After intubation, 150 mL of NS was infused, and the patients eventually died. Another 2 cases of severe EAH (encephalopathic) received HTS and made a full recovery. The figures depicting time vs serum sodium concentrations show both of the latter 2 patients had resolution of central nervous system symptoms several hours before HTS infusion.⁴ Conclusion: Severe EAH requiring intubation has a dismal prognosis. No subsequent sodium levels were provided in the first 2 cases, so it is unknown what effect 150 mL of NS had on the hyponatremic state or clinical condition, but caution may be warranted to avoid NS in severe EAH.

The most robust of the references was a retrospective chart review combined with a small prospective case series, comparing 2 years of different EAH treatments. The retrospective analysis was of 16 marathon runners who received NS in the emergency department for EAH. Of the 5 admissions, 3 had severe EAH on arrival and were intubated before receiving NS, and 2 eventually received HTS in the intensive care unit, but there was no description of the fluids the third patient received. All 3 were discharged with full recovery several days later. The other 2 runners with mild EAH who received NS were admitted, but not to critical care. A year later, a prospective analysis of 5 runners, 4 with mild and 1 with severe EAH (encephalopathic) received HTS as the initial emergency department infusion, and all were discharged. The article contains a [Figure](#) showing correction rates from a sample of the 2 groups: the NS group had 2 of 8 (25%) patients' serum sodium concentration initially decrease before increasing; the HTS group had 1 of 4 (25%) with the same trend.⁵ Conclusion: In critical EAH, NS had the same outcome as HTS therapy; in mild EAH, NS may worsen outcome requiring hospital admission but not to critical care levels. EAH patients may experience a transient decrease in serum sodium with both isotonic and hypertonic IV therapy.

The above cases describe the clinical spectrum of EAH. It is a potentially fatal disease that requires medical providers at endurance and ultraendurance events to be comfortable with airway management and critical care. Although caution is warranted in treating any patient with symptomatic EAH, IV rehydration with isotonic fluids has not been causally related to worsening outcomes necessitating intensive level care. However, delay in treating an athlete's hypovolemia with anything other than a few milliliters of hypertonic saline when transportation to a care facility is many hours away may have grave consequences. If there is any question or concern of neurologic impairment, HTS is the most efficacious agent and NS should be avoided. With a qualified definition of EAH, an IV fluid treatment approach is possible that is evidence-based and clinically oriented to assist the medi-

cal provider when there is no available POC serum sodium testing. The [Figure](#) shows a treatment algorithm emphasizing symptom recognition rather than serum sodium testing to provide guidelines for clinical assistance when technological support is unavailable.

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In Reply to Clinical Practice Guidelines for Treatment of Exercise-Associated Hyponatremia

The Letter to the Editor of Dr Lipman demonstrates an unfortunate persistent confusion about potential dangers from exercise-related dehydration and the appropriate management of exercise-associated hyponatremia (EAH). We welcome the opportunity to respond to this letter with hopes that readers will be enlightened to nearly 40 years of evidence and that we can provide further support for our position in the WMS Practice Guideline on Treatment of Exercise-Associated Hyponatremia.¹

Dr Lipman seems to express 2 primary concerns with our EAH Practice Guideline. One of his issues is that he believes there is the potential for “grave consequences” from delay in treating an athlete's hypovolemia out of concern that the athlete might have EAH. In his proposed algorithm, he recommends provision of 1 to 2 L of intravenous normal saline (NS) or NS with 5% dextrose in athletes who have symptoms suggestive of EAH and also display signs and symptoms of severe dehydration. He emphasizes symptom recognition in the diagnosis of

dehydration. However, the clinical assessment of dehydration in athletes has been shown to be unreliable.² More importantly, as far as we know, there is no documentation in the scientific literature linking exercise-related dehydration to life-threatening conditions.³ Endurance athletes have been shown to have body mass losses of 8% or greater without significant clinical symptomatology or adverse consequences.^{4,5} In most cases, such levels of dehydration are not hazardous and rarely require intravenous rehydration if oral fluids can be tolerated. Furthermore, a recent evidence-based review revealed that it is transient postural hypotension that is the most common etiology for why endurance athletes receive postevent medical attention.⁶ This is caused by lower extremity pooling of blood once the athlete stops moving and the resultant impairment of cardiac baroreceptor reflexes. These athletes present with lightheadedness, dizziness, or syncope, which has historically been managed as dehydration or hyperthermia by race event medical providers.⁶ The perception that heat exhaustion is caused by dehydration and that the levels of dehydration observed in endurance sports must be treated immediately with intravenous fluids has been termed the “dehydration myth” and was noted to have been “relentlessly perpetuated” well over a decade ago.³ Unfortunately, it appears that the confusion continues to be perpetuated.

The second primary concern expressed in the letter of Dr Lipman appears to be that he believes that when point-of-care testing of serum sodium is not available, the literature does not support our recommendations and grading relative to the use of intravenous hypertonic saline (HTS) over NS in the treatment of presumed EAH. In this regard, he also indicates we do not provide ample evidence to support the concern we raise about treating EAH with intravenous NS. We believe that in Dr Lipman’s review of the 4 references we cite to support our position, he has unfortunately arrived at faulty conclusions. We will critically reexamine and clarify the findings of those papers that support our position.

The earliest report that we reference in support of treating EAH with HTS and the potential risk of treatment with NS was that of Frizzell et al.⁷ This report is of 2 overhydrated ultramarathon runners with symptomatic EAH. One received intravenous NS, and was hospitalized for 5 days with 36 hours in a semicomatose state. The other runner received intravenous 3% HTS and was fully alert in 3 hours and discharged after 8 hours. Although not definitive proof of the effectiveness of HTS, this is early suggestive evidence. We disagree with Dr Lipman’s conclusion that the report indicates “severe EAH resolves with both NS and HTS” as we do not find a 5-day hospitalization with

prolonged impairment of central nervous system function to be an acceptable outcome.

The second cited publication describes 7 marathon runners with symptomatic EAH.⁸ For the 1 patient who died, the diagnosis of EAH was unsuspected, and the patient therefore did not receive treatment for EAH. In contrast, all 6 patients who were correctly diagnosed and treated with 3.3% HTS survived. We accept that, at best, this report suggests that there might be potential value of HTS in the treatment of EAH. We also agree that the additional 2 cases of EAH noted to have survived after receiving intravenous NS do not support that premise. But we do not agree with Dr Lipman’s conclusion that “HTS is beneficial for critical EAH, and mild EAH can be treated with either HTS or NS.” Rather, we would reserve such conclusion recognizing that some patients will survive despite inappropriate management.

The third cited publication describes a retrospective and prospective analysis of EAH treatment.⁹ The retrospective analysis involved 11 overhydrated marathon runners with severe EAH (defined as blood sodium ≤ 125 mEq/L by the authors). They were all treated initially with intravenous NS. Five of these runners (blood sodium concentration range, 119–121 mEq/L) required hospitalization, with 3 requiring intubation. Two of these patients ultimately received HTS in the intensive care unit. Fortunately, all runners survived and were discharged several days later. The prospective treatment trial involved 4 cases of overhydrated marathon runners with severe EAH (blood sodium concentration range, 117–123 mEq/L) treated with 3% HTS. None required hospitalization, and the rate of blood sodium correction with HTS was shown to be more rapid than for those runners in the retrospective analysis who were treated with NS. Dr Lipman’s conclusion that “in critical EAH, NS had the same outcome as HTS therapy” is not supported given the extended hospitalization evident among those treated initially with NS.

The fourth cited publication describes 4 marathon runners who became unresponsive and were found to have EAH.¹⁰ The 2 patients treated with intravenous NS died with evidence of cerebral edema on postmortem examinations. In contrast, in the 2 runners receiving 3% HTS, the treatment “resulted in rapid neurologic improvement without adverse effects.” Furthermore, the graphs provided in the publication that show the time course for treatment with HTS demonstrate that the resolution of neurological symptoms occurred in less than 2 hours after this treatment was initiated. Regarding the 2 patients treated with NS, the article has an unfortunate typographical error (indicating the infusion was initially “at 150 mL,” denominator not provided), but it is understood that the infusion rate was 150 mL/h

(Art Siegel, MD, personal communication, June 4, 2013). Dr Lipman's conclusion that "severe EAH requiring intubation has a dismal prognosis" is unsupported given that runners with EAH who were treated with HTS had good outcomes and those who were treated with NS did not. Furthermore, his conclusion that "it is unknown what effect 150 mL of NS had on the hyponatremic state or clinical condition" seems to result from misinterpretation of the paper.

In addition to these 4 reports demonstrating the potential adverse consequences of intravenous NS hydration and benefits from HTS in athletes with EAH, another early case report provides further support for our concern about the potential dangers of intravenous NS hydration in EAH.¹¹ This case was of a runner who was taken to a hospital after arriving at 70 km in a 90-km ultramarathon with confusion. Presuming she was dehydrated, she was given 1 L of intravenous NS. She subsequently suffered a grand mal epileptic seizure and lapsed into coma. Her serum sodium concentration was found to be 115 mEq/L, and she was found to have neurogenic (noncardiac) pulmonary edema.

Given the supporting evidence for adverse consequences from intravenous NS hydration in athletes with EAH, the evidence that HTS is effective in treating EAH, and the finding that there is no evidence for life-threatening conditions from exercise-related dehydration resulting in a need for intravenous rehydration, we stand by our recommendation that "hypotonic or isotonic fluid intake should be restricted in known or suspected EAH until urination begins." A recommendation grade 1A is supported as there is overwhelming evidence from the series of observational studies we cite to support this recommendation. We also believe that our recommendation for the use of HTS rather than NS in symptomatic confirmed or suspected EAH is well supported from the publications described above.

With regard to Dr Lipman's proposed algorithm for treatment of EAH without point-of-care confirmation, we can agree that the right and left sides of his treatment scheme are consistent with our position. It is the central portion of the algorithm outlining treatment for the athlete who is symptomatic for EAH and dehydration with intravenous infusion of 1 to 2 L of NS where we strongly disagree. The cases described above demonstrating the adverse consequences of such treatment provide the support for our stance.

Finally, we would like to make it clear to your readers that a Letter to the Editor does not undergo peer review. We have expressed our concern to the Editor and asked that Dr Lipman's contradictory algorithm not be published without peer review given that a published

algorithm becomes easy for a reader to use without scrutinizing the accompanying text. We hope that the readers of your journal will closely review our response before accepting this algorithm.

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Painful Sting After Exposure to *Dendrocnide* sp: Two Case Reports

To the Editor:

The stinging trees, or *Dendrocnide* sp, are native plants from the rainforest area of northern Australia and Southeast Asia and belong to the Urticaceae family. When touched, these plants are known for inducing severe pain that can last for months. We describe in this letter 2 French tourists who came in contact with stinging trees in March 2012.

In the first case, a 49-year-old man without medical history vacationed to the Philippines on the island of Mindoro. While walking in the tropical rainforest, he happened to contact the stinging tree leaves (*Dendrocnide* sp). A few hours later, the volar region of both his hands

became painful with a burning sensation, and he complained about paresthesia of the soles of his feet. He claimed he had not been in contact with cnidarians (marine organisms that eject nematocysts), which are another source of pruritus and painful reaction after skin exposure. Three weeks later, the subject still had moderate pruritus but no more burn injuries on his hand. However, paresthesia of both his feet continued. He was seen at the University Hospital Institute for Infectious and Tropical Diseases in Marseille.

In the second case, a 33-year-old man without medical history made contact with a *Dendrocnide* stem during his holidays in Vietnam. Within a few minutes, severe pruritus had developed on his ear and his arm. He quickly took a symptomatic treatment containing trolamine and acetaminophen. A family member contacted the Poison Control Center in Marseille where the doctor on duty advised him to disinfect the wounds and to continue the symptomatic treatment. Three days later, he observed a complete recovery of the burning wounds.

The *Dendrocnide* sp, or “stinging trees,” belong to the Urticaceae family, like the European stinging nettle. There are about 37 species within the *Dendrocnide* genus: evergreen trees (for example, *D excelsa*, *D photinophylla*, *D meyeniana*) and shrubs (for example, *D moroides*, *D cordifolia*). The leaves are cordate, alternate, and covered with silicon/calcium-containing stinging hairs, as are the stems. Immature leaves are densely covered with stinging hairs, which become fewer in number on the surface of old leaves.^{1–3} They are often heavily chewed by insects, which gives a typical aspect to the leaves. The *Dendrocnide* sp fruits are aggregated, large, fleshy, and drupaceous and generally edible but some species (for example, *D excelsa*) possess stinging

Figure 1. *Dendrocnide moroides*.