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Maximum Water Temperature Limit in Open-Water Swimming Events

To the Editor:

In a recent review published in *Wilderness & Environmental Medicine*, we highlighted the concept of the potential risk of heat stroke for swimmers during long-distance events in warm or hot water.¹ After reviewing the scientific literature relevant to the topic, we indicated the factors that may accentuate the risk in open-water swimmers, namely high exercise intensity, specific body composition, heat gain by sunlight radiation, and low potential for convective cooling during swimming.^{1–4} A death during a FINA (Federation Internationale De Natation) competition (10-km Marathon Swimming World Cup race that took place in Fujairah, United Arab Emirates, on October 23, 2010) has been imputed to heat stroke because the water temperature exceeded 31°C. The FINA and ITU (International Triathlon Union) regulation books did not have rules that defined a maximum water temperature, although a minimum water temperature was set.

It is with great interest that we learned about the new regulation maximum water temperature limit of 31°C in the FINA rule book regarding open-water swimming events (effective from September 17, 2013 to 2017).⁵ The decision was announced on July 16, 2013, by the FINA president at the Technical Open Water Swimming Congress in Barcelona, based on a study conducted at the University of Otago in New Zealand in collaboration with the FINA and ITU. Although the experimental data obtained by this research group are yet to be published, the established maximum water temperature set by the FINA is not too different from the temperature extrapolated from the literature by our research group (33°C).¹ Moreover, the cooler water temperature limit established by the FINA is likely safer for swimmers.

In conclusion, assessment of the risk for heat stroke by examining physiological responses of swimmers during long-distance events assists sports organizations to update rules to safeguard athletes. We maintain that the journal and the editors have accomplished an important task of the sciences “to disseminate knowledge and scientific findings to the broader community.”

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Regarding the Wilderness Medical Society Practice Guidelines for Heat-Related Illness

To the Editor:

Exercise-associated hyponatremia (EAH) is recognized as a potential and serious complication from improper hydration strategies during endurance exercise. Several deaths attributable to EAH have been reported,¹ with morbidity and mortality likely exacerbated by inappropriate medical management with isotonic or hypotonic fluids under the clinical presumption that the athlete was dehydrated.^{1,2} It is now clear that isotonic and hypotonic fluids will worsen cerebral encephalopathy in cases of dilutional EAH and, at a minimum, result in a prolonged recovery.² Proper management of symptomatic EAH from either a dilutional or depletional mechanism is with hypertonic saline as discussed in the recently published Wilderness Medical Society (WMS) Practice Guidelines for EAH.³ Furthermore, the risks and benefits of intravenous isotonic fluid administration in symptomatic EAH have been outlined in a response to a letter to the editor.⁴ The physiological basis for avoiding isotonic fluids in EAH becomes evident when it is recognized that the usual pathophysiology of nonosmotic arginine vasopressin secretion in both dilutional and depletional EAH will facilitate urinary water retention and urinary sodium excretion,^{1,2} the latter possibly enhanced by circulating brain natriuretic hormone.⁵ Thus, in these cases of EAH as a variant of the syndrome of inappropriate secretion of antidiuretic hormone (SIADH), isotonic fluid administration will either worsen the condition or prolong recovery as originally described in 1967.¹

We are concerned that the recently published WMS Practice Guidelines for heat-related illness⁶ do not address the potential consequences from the provision of isotonic fluids in the presence of dilutional EAH from an SIADH-like mechanism. As discussed in the WMS Practice Guidelines for EAH and acknowledged in the Practice Guidelines for heat-related illness, the constellation of symptoms of EAH overlap with those of dehydration and heat illness. As such, appropriate diagnosis in the field by clinical symptoms alone is rarely possible, and could result in inappropriate treatment leading to potentially disastrous clinical outcomes.^{1,2} Therefore, we advocate caution when using intravenous isotonic or hypotonic fluids in the field when blood sodium concentration is unknown because the

benefits may be negligible⁷ and the risks of either worsening or delaying recovery from EAH will be significantly increased.^{1,2} Furthermore, when treating endurance athletes in the field with intravenous isotonic or hypotonic fluids for presumed dehydration, it is prudent to have immediate access to hypertonic saline should the patient deteriorate.

We must also express concern with the WMS Practice Guidelines for heat-related illness relative to the recommendations that promote overhydration. Without providing references, the document indicates that "heat" cramps are related to hydration or electrolyte disturbances and that treatment includes oral isotonic or hypertonic salt or electrolyte replacement. First, to label exercise-associated muscle cramps (EAMC) as heat cramps is incorrect. EAMC can occur in any environmental conditions, not only during exercise in hot, humid environmental conditions. Second, there is very strong evidence from a number of prospective cohort studies and a recent laboratory study that convincingly show that neither fluid and electrolyte disturbances nor dehydration are associated with EAMC.⁸ Rather, this work indicates that fatiguing exercise, past history of EAMC, underlying muscle or tendon injury, and possibly genetic predisposition are more predictive of the risk of EAMC.⁸ Furthermore, these data can best be explained by a common hypothesis whereby a final common pathophysiological pathway of altered neuromuscular control is responsible for the development of EAMC in athletes.⁸ As such, the recommendation to treat muscle cramps with oral hydration, salt, or electrolyte replacement is not only unfounded, but may encourage overhydration with the possibility of inducing dilutional EAH.

We also take issue with the general recommendation to avoid loss of over 2% of body weight during exercise. To understand why, consider the basic physiological principle that water is bound to glycogen, and that this water is released as glycogen is oxidized. It has been estimated that approximately 1.5 kg of water is bound to glycogen, and when considering the weight associated with substrate utilization, a weight loss of over 2 kg (approximately 3%) is required to maintain euhydration during prolonged exercise in a 70-kg athlete.⁹ It is also evident that endurance athletes are well tolerant of weight losses of at least 8%.^{9,10} Furthermore, body weight losses greater than 3% to 4% have been shown to be common among athletes with the best performances in various endurance events.¹⁰ Therefore, a general recommendation to avoid more than 2% weight loss during prolonged exercise in a competitive field setting is unsupported and could precipitate the development of dilutional EAH.

We raise legitimate concerns about overhydration because symptomatic EAH is generally associated with