water retention from the overconsumption of fluids or sustained consumption beyond the capacity for urinary free water excretion. EAH is not uncommon with considerable weight loss in some environments,¹⁰ but symptomatic cases are generally associated with weight gain and weight loss of less than 3% of body weight.⁹ As noted in the WMS Practice Guidelines for EAH, athletes should be encouraged to drink to thirst, and strive for a weight loss of 2% to 4% during prolonged exercise.³ Recommendations to avoid more than 2% weight loss serve to promote the overconsumption of fluids.

The issues we raise are not trivial, as the failure to prevent and appropriately manage EAH can mean the difference between life and death. We hope that by highlighting the potential dangers of overhydration and use of intravenous isotonic or hypotonic saline in those with EAH, your readers will be better prepared to appropriately advise endurance athletes about hydration and make better medical decisions when faced with a potential case of EAH.

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The Importance of Keeping Cool: Reply Regarding the Wilderness Medical Society Practice Guidelines for the Prevention and Treatment of Heat-Related Illness

To the Editor:

As there is significant morbidity and mortality from heat stroke in wilderness activities, mass gatherings, and regional heat waves, our recently published Wilderness Medical Society's Practice Guidelines for the Prevention and Treatment of Heat-Related Illness¹ were intended to elucidate and provide guidance in the appropriate recognition and management of the spectrum of disease of heat injury. We emphasized the need for rapid recognition and implementation of cooling treatments, highlighted the safety and evidence-based recommendation for treatment by cold-water immersion, and stressed the importance of continuation of cooling treatments until the victim's core temperature could be stabilized, as injury is directly related to both magnitude and duration of hyperthermia. The concern of Hoffman et al that rapid cooling and volume replacement in an endurance athlete suffering from heat stroke should be withheld owing to the possible coexistence of exercise-associated hyponatremia (EAH) is a somewhat myopic perspective that could inadvertently lead to dangerous mismanagement of heat-related illnesses.

The majority of athletes with exercise-induced heat illness are dehydrated with minimal dysnatremia.² When hyperpyrexia occurs or an altered sensorium becomes evident, the severe disease states manifest and become distinctive. The apparent confusion by Hoffman et al is likely based on their erroneous presumption that heat stroke and severe EAH (EAH with encephalopathy) often overlap, because there is a lack of any evidence supporting the coexistence of these 2 severe diseases. Heat stroke is marked by multiorgan dysfunction,

systemic shock, and high mortality when presenting with hypotension, necessitating prompt aggressive cooling and consideration of systemic support through intravenous hydration. The presence or absence of hyperpyrexia, along with the clinical context of the victim, should assist the medical provider with guidance in the resuscitation of the altered patient. Our treatment recommendations are not predicated on any diagnostic tool more technologically advanced than a thermometer. As such, we disagree with Hoffman et al that “the appropriate diagnosis in the field is rarely possible.”

There is complete agreement as outlined in our Practice Guidelines that severe EAH should be treated with 3% hypertonic saline boluses; however, we disagree with the statement that *all* symptomatic EAH need be. The crux of the disagreement is that in mild EAH (symptoms without evidence of encephalopathy) there has been no substantial evidence that isotonic fluids may worsen the clinical hyponatremic state, lead to “disastrous clinical outcomes,” or even reduce quantitative serum sodium levels more than hypertonic fluids.³ To repeat this clinically valuable point that may be misunderstood: in the setting of a patient with mild EAH (without altered mental status or other symptoms or signs of cerebral edema) and unable to tolerate oral fluids, isotonic intravenous fluids for either EAH or heat-related illness have not been demonstrated to be harmful. For a useful treatment algorithm that incorporates the available evidence when point-of-care sodium testing is unavailable, please refer to the recently published Clinical Practice Guidelines for EAH.⁴

Symptoms of heat exhaustion and EAH may present similarly.⁵ Heat exhaustion is a constellation of symptoms that limits one’s ability to continue to exercise in the heat. It is usually self-limited and presumed to be caused by loss of fluid and electrolytes.⁶ Hypohydration has been shown to decrease sweat rates and increase core temperature⁷ and predisposes to worsen the severity of heat illness; the National Athletic Trainers’ Association lists hydration as an important factor in reducing hyperthermia.⁸ The priority in treatment is to minimize progression of heat illness, and ignoring the dehydrated state in this context may do just that. We appreciate the concern of Hoffman et al for EAH in the endurance athlete as well as minimal risks posed by dehydration, but applying this paradigm to the context of a symptomatic heat-stressed individual who cannot tolerate oral fluids is potentially injurious. To the contrary of their disapprobation, we share their concern regarding severe EAH, and as such our Practice Guidelines repeatedly recommended caution if intravenous isotonic fluids are given when the possibility for EAH and altered mental status are present.

We thank Hoffman et al for their informative comments on weight loss during exercise and muscle cramps in elite athletes, but please keep in mind that our Practice Guidelines are intended to be generalizable to both athletes and nonathletes. Targeting one specific group was outside the scope of this panel. Our recommendation to hydrate to avoid more than 2% weight loss was chosen based on the available evidence that this threshold at activity or at rest renders the participant more susceptible to heat accumulation, thus hyperthermia. We deemed it unwise to strive for fluid losses that could elevate risk for excess heat accumulation. This is supported by a recently published cycling trial that found that a more than 2% fluid loss was associated with elevated core temperatures.⁹ Furthermore, we do not promote hypotonic fluid ingestion because of the well-founded concern for dilutional hyponatremia, and as such, there is not a single mention of hypotonic fluids anywhere in our entire article.

Heat cramps was a term initially coined in the 1930s to describe the clinical phenomenon of involuntary diffuse muscle contractions associated with exertion in hot environments, which may be distinct from the focal muscle cramping of an endurance athlete during repetitive exercises. Observational studies have associated dehydration and electrolyte disturbance with the former scenario.¹⁰ Although association does not prove causation, the connection between heat-related illness, muscle cramps, dehydration, and electrolyte disturbance is consistent enough that the US National Institutes of Health and the Centers for Disease Control and Prevention continue to mention dehydration and electrolyte disturbance as factors that potentially contribute to “heat cramps” in their online educational materials. The Practice Guidelines were not intended to address the underlying pathophysiology of the contractions, and although alternative classifications have been proposed, the existing data would not place exercise-associated muscle cramps (EAMC) firmly into any one category. Although the terminology remains imperfect, “heat cramps” is intended to represent EAMC in a hot environment, as outlined in Table 2 of the Practice Guidelines. We do not believe that this treatment or others provided promote overhydration; in fact, the exact opposite is stated, “care should be taken to not overhydrate patients” with intravenous isotonic or hypertonic fluids.¹ Indeed, we agree with Hoffman et al and support and encourage a “drink to thirst” approach.

Heat-related illness is a very common and unfortunately often mistreated disease. Our intention of the Practice Guidelines was to avoid the propagation of myths in treatment and to provide evidence-based recommendations to optimize care in the wilderness,

prehospital, and urban settings. We thank the authors of the letter for their continued efforts to elevate the science and awareness of EAH, and hope that in the setting of symptomatic heat-stressed individuals, they as well as other clinicians will apply a keen differential diagnosis to the victim, including a comprehensive history and physical, and avoid delay in cooling if concern for heat stroke is present.

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Heat-Related Illness: Time To Update Our Lexicon

To the Editor:

We read with great interest the newest practice guidelines offered by the Wilderness Medical Society (WMS) on prevention and treatment of heat-related illness. However, we believe that several points should be refined to reflect the more recent body of literature, modern nomenclature, and pathophysiologic understanding of the disease entities described.

Lipman et al¹ begin their definition of heat-related illness with a discussion of the spectrum of diseases related to heat. We submit that there is no objective evidence in the existing body of literature to suggest a necessary link between the development of so-called heat cramps, heat syncope, heat exhaustion, and heat stress. Further, there is no objective evidence to support the dogma that heat cramps or heat exhaustion will progress to heat stroke (exertional hyperthermia), just as there is no evidence to suggest that hypertension continues to worsen until hypertensive emergency occurs.

Rather than providing supportive objective evidence, the existing body of literature in fact refutes the concept of a spectrum of diseases related to heat. In 2006, for example, Byrne et al² performed continuous core temperature monitoring in 18 volunteers at a half-marathon in Singapore. All runners remained asymptomatic from any heat illness despite the fact that 56% of them had core temperatures greater than 40°C and 11%, greater than 41°C.

By the definition used in the newest guidelines, the body remains normothermic during heat exhaustion. We invite the reader to review work done by Noakes,³ who articulates why heat exhaustion is an inaccurate description of a phenomenon that is not objectively linked to heat accumulation or hyperthermia. Alternatively, Noakes suggests that we begin to update our nomenclature to be more accurate and commensurate with known pathophysiology. Instead of labeling the condition heat exhaustion, it is more appropriately understood as “exhaustion that may or may not happen in the heat.”

We also submit that heat cramps is a confusing and inaccurate title for a disease process likely unrelated to