure in the air approaches that on the skin, less and less sweat evaporates. Sweating, by itself, is not enough to release heat, and, in fact, if sweat rolls off or is wiped off, its ability to release heat is lost.[2]

Exercise-induced dilutional hyponatremia

Water intoxication is an old term for exercise-induced dilutional hyponatremia, which occurs when the proportion of total body water (TBW) to total sodium is excessive and a hypervolemic hyponatremia develops. Several mechanisms are responsible for this.

The first problem is drinking too much free water and not replacing lost

PHOTO COURTESY NATIONAL PARK SERVICE

EMS and National Park Service personnel carry a rescued hiker to a location accessible for aero-medical evacuation. Last year, there were 32 documented cases of exertional hyponatremia in hikers at Grand Canyon National Park.

electrolytes through electrolyte-supplemented fluids, such as Gatorade or salted water. The second is third-spacing of hypotonic fluids in the gut during exertion. The third problem is the development of a condition known as syndrome of inappropriate antidiuretic hormone (ADH) response (SIADH). All three mechanisms can work together.

If you treat a heat-exhausted patient with hydration & rest, he will get better. If you treat a hyponatremic patient with fluids & rest, he will get worse.

In addition, sodium loss through sweating and exercise with no electrolyte replacement can result in both dehydration and hyponatremia, although the symptoms of hyponatremia are mild.

The role of ADH

Osmolarity is the concentration of ions in any given solvent. Decreased osmolarity in the extracellular compartments may cause a fluid shift into the cells, resulting in swelling of cells. Swelling of brain cells results in the same symptoms as increased intracranial pressure, such as headache, confusion and seizures. As plasma osmolarity increases above normal (275–295 mOsm/kg), the ion concentration is increased and ADH is released.

ADH prevents loss of water by the kidneys, thus conserving more water in the body and decreasing plasma osmolarity. When we are dehydrated, ADH helps conserve water and produces thirst to get us to drink.

An interesting phenomenon occurs in people who are normovolemic and hydrated: ADH continues to act to conserve water, creating hyperhydration and dilution of plasma sodium. Why would ADH continue at inappropriate levels if extracellular fluid volume (ECFV) returns to normal? Nausea, vomiting and fear are potent stimuli for excessive ADH secretion.

In each exercise-induced hyponatremia case, at least one of these symptoms occurs. The fear associated with hyponatremia is so severe that patients hyperventilate and have stated that they thought they were separating from their body or floating away from their body, and, therefore, they believed they were surely dying.

Inappropriate ADH levels increase the ECFV. This tends to suppress aldosterone secretion. Aldosterone suppression increases sodium excreted by the kidneys (but interestingly conserves potassium, which is why we see normal serum potassium). In this case, the ECFV rather than the plasma sodium influences aldosterone secretion. This explains renal sodium loss during a hyponatremic state. This syndrome of inappropriate ADH response (SIADH) may be more significant in the development of acute hyponatremia than sweat loss and water consumption. SIADH may be a phenomenon that exacerbates the patient's situation from mild

hyponatremia to coma.

Many have read articles that emphasize the need to hydrate with water when exercising and indicate that fancy sport drinks aren't necessary. The assumption is that the average person will eat at fairly regular intervals and, therefore, replenish their electrolytes. So they drink and drink and sweat and sweat. Sodium and water are lost through sweat and not replaced. ADH conserves free water, causing serum osmolarity to decrease. This triggers aldosterone release, which spits out high concentrations of sodium in urine. Sodium levels continue to drop, serum osmolarity continues to drop. Fluids shift from outside to inside the cell, causing swelling. Brain cells begin to swell, causing increased ICP.

Not only may people exercising in warm conditions experience dilutional hyponatremia during exercise, but the symptoms sometimes are delayed several hours, often occurring after the event is over. Third-spacing

PHOTO RICK McCLURE, LAFD

On May 2, 2004, the Los Angeles Fire Department (LAFD) responded to a heat-related MCI at Lincoln Park, where thousands of people had gathered in record-breaking heat for a Cinco de Mayo weekend festival. Here, firefighter Tim Toledo sprays water into the crowd to help cool them. Responders included 23 LAFD rescue ambulances, 17 firefighter companies, five EMS battalion captains, four battalion command teams and other LAFD support resources under the direction of Assistant Chief Michael Fulmis. More than 100 people were treated for heat-related complaints.

PHOTO BETSY HEIGHTMAN

Once known as water intoxication, exertional hyponatremia occurs when the proportion of total body water to total sodium is excessive.

of fluids in the gut occurs because it is ischemic. This free water often sits for hours. Delayed dilutional hyponatremia occurs when dehydration shock has occurred, and the patient probably has had severe gut ischemia with minimal water absorption in the intestinal area. The patient now consumes a large quantity of water or exercise drink that is stored in the stomach and intestines. Upon rest and redistribution of body fluids, the gut is no longer ischemic and releases hypotonic fluid into the ECF, diluting sodium levels.

Approach to patient assessment

The physical exam of the hyponatremic patient will reveal a variety of atypical vital signs Temperature can be normal, low or high. Heart rate can be elevated if the patient is hypovolemic and trying to compensate by increasing the rate. If the patient is normovolemic but hyponatremic, their pulse rate and blood pressure will be within normal ranges. If the patient is in a true shock state, blood pressure can be low. The respiratory rate can be elevated if true ischemia (such as in gut ischemia described above) is present and prolonged or in cases of hyperventilation, described above.

Mental status changes are the key to assessment. Early symptoms include general malaise, fatigue, headache and nausea. The similarity of these symptoms to heat exhaustion can complicate the assessment. If you treat a heat-exhausted patient with hydration and rest, he will get better. If you treat a hyponatremic patient with fluids and rest, he will get worse. *Example:* A recent fatality on an army base occurred when a recruit in boot camp died of cerebral and pulmonary edema after an army medic mistook his hyponatremia for severe heat exhaustion and forced fluids on him.

Neurological changes include ataxia, slowed speech and impaired cognitive thinking. Inappropriate behaviors, such as combativeness, apathy and withdrawal, may appear. Other symptoms include fear, a sense of impending doom and what is often described as an out-of-body experience.

Hyperventilation is common in exertional-induced hyponatremia. Hyperventilation accounts for dizziness, vision disturbances, tingling of the hands, tetany (the shakes), carpopedal spasms and **paresthesia** in the distal extremities and perioral areas. Hyperventilation raises pH levels, which increases the irritability of brain cells. This, coupled with brain swelling and rapid sodium loss, makes seizures a common event in hyponatremia patients. Seizures can occur at any time and appear to be influenced by the rate of sodium loss rather than the measured level. Once the patient seizes, he or she may remain obtunded for periods of 24–72 hours. There's a tendency for patients to projectile vomit, especially during helicopter flights and ambulance rides.

Because the clinical assessment can be difficult and mimics a variety of other medical emergencies, blood chemical analyzers can be used to clarify a patient's sodium status. I-STAT handheld mobile units can be adapted to environmental conditions, with special care given to maintaining the machine within its operating temperature. Use of ice packs and soft-sided lunch coolers also works well. Many basic EMTs can use capillary tubes to draw a fingerstick blood sample, and ALS providers can use venipuncture to access a blood sample.

Prehospital management

Place patients in a sitting up position. Lying the patient down could increase intracranial pressure and accelerate cerebral edema. If the patient is P or lower on the **AVPU scale**, place him or her in a left lateral position. Vomiting should be an anticipated complication.

Increase sodium levels slowly and usually only with IV NS. The use of 3% saline is controversial and is typically reserved for patients being monitored in the ICU with Na levels < 120. Too rapid replenishment of Na levels can cause central pontine **myelinolysis**. NS should be administered at a rate no faster than 250–500 cc/hour. These patients do well with fluid replacement. Just don't overload them. Even 2,000 cc will improve sodium levels, if given slowly.

Keep the patient calm. Fear, along with a gloom-and-doom attitude, can stem from the detached, almost floating sensation many such patients experience. Anxiety-induced hyperventilation should be discouraged.

Watch for seizures. If the patient seizes, airway management is a top priority. Once the patient seizes, his level of consciousness may remain altered.

Summary

As with most medical conditions, especially in a field or remote environment, prevention is the key. Many people may begin their exercise in hot weather in a dehydrated state due to such lifestyle influences as low-sodium food, caffeine consumption

and alcohol intake. Advise people to prepare for exercise in hot conditions at least 72 hours in advance by ensuring they are hydrated and nourished. Once hiking, individuals should be encouraged to keep up a steady intake of water or electrolyte replacement, drink and *eat*. We cannot emphasize this enough.

Sports physiologists often assume people are eating and, therefore, don't need commercial electrolyte replacement. The truth of the matter is that people often don't eat when they're hot, and they don't eat once they begin to become dehydrated and sick.

Gatorade, which contains the highest sodium concentration, doesn't even come close to the 35 mEq/liter/hour needed to replace lost salt through sweat. Salted snack food should be consumed to replace sodium lost through sweating. JEMS

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This continuing education activity is approved by the Center for Emergency Medicine, an organization accredited by the Continuing Education Board for Emergency Medical Services (CECBEMS), for 1.5 hours credit for First Responder, Basic and Advanced providers.

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