Preventing Deaths Due to Exercise-Associated Hyponatremia: The 2015 Consensus Guidelines

Mitchell H. Rosner, MD

In the summer of 2014, 2 otherwise healthy 17-year-old high-school football players died from hyponatremic encephalopathy.[1,2] In both cases, the common denominator was excessive fluid consumption; a peril that has been well described in athletes and had been the subject of 2 previous consensus documents on exercise-associated hyponatremia (EAH).[3,4] Unfortunately, despite strong evidence of the dangers of overly aggressive fluid intake beyond the body's maximal fluid excretion rate, many athletes, coaches, trainers, and lay public continue to embrace the idea that "hyperhydration" has some health and performance benefits while the risks of EAH are overstated. It is the time that these dangerous practices cease. In response to these deaths and to continued cases of symptomatic EAH, a third consensus conference that focused on new developments regarding this condition and a focus on educating key stakeholders about the risks of EAH was held and published in this edition. A stated goal of this consensus conference was to educate the public so that no further EAH deaths occur.

Although it is true that the risks of symptomatic EAH are low, the consequences can be catastrophic. Most studies show that the incidence of symptomatic EAH in endurance sports, for which there are the best data, is less than 1%.[5,6] Estimating the number of deaths due to EAH is difficult due to lack of accurate reporting, but it is likely in excess of 14 or more.[7] These are deaths in young healthy people in the "prime" of their life, and most importantly, they are 100% preventable. How can anyone overstate the significance of a death in a healthy athlete who had the promise of their future cut short, especially when it was avoidable?

Prevention starts with understanding the risks associated with the condition, and in the case of EAH, abundant evidence accrued over the past decade is consistent, in which excessive fluid consumption is the most common and strongest driving factor.[8] For example, a college football player developed hyponatremia after receiving 5 liters of intravenous fluids while ingesting 3 additional liters of hypotonic fluid (a sports beverage) within 5 hours[9] One of the high-school football players who died this year reportedly drank 16 L of fluid during practice to alleviate muscle cramps.[1] There is seldom a good reason to support these massive fluid intakes. Of course, there are other factors that are important in the development of EAH, such as arginine vasopressin–induced concentration of the urine, but these are more permissive factors and not as critical as the major influence of excessive fluid consumption.[8]

Given that there may be wide variation of sweat production and in the capacity of the kidneys to excrete water (dependent on both the glomerular filtration rate and level of urine concentrating ability) both between individual athletes and in the same individual depending on the ambient conditions during the activity, universal guidelines for prevention seem to be difficult. Furthermore, there has been great concern that limiting fluid intake during strenuous activities risks both dehydration and heat-related illnesses. However, sensible and safe drinking guidelines that balance all of these issues and prevent EAH are feasible and rest on both sound physiological principles and some limited evidence.

What are these prevention techniques? The first is perhaps the most physiological and simple: drink according to thirst. Our thirst sensation is a finely tuned regulatory mechanism that protects plasma osmolality from rising more than a few percentage points

From the Department of Medicine, University of Virginia Health System, Charlottesville, Virginia.
The author reports no conflicts of interest.
Corresponding Author: Mitchell H. Rosner, MD, Department of Medicine, University of Virginia Health System, Box 800133, Charlottesville, VA 22908 (mhr9r@virginia.edu).

above normal.[10] Thus, our thirst sensation will prompt drinking and help to guard against excessive dehydration. Now, there are some, relatively uncommon, circumstances where the thirst mechanism may not be sufficient or adequate to protect against dehydration; for instance, in those extreme environments or with especially aggressive activity where sweat losses may be very high and thereby lead to rapid development of dehydration. Although somewhat controversial, the bulk of evidence supports that mild levels of dehydration (up to 2%-3% of body weight) are well tolerated and minimally affect athletic performance.[11] Thus, drinking according to thirst should be protective of significant levels of dehydration while at the same time preventing overly excessive fluid intake and the development of EAH. In fact, recent evidence suggests that the brain has adapted mechanisms to avoid excessive oral fluid intake, and its attendant dangers, with activation of areas in the midcingulate cortex, insula, amygdala, and periaqueductal gray that make overdrinking feel unpleasant.[12] These regions may contribute to the termination of drinking. Overriding these mechanisms with continued volitional drinking behavior is unnecessary and potentially dangerous.

For those doubting the protective effect of our thirst sensation or concerned about the risk for dehydration, another simple protective strategy is to estimate hourly sweat losses during exercise and avoid consuming amounts that are greater than this amount during endurance or other athletic events.[13] This is facilitated by serial measurements of weights during and after exercise with the goal to maintain weight or even finish exercise with a slighter lower weight. A concern is that this technique may be more time consuming and less likely to be followed by casual athletes. This strategy may be particularly attractive to certain sporting events such as football where sideline scales can easily be available to guide fluid intake.

In all cases, blanket statements that can be found on the internet such as "don't wait until you feel thirsty" make little sense for the majority of casual athletes and have the potential for disastrous consequences as they promulgate the idea that near constant fluid ingestion during athletic events is a reasonable and even necessary thing to do.[14] These statements have, indirectly, led to dangerous behaviors and resulting EAH.

The 2015 EAH Consensus Guidelines focuses on safe drinking guidelines balancing the risks for dehydration and, most importantly, the well-described deleterious effect of aggressive hydration. The consensus group feels that EAH is completely avoidable and with training and education regarding safe drinking/hydration that this goal is achievable. Indeed, the next step for the consensus group is dissemination of this information to all stakeholders and athletes. Our goal is that no other athletes die from EAH.

REFERENCES

1. Web site. http://www.washingtonpost.com/blogs/early-lead/wp/2014/08/13/a-high-school-football-player-dies-after-reportedly-consuming-two-gallons-each-of-water-and-gatorade/. Accessed May 26, 2015.
2. Web site. http://www.clarionledger.com/story/news/local/2014/08/25/walker-wilbanks-died/14584793/. Accessed May 26, 2015.
3. Hew-Butler TD, Ayus JC, Kipps C, et al. Statement of the Second international exercise-associated hyponatremia consensus development conference, New Zealand, 2007. *Clin J Sport Med.* 2008;18:111–121.
4. Hew-Butler T, Almond C, Ayus JC, et al; Exercise-Associated Hyponatremia (EAH) Consensus Panel. Consensus statement of the 1st international exercise-associated hyponatremia consensus development conference, Cape Town, South Africa 2005. *Clin J Sport Med.* 2005;15:208–213.
5. Hoffman MD, Hew-Butler T, Stuempfle KJ. Exercise-associated hyponatremia and hydration status in 161-km ultramarathoners. *Med Sci Sports Exerc.* 2013;45:784–791.
6. Noakes TD, Sharwood K, Speedy D, et al. Three independent biological mechanisms cause exercise-associated hyponatremia: evidence from 2,135 weighed competitive athletic performances. *Proc Natl Acad Sci U S A.* 2005;102:18550–18555.
7. Hew-Butler T, Rosner MH, Fowkes-Godek S, et al. Statement of the 3rd international exercise-associated hyponatremia consensus development conference, Carlsbad, California, 2015. *Clin J Sports Med.* 2015;25:303–320.
8. Rosner MH. Exercise associated hyponatremia. *Semin Nephrol.* 2009;29:271–281.
9. Herfel R, Stone CK, Koury SI, et al. Iatrogenic acute hyponatraemia in a college athlete. *Br J Sports Med.* 1998;32:257–258.
10. McKinley MJ, Johnson AK. The physiological regulation of thirst and fluid intake. *News Physiol Sci.* 2004;19:1–6.
11. Cheuvront SN, Keneflick RW. Dehydration: physiology, assessment and performance effects. *Compr Physiol.* 2014;4:257–285.
12. Saker P, Farrell MJ, Abid FRM, et al. Regional brain responses associated with drinking water during thirst and after its satiation. *Proc Natl Acad Sci U S A.* 2014;111:5379–5384.
13. Hoffman MD, Stuempfle KJ. Hydration strategies, weight change and performance in a 161 km ultramarathon. *Res Sports Med.* 2014;22:213–225.
14. Web site. http://www.healthalkaline.com/drinking-water-before-you-get-thirsty/. Accessed May 26, 2015.