

Exertional Hyponatremia Too Much of a Good Thing

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Since 1989, Grand Canyon has been experiencing an ever-increasing rate of hyponatremia. Hyponatremia means low sodium concentration in the blood. Sometimes called water intoxication due to the neurological symptoms, the mechanism is far more complex than simply drinking too much water. This is not just a Grand Canyon phenomenon. Ultra endurance events including marathons, triathlons and even the popular “eco-challenges” are seeing an increase in incidents. Last year, there were 30 documented cases of exertional hyponatremia, seven of which were admitted into the ICU with intracranial pressure (ICP). Although most of these occur with hikers, passengers on commercial/ private river trips have also developed hyponatremia. Let's look at how our bodies respond to a heat challenge situation. Dehydration is a form of hypovolemic shock. Dehydration-volume shock is a progressive disorder that, if unhalted, spirals downward into deeper levels of hemodynamic and metabolic deterioration. Exertion further exacerbates the perfusion pressure in the system. Exercise in a hot environment forces the body to shift large amounts of fluid way from core circulation (brain, heart, and lungs) to the skin and skeletal muscle in order to dissipate heat. This is referred to a core-shell shift. Fluid is also lost through secretion of sweat and from breathing. Sweat can contain as much as 90 mEq/liter of sodium. Compare that with Gatorade which contains only 10 mEq/ liter. The tank gets lower and lower, perfusion pressure drops, the hiker's performance drops, fatigue sets in, and you have the beginnings of heat exhaustion. The body in its amazing ability to maintain equilibrium will begin shunting blood from the Gastrointestinal (GI) tract (gut) and other less needed areas. This results in little absorption of food and water from the stomach and intestines—the power bar and Gatorade just sit there. Any guess what happens next...you begin to feel nauseous and sick to your stomach. As volume, shock continues the body will shift from a core-shell movement to a shell-core. The long-term risk of this is soaring core temperatures. Because the maximum rate of gastric emptying is much less than the maximum sweat rate (1.2 verses 3.7 l/hr). Rehydration cannot keep pace with sweat losses under those conditions. This puts you behind the power curve. Rehydration and replacement of electrolytes lost in sweat via oral intake may require 36 hours with occasional urination to complete.

Exertional Hyponatremia

Water intoxication is an old term for exercise induced dilutional hyponatremia or exertional hyponatremia for short. Hyponatremia occurs when the proportion of Total Body Water (TBW) to total sodium is excessive and a hypo-osmolar hyponatremia develops. You can get this and still be dehydrated. How did we get there is the question. Actually, there are several mechanisms coming into play here. The first

problem is drinking too much freewater and not replacing lost electrolytes. The second is the spacing of hypotonic fluids in the gut during exertion. The third problem is a syndrome of inappropriate antidiuretic hormone (adh) response, known as siadh. All three mechanisms can be working together.

The Role of AIH

One compensating mechanism of the body is the release of adh (antidiuretic hormone). It is released by the pituitary gland in response either blood volume or changes in sodium concentration, known as osmolarity. Osmolarity is the concentration of ions in any given solvent. A fall in blood volume causes a release of adh—to try to get the kidney to conserve more water and bring the volume back up. A low concentration of sodium causes the pituitary to decrease the amount of adh and you pee out more urine. Thus, low sodium diets help hypertension by decreasing volume.

So what happens when we begin to lose both sodium and volume? The former tells the pituitary gland to decrease adh and the latter tells it to increase. The result is conflicting messages, but protecting blood volume is a survival mechanism and in the case of a hiker who is dehydrated and hyponatremic, the pituitary will continue to release adh to hold onto water, worsening the hyponatremia. Hopefully the brain thinks, this joker will stop hiking and find a salt lick soon.

Why then when people are normal volume and hydrated, does adh continue to conserve water creating a hyper-hydration and dilution of plasma sodium ions? I do not know, but I have some theories. Many hikers at Grand Canyon probably begin their hike in a dehydrated state due to lifestyle influences of caffeine consumption, alcohol intake, etc. Nausea, vomiting, and fear are also potent stimuli for excessive adh secretion. In each exercise induced hyponatremia case I have seen, there has been at least one of these symptoms. The fear associated with hyponatremia is so severe the patients hyperventilate and have stated that they thought they were separating from their body or floating away from their body and, therefore, they were surely dying. Siadh may be more significant in the development of acute hyponatremia than sweat loss and water consumption. Siadh may be a phenomenon that worsens the patient's situation from mild hyponatremia to coma.

So, let me give you a summary scenario. Hikers come into their hike dehydrated chronically. Adh is circulating. They read in Backpacker Magazine that you need to hydrate yourself while hiking and that these fancy sport drinks are not needed. So they drink and drink and sweat and sweat. Sodium and water is lost through sweat and not replaced. Adh conserves free water, causing blood sodium osmolarity to decrease. Sodium levels continue to drop, serum osmolarity continues to drop. Hyponatremia sets in. Bad things begin to happen. Fluids shift from outside the cell inside, causing swelling. Brain cells begin to swell, causing increased intracranial pressure and bad neurological changes including headache, confusion, staggered gait, seizures, and unconsciousness.

Let me muddy the waters. Not only will hikers or triathlon participants experience extensional hyponatremia during their hike, but the symptoms sometimes are delayed several hours, usually after the hike or event is over. Remember we talked about the third spacing of fluids in the gut 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. Add that to what was stored in the gut and you have a high level of hypotonic fluids. You rest, your gut is no longer ischemic and releases hypotonic fluid into the bloodstream, diluting sodium levels, and bingo you start seizing in camp, long after the hike is over.

Approach to Patient Assessment

Physical exam will reveal normal vital signs. Temperature is normal or low. Mental status changes are the key to assessment. Early symptoms include general malaise, fatigue, headache and nausea. As you can see these are very similar to heat exhaustion. That is where the assessment criteria gets tricky. If you treat a heat exhausted patient with hydration and rest, they will get better. If you treat a hyponatremic patient with fluids and rest, they will get worse. Although we have never had a death at Grand Canyon, there was a recent fatality on an army base, where a recruit in boot camp died of cerebral and pulmonary edema after army medic mistook his hyponatremia for severe heat exhaustion and forced fluids on him.

Because hyponatremia looks so much like heat exhaustion in its early stages and can mimic heat stroke once the person seizes, the park rangers have come to rely on portable blood chemical analysis in the field. These machines will measure the level of sodium in the blood stream and give us a better idea of what is going on. They are expensive but worth it.

Management of Exertional Induced Hyponatremia

- Place patients in a sitting up position. Lying down could increase intracranial pressure and accelerate cerebral edema. If the patient is p or lower on the avpu (Alert, Verbal, Pain, Unconscious) scale, place them in a left lateral position. Watch for vomiting. It should be an anticipated problem. Patients are prone to aspiration.
- Increase sodium levels slowly.
- Keep the patient calm. Fear along with a gloom and doom attitude stems from a detached, almost floating sensation these patients experience allowing an onset of anxiety induced hyperventilation syndrome to ensue. Anxiety induced hyperventilation should be discouraged.
- Watch for seizures. If patient seizes, airway management is a top priority. Once the patient seizes, they will remain aloof.

So what is the take home message...Prevention is the key.

Stay hydrated and nourished. Once hiking, keep a steady intake of water or electrolyte replacement drink and eat. I cannot emphasize this enough. Sport physiologists assume people are eating and therefore do not need commercial electrolyte replacement. The truth of the matter is that people don't eat when they are hot, and they don't eat once they 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. What kind of food, my personal preference is salty snack food. This is not a time for power bars. Leave the health food behind. Junk food is great. Stock up. The rangers now routinely give out saltine crackers, pretzels and cheezits. Stay ahead of the sodium curve!