

# National Athletic Trainers' Association Position Statement: Exertional Heat Illnesses

Douglas J. Casa, PhD, ATC, FNATA, FACSM\*; Julie K. DeMartini, PhD, ATC†; Michael F. Bergeron, PhD, FACSM‡; Dave Csillan, MS, ATC, LAT§; E. Randy Eichner, MD, FACSM||; Rebecca M. Lopez, PhD, ATC, CSCS¶; Michael S. Ferrara, PhD, ATC, FNATA#; Kevin C. Miller, PhD, ATC\*\*; Francis O'Connor, MD, MPH, FACSM††; Michael N. Sawka, PhD, FACSM‡‡; Susan W. Yeargin, PhD, ATC§§

\*University of Connecticut, Storrs; †Sacred Heart University, Fairfield, CT; ‡Youth Sports of the Americas, Birmingham, AL; §Ewing High School, NJ; ||University of Oklahoma Health Sciences Center, Oklahoma City; ¶University of South Florida, Tampa; #University of New Hampshire, Durham; \*\*Central Michigan University, Mount Pleasant; ††Uniformed Services University, Bethesda, MD; ‡‡Georgia Institute of Technology, Atlanta; §§University of South Carolina, Columbia

**Objective:** To present best-practice recommendations for the prevention, recognition, and treatment of exertional heat illnesses (EHIs) and to describe the relevant physiology of thermoregulation.

**Background:** Certified athletic trainers recognize and treat athletes with EHIs, often in high-risk environments. Although the proper recognition and successful treatment strategies are well documented, EHIs continue to plague athletes, and exertional heat stroke remains one of the leading causes of sudden death during sport. The recommendations presented in this document provide athletic trainers and allied health providers with an integrated scientific and clinically applicable approach to the prevention, recognition, treatment of, and return-to-activity guidelines for EHIs. These recommendations are given so that

proper recognition and treatment can be accomplished in order to maximize the safety and performance of athletes.

**Recommendations:** Athletic trainers and other allied health care professionals should use these recommendations to establish onsite emergency action plans for their venues and athletes. The primary goal of athlete safety is addressed through the appropriate prevention strategies, proper recognition tactics, and effective treatment plans for EHIs. Athletic trainers and other allied health care professionals must be properly educated and prepared to respond in an expedient manner to alleviate symptoms and minimize the morbidity and mortality associated with these illnesses.

**Key Words:** heat cramps, heat syncope, heat exhaustion, heat injury, heat stroke, dehydration



The prevention, recognition, and treatment of exertional heat illnesses (EHIs) are core components of sports medicine services at all levels of sport. The risk of EHI is ever present during exercise in the heat but can also occur in “normal” environmental conditions. Our current knowledge base has allowed us to greatly enhance the level of care that can be provided for athletes with these medical conditions. This document serves as the current position statement for the National Athletic Trainers’

Association (NATA) and replaces the document that was published in 2002.<sup>1</sup>

The care of exertional heat-stroke (EHS) patients has come a long way in the past millennia. We now possess the knowledge to nearly assure survival from this potentially fatal injury if EHS is quickly and appropriately recognized and treated at the time of collapse.<sup>2,3</sup> Additionally, our knowledge base and proven management protocols allow us to establish effective prevention and management strategies to minimize the risk of and improve the outcome

from EHS, thereby affecting public health via policy creation and modification.

## DEFINITIONS OF EHIS

### Exercise-Associated Muscle Cramps

Exercise-associated muscle cramps (EAMCs) are sudden or sometimes progressively and noticeably evolving, involuntary, painful contractions of skeletal muscle during or after exercise.<sup>4,5</sup> *Heat cramps* is a popular but technically inappropriate term for a certain category of EAMCs because they are not directly related to an elevated body temperature,<sup>5,6</sup> do not readily occur after passive heating at rest, and can present during exercise in warm or even cool<sup>6-8</sup> and temperature-controlled conditions,<sup>9</sup> although extensive sweating is typical. The signs and symptoms of incipient EAMCs can be described as *tics*, *twinges*, *stiffness*, *tremors*, or *contractures*, but these terms refer to conditions that are typically painless and do not demonstrate muscle activity on electromyography, unlike full-blown EAMCs.<sup>10</sup> The cause of EAMCs is not fully confirmed; proposed contributing factors and conditions include dehydration,<sup>5</sup> electrolyte imbalances,<sup>5,11</sup> altered neuromuscular control,<sup>4</sup> fatigue, or any combination of these factors.<sup>5-10</sup>

### Heat Syncope

Heat syncope, or orthostatic dizziness, often occurs in unfit or heat-unacclimatized persons who stand for a long period of time in the heat or during sudden changes in posture in the heat, especially when wearing a uniform or insulated clothing that encourages and eventually leads to maximal skin vasodilation. This condition is often attributed to dehydration, venous pooling of blood, reduced cardiac filling, or low blood pressure with resultant cerebral ischemia.<sup>12</sup> Heat syncope usually occurs during the first 5 days of unaccustomed heat exposure (eg, during the preseason), before the blood volume expands and cardiovascular adaptations are complete, and in those with heart disease or taking diuretics.<sup>13</sup>

### Heat Exhaustion

Heat exhaustion is the inability to effectively exercise in the heat, secondary to a combination of factors, including cardiovascular insufficiency, hypotension, energy depletion, and central fatigue.<sup>14</sup> This condition is manifested by an elevated core body temperature (usually <40.5°C) and is often associated with a high rate or volume of skin blood flow, heavy sweating, and dehydration.<sup>15</sup> It occurs most frequently in hot or humid (or both) conditions, but it can also occur in normal environmental conditions with intense physical activity. Heat exhaustion most often affects heat-unacclimatized or dehydrated individuals with a body mass index >27 kg/m.<sup>16</sup> By definition, absent from heat exhaustion are end-organ damage, which would indicate heat injury (eg, renal insufficiency, rhabdomyolysis, or liver injury), and significant central nervous system (CNS) dysfunction with marked temperature elevation (>40.5°C [105°F]), which would indicate the possibility of EHS.

### Exertional Heat Injury

Heat injury is a moderate to severe heat illness characterized by organ (eg, liver, renal) and tissue (eg, gut, muscle) injury associated with sustained high body temperature resulting from strenuous exercise and environmental heat exposure. Body temperature is usually but not always greater than 40.5°C (105°F).<sup>17,18</sup>

### Exertional Heat Stroke

Exertional heat stroke is the most severe heat illness. It is characterized by neuropsychiatric impairment and a high core body temperature, typically >40.5°C (105°F).<sup>16,19</sup> This condition is a product of both metabolic heat production and environmental heat load and occurs when the thermoregulatory system becomes overwhelmed due to excessive heat production (ie, metabolic heat production from the working muscles) or inhibited heat loss (ie, decreased sweating response, decreased ability to evaporate sweat) or both. Although this illness is most likely to occur in hot and humid weather, it can manifest with intense physical activity in the absence of extreme environmental conditions. The first sign of EHS is often CNS dysfunction (eg, collapse, aggressiveness, irritability, confusion, seizures, altered consciousness).<sup>19</sup> A medical emergency, EHS can progress to a systemic inflammatory response and multi-organ system failure unless promptly and correctly recognized and treated. The risks of morbidity and mortality increase the longer an individual's body temperature remains elevated above the critical threshold (>40.5°C [105°F]) and are significantly reduced if body temperature is lowered promptly.<sup>20</sup>

## RECOMMENDATIONS

The NATA advocates the following prevention, recognition, and treatment strategies for EHIs. These recommendations are presented to help certified athletic trainers and other health care providers maximize health, safety, and sport performance. However, individual responses to physiologic stimuli and environmental conditions vary widely. Therefore, these recommendations do not guarantee full protection from exertional heat-related illnesses but could mitigate the risks associated with athletic participation and physical activity. These recommendations and prevention strategies should be carefully considered and implemented by certified athletic trainers and the health care team as part of an overall strategy for the prevention and treatment of EHIs. The strength of each recommendation follows the Strength of Recommendation taxonomy (SORT; Table 1).<sup>21</sup>

### Prevention

1. Conduct a thorough, physician-supervised preparticipation medical screening before the start of the season to identify athletes with risk factors for heat illness or a history of heat illness (Table 2).<sup>22,23</sup> *Strength of recommendation: C*
2. Individuals should be acclimatized to the heat gradually over 7 to 14 days.<sup>22-26</sup> Heat acclimatization involves progressively increasing the intensity and duration of physical activity and phasing in protective equipment (if

**Table 1. Strength of Recommendation Taxonomy (SORT)<sup>a</sup>**

Strength of Recommendation	Definition
A	Recommendation based on consistent and good quality experimental evidence (morbidity, mortality, exercise and cognitive performance, physiologic responses).
B	Recommendation based on inconsistent or limited quality experimental evidence.
C	Recommendation based on consensus; usual practice; opinion; disease-oriented evidence <sup>b</sup> ; case series or studies of diagnosis, treatment, prevention, or screening; or extrapolations from quasi-experimental research.

<sup>a</sup> Reprinted with permission from Ebell MH, Siwek J, Weiss BD, et al, Strength of recommendation taxonomy (SORT): a patient-centered approach to grading evidence in the medical literature, 2004;69(3):548–556, *Am Fam Physician*. Copyright 2004 American Academy of Family Physicians. All Rights Reserved.<sup>14</sup>

<sup>b</sup> Patient-oriented evidence measures outcomes that matter to patients: morbidity, mortality, symptoms improvement, cost reduction, and quality of life. Disease-oriented evidence measures intermediate, physiologic, or surrogate end points that may or may not reflect improvements in patient outcomes (eg, blood pressure, blood chemistry, physiologic function, pathologic finding).

applicable). If heat acclimatization is not maintained, the physiologic benefits provided by this process will decay within 3 weeks.<sup>24–26</sup> The first 2–3 weeks of preseason practice typically present the greatest risk of EHI, particularly in equipment-intensive sports.<sup>26,27–29</sup> All possible preventive measures should be used during this time to address this high-risk period (Figure 1). *Strength of recommendation: B*

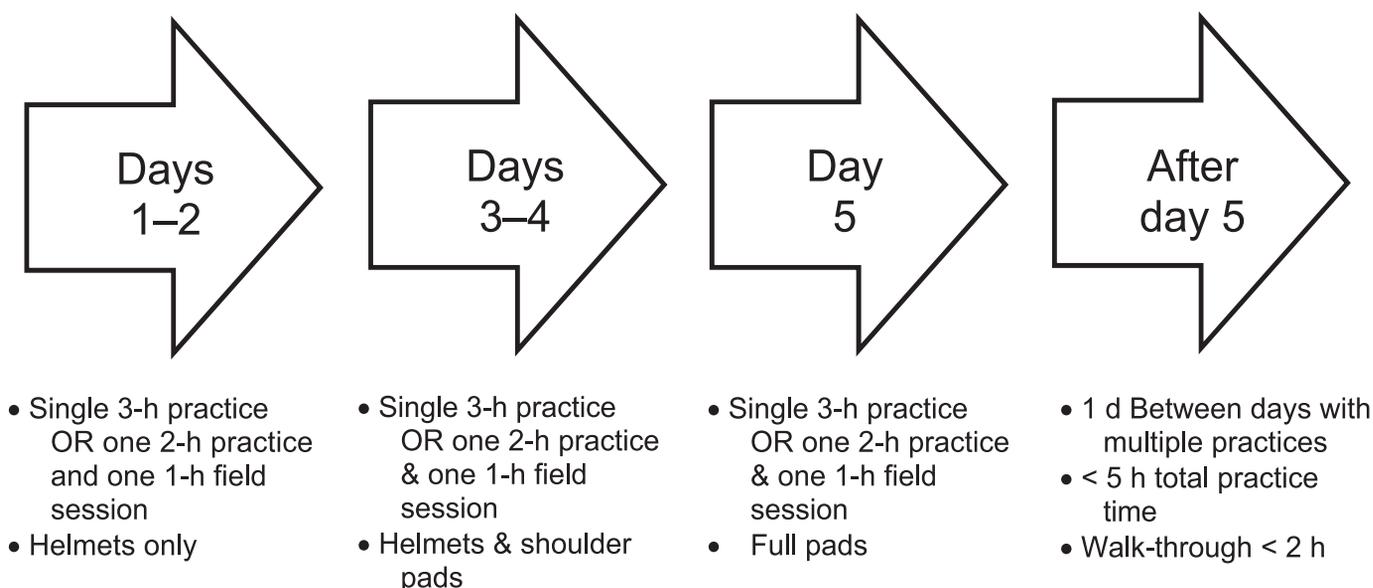
- Athletes who are currently sick with a viral infection (eg, upper respiratory tract infection or gastroenteritis) or other illness or have a fever or serious skin rash should not participate until the condition is resolved.<sup>16,27,30</sup> Even after symptoms resolve, the athlete may still be susceptible to heat illness and should be observed carefully upon return to exercising in the heat. *Strength of recommendation: B*
- Individuals should maintain euhydration and appropriately replace fluids lost through sweat during and after games and practices (see the NATA position statement on fluid replacement in athletes<sup>31</sup>). Players should have free access to readily available fluids at all times, not just during designated breaks. Instruct them to eat or drink

appropriate sodium-containing fluids and foods to help replace sodium losses in sweat and urine and to enhance hydration (ie, water retention and distribution). The aims of fluid consumption or replacement are to prevent a body mass loss of more than 2% (as measured before and after the practice or game) and to keep morning urine light in color.<sup>31,32</sup> These strategies may reduce the risk of acute and chronic significant dehydration and decrease the risk of EHI.<sup>27,31–34</sup> *Strength of recommendation: B*

- The sports medicine staff must educate relevant personnel (ie, coaches, administrators, security guards, emergency medical services [EMS] staff, athletes) on preventing and recognizing EHI and, in particular, EHS.<sup>35,36</sup> Signs and symptoms of a medical emergency should be reviewed, and every institution should have and personnel should practice an emergency action plan specific to each practice and game site. Review and rehearsal of the emergency action plan should include all relevant members of the sports medicine team (ie, coaches, athletic trainers, EMS). *Strength of recommendation: C*
- Appropriate medical care must be available, and all personnel must be familiar with EHI prevention, recognition, and treatment.<sup>35–37</sup> Certified athletic trainers and other health care providers covering practices or events are the primary providers of medical care for athletes who display signs or symptoms of EHI and have the authority to restrict an athlete from participating if EHI is suspected or to refer the athlete for a significant EHI condition. *Strength of recommendation: C*
- When environmental conditions warrant, a cold-water or ice tub and ice towels should be available to immerse or soak a patient with a suspected heat illness.<sup>33,37</sup> Immediate whole-body cooling is essential for treating EHI and EHS in particular. Onsite facilities are needed for immediate treatment. *Strength of recommendation: B*
- The assessment of rectal temperature is the clinical gold standard for obtaining core body temperature of patients with EHS<sup>38</sup> and the medical standard of practice and accepted protocol. No other field-expedient methods of obtaining core body temperature (eg, oral, axillary, tympanic, forehead sticker, temporal) are valid or reliable after intense exercise in the heat, and they may lead to inadequate or inappropriate treatment, thereby endangering a patient's health.<sup>38–41</sup> Parents, administrators, coaches, and student-athletes should be educated ahead of time that this procedure will be used for heat-illness emergencies, especially in patients suspected of having heat exhaustion or EHS. Esophageal and gastrointestinal

**Table 2. Sample Preparticipation Physical Examination Questions Related to Exertional Heat Stroke<sup>69</sup>**

- Have you ever previously been diagnosed with exertional heat stroke? If yes
  - How long ago?
  - Have you had any complications since then?
  - How long did it take you to return to full participation?
  - Did you have any complications upon your return to play?
  - Was an exercise heat tolerance test conducted to assess your thermoregulatory capacity?
- Have you ever been diagnosed with heat exhaustion? If yes
  - When?
  - How many times?
- Have you ever had trouble or complications from exercising in the heat (eg, feeling sick, throwing up, dizzy, lack of energy, decreased performance, muscle cramps)?
- How much training have you been doing recently (in the past 2 weeks)? Has this been performed in warm or humid weather?
- Have you been training during the last 2 months? Would you say you are in poor, good, or excellent condition?
- Describe your drinking habits. (Are you conscious of how much you consume? Is your urine consistently dark?)
- Would you consider yourself a heavy or a salty sweater?
- How many hours of sleep do you get per night? Do you sleep in an air-conditioned room?
- Do you take any supplements or ergogenic aids?



**Figure 1. National Collegiate Athletic Association heat-acclimatization guidelines.**

(via ingestible thermistor) measurements may be appropriate alternatives for temperature assessment but require advanced training for the former and careful planning for the latter. Under all circumstances in which EHS is possible, a rectal temperature assessment should be able to be obtained. *Strength of recommendation: A*

9. Because the effects of heat are cumulative, athletes should be encouraged to sleep at least 7 hours per night in a cool environment; eat a balanced diet; and properly hydrate before, during, and after exercise.<sup>16</sup> Individuals should also be advised to rest in a cool environment during periods of inactivity (eg, off days, between sessions on double-practice days) to maximize recovery. Rest periods should incorporate meal times and allow 2 to 3 hours for food, fluids, electrolytes (primarily sodium and chloride), and other nutrients to be digested and absorbed before the next practice or competition. *Strength of recommendation: C*
10. To anticipate potential problems, a preseason heat-acclimatization policy should be developed for organized sports and event guidelines formulated for hot, humid weather conditions based on the type of activity and wet-bulb globe temperature (WBGT).<sup>23,26</sup> In stressful environmental conditions, particularly during the first 2–3 weeks of preseason practice, activity should be delayed or rescheduled or the practice session shortened to reduce the risk to participants. Special attention should be given to practice drills that involve high-intensity activity and full protective equipment worn by players, as these factors may exacerbate the amount of heat stress on the body. *Strength of recommendation: B*
11. Individuals who may be particularly susceptible to EHI must be identified.<sup>42–45</sup> They should be closely monitored during stressful environmental conditions, and preventive steps should be taken.<sup>45,46</sup> In addition, emergency supplies and equipment (eg, tubs for cold-water immersion [CWI], rectal thermometer) should be onsite, easily accessible, and in good working order to allow for immediate intervention and treatment if needed. *Strength of recommendation: B*

12. Rest breaks should be planned and the work-to-rest ratio modified to match the environmental conditions and the intensity of the activity.<sup>45–47</sup> Breaks should be in the shade or in a predetermined cooling zone and should allow enough time for all athletes to consume fluids. Additionally, players should be permitted to remove equipment (eg, helmets) during rest periods. *Strength of recommendation: B*
13. The use of dietary supplements and other substances that have a dehydrating effect, increase metabolism, or affect body temperature and thermoregulation is discouraged.<sup>48</sup> Because supplements may increase the risk of EHI, their use should be carefully monitored. *Strength of recommendation: C*
14. Minimal experimental evidence exists regarding the most effective method of preventing EAMCs due to the variety of causes. Supplemental sodium ingestion and fluid monitoring<sup>9</sup> or neuromuscular reeducation<sup>49</sup> may help to prevent EAMC recurrences. Clinicians should identify the patient's unique intrinsic (eg, hydration, acclimatization, biomechanics, training status) and extrinsic (eg, climate conditions, exercise intensity) risk factors that preceded EAMCs before implementing a prevention strategy. *Strength of recommendation: C*

## Recognition

### Exercise-Associated Muscle Cramps.

15. A patient experiencing EAMCs will likely show 1 or more of the following signs and symptoms: visible cramping in part or all of the muscle or muscle groups, localized pain, dehydration, thirst, sweating, or fatigue.<sup>4,5,50</sup> *Strength of recommendation: C*
16. A thorough medical history should be obtained to distinguish muscle cramping as a result of an underlying clinical condition (eg, sickle cell trait) from EAMCs.<sup>50</sup> The latter is often preceded by subtle muscle twitching,<sup>4</sup> whereas the former is not. *Strength of recommendation: C*
17. Most EAMCs related to overload or fatigue tend to be short in duration (less than 5 minutes) and mild in

severity.<sup>7,51</sup> However, some EAMCs severely affect athletic performance and as a result, prohibit further exercise; require further medical attention to resolve; or elicit soreness for several days.<sup>7,49–51</sup> *Strength of recommendation: B*

### Heat Syncope.

18. A patient who experiences a brief episode of fainting associated with dizziness, tunnel vision, pale or sweaty skin, and a decreased pulse rate while standing in the heat or after vigorous exercise (with a relatively low rectal temperature [ $<39^{\circ}\text{C}$ ]) is likely experiencing heat syncope.<sup>12</sup> However, responsiveness, breathing, and pulse must be assessed to rule out a cardiac event, which can present with similar signs and symptoms but is a more serious condition. *Strength of recommendation: B*
19. A thorough medical history and physical examination should be performed to eliminate any other medical conditions that could cause syncope. *Strength of recommendation: C*

### Exertional Heat Exhaustion.

20. Heat exhaustion may be present if the patient demonstrates excessive fatigue, faints, or collapses with minor cognitive changes (eg, headache, dizziness, confusion) while performing physical activity,<sup>15</sup> yet the athletic trainer should assess the patient's CNS function by noting any bizarre behavior, hallucinations, altered mental status, confusion, disorientation, or coma that may indicate a more serious condition such as EHS. Other signs and symptoms of exertional heat exhaustion may include fatigue, weakness, dizziness, headache, vomiting, nausea, lightheadedness, low blood pressure, and impaired muscle coordination. *Strength of recommendation: B*
21. It is strongly recommended that a rectal temperature be obtained to differentiate exertional heat exhaustion from the more serious EHS. With heat exhaustion, core body temperature (measured rectally) is usually less than  $40.5^{\circ}\text{C}$  ( $105^{\circ}\text{F}$ ), a key characteristic that differentiates it from EHS. *Strength of recommendation: A*

### Exertional Heat Stroke.

22. The 2 main diagnostic criteria for EHS are CNS dysfunction and a core body temperature greater than  $40.5^{\circ}\text{C}$  ( $105^{\circ}\text{F}$ ).<sup>16,19,52</sup> However, if a suspected EHS victim exhibits CNS dysfunction even though the rectal temperature is slightly lower (ie,  $40^{\circ}\text{C}$  [ $104^{\circ}\text{F}$ ]), it is prudent to assume the patient is suffering from EHS and begin the appropriate treatment. After initial collapse, recognition is often delayed, and the patient may begin to cool passively, dropping below the  $40.5^{\circ}\text{C}$  ( $105^{\circ}\text{F}$ ) threshold. Rectal temperature thermometry is the only method of obtaining an immediate and accurate measurement of core body temperature. Other devices, such as oral, axillary, aural canal, tympanic, forehead sticker, and temporal artery thermometers, inaccurately assess the body temperature of an exercising person.<sup>38–41</sup> A delay in accurately assessing temperature during diagnosis may also explain a body temperature that is lower than expected. *Strength of recommendation: A*
23. Because immediate treatment is vital in EHS, it is important to not waste time by substituting an invalid method of temperature assessment if rectal thermometry

is not available. Instead, the practitioner should rely on other key diagnostic indicators (ie, CNS dysfunction, circumstances of the collapse). If EHS is suspected, CWI (or another rapid cooling mechanism if CWI is not available) should be initiated immediately. *Strength of recommendation: C*

24. In a patient suspected of having EHS, CNS function should be assessed. Signs and symptoms can include disorientation, confusion, dizziness, loss of balance, staggering, irritability, irrational or unusual behavior, apathy, aggressiveness, hysteria, delirium, collapse, loss of consciousness, and coma. In some cases, a lucid interval may be present; however, if EHS is present, the patient will likely deteriorate quickly. *Strength of recommendation: B*
25. Other signs and symptoms of EHS that may be present include dehydration, hot and wet skin, hypotension, and hyperventilation. Most patients with EHS have hot, sweaty skin as opposed to those with the classical type of heat stroke (the passive condition that typically affects children and the elderly), who present with dry skin. (Table 3). *Strength of recommendation: B*

### Heat Injury.

26. Heat injury is a moderate to severe heat illness characterized by end-organ damage but the absence of the profound CNS dysfunction often found with EHS.<sup>17,18</sup> Evaluation usually reveals very dark (cola-colored) urine, severe muscle pain, and abnormal blood chemistry levels. *Strength of recommendation: B*

## Treatment

### Exercise-Associated Muscle Cramps.

27. The immediate treatment for acute EAMCs related to muscle overload or fatigue is rest and passive static stretching of the affected muscle until cramps abate.<sup>7,51,53</sup> Icing, massage, or both may also help relieve some of the discomfort after EAMCs.<sup>5</sup> For EAMCs related to excessive sweating and a suspected whole-body sodium deficit, the patient must ingest sodium-containing fluids (preferably) or foods (or both) to help return the body to normal fluid, electrolyte, and energy distribution. *Strength of recommendation: B*
28. Fluid absorption, retention, and distribution are enhanced by beverages that contain sodium and carbohydrates. A high-sodium product (eg, salt packet) may be added to a beverage to help offset sodium lost via exercise-induced sweating. Similarly, small volumes (eg, 1 mL per 1 kg body weight) of a salty solution such as pickle juice may be consumed, if tolerated, without negatively affecting ad libitum water ingestion,<sup>54</sup> plasma electrolyte concentrations,<sup>55</sup> or thirst or causing nausea or stomach fullness.<sup>54</sup> *Strength of recommendation: B*
29. Patients with EAMCs are normally conscious and responsive and have normal vital signs.<sup>50</sup> Thus, clinicians can provide fluids orally to a patient suffering from EAMCs who is compliant and tolerating fluid intake. The use of intravenous fluids should be considered if the patient is noncompliant or unable to tolerate fluids.<sup>5</sup> *Strength of recommendation: A*
30. Patients with recurring EAMCs should undergo a thorough medical screening to rule out more serious

**Table 3. Clinical Distinctions of Exertional Heat Illnesses**

Characteristic	Heat Illness			
	Exercise-Associated Muscle (Heat) Cramps	Heat Syncope	Heat Exhaustion	Exertional Heat Stroke
Description	Acute, painful, involuntary muscle contractions presenting during or after exercise	Collapsing in the heat, resulting in loss of consciousness	Inability to continue exercise due to cardiovascular insufficiency	Severe hyperthermia leading to overwhelming of the thermoregulatory system
Physiologic cause	Dehydration, electrolyte imbalances, and/or neuromuscular fatigue	Standing erect in a hot environment, causing postural pooling of blood in the legs	High skin blood flow, heavy sweating, and/or dehydration, causing reduced venous return	High metabolic heat production and/or reduced heat dissipation
Primary treatment factors	Stop exercising, provide sodium-containing beverages	Lay patient supine and elevate legs to restore central blood volume	Cease exercise, remove from hot environment, elevate legs, provide fluids	Immediate whole-body cold-water immersion to quickly reduce core body temperature
Recovery	Often occurs within minutes to hours	Often occurs within hours	Often occurs within 24 h; same-day return to play not advised	Highly dependent on initial care and treatment; further medical testing and physician clearance required before return to activity

neuromuscular conditions (eg, fatigue, hydration level, improper nutrition).<sup>50</sup> *Strength of recommendation: C*

**Heat Syncope.**

- 31. The clinician should move the patient to a shaded area, monitor vital signs, elevate the legs above the level of the heart, cool the skin, and rehydrate.<sup>12</sup> *Strength of recommendation: C*

**Exertional Heat Exhaustion.**

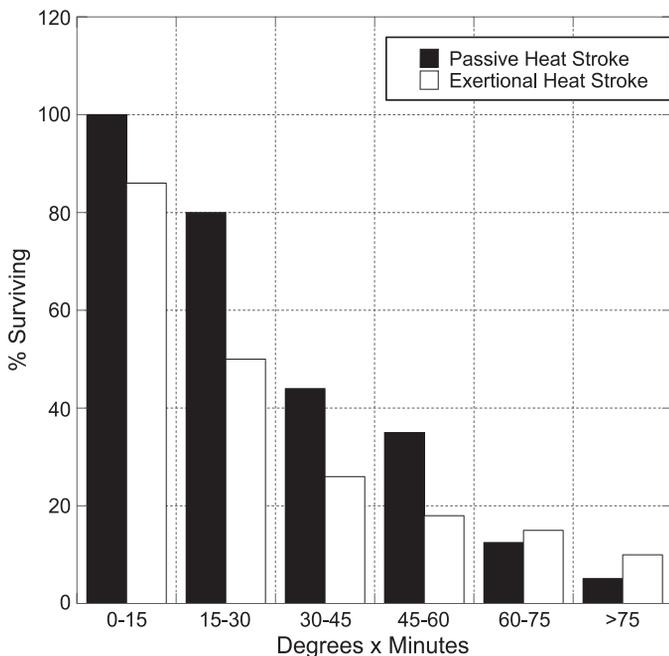
- 32. Removing any excess clothing and equipment increases the evaporative surface of the skin and facilitates cooling. *Strength of recommendation: C*
- 33. The patient should be moved to a cool or shaded area. Further body cooling should be accomplished via fans or ice towels if necessary. *Strength of recommendation: C*
- 34. While monitoring vital signs, the clinician should place the patient in the supine position with legs elevated above the level of the heart to promote venous return.<sup>15,16,56</sup> *Strength of recommendation: C*
- 35. If intravenous fluids are needed or if recovery is not rapid (within 30 minutes of initiation of treatment) and uneventful, fluid replacement should begin and the patient’s care transferred to a physician. If the condition worsens during or after treatment, EMS should be activated.<sup>15,16</sup> Additionally, rectal temperature should be obtained; if >40.5°C (105°F), the patient should be treated for EHS. *Strength of recommendation: C*

**Exertional Heat Stroke.**

- 36. For any EHS patient, the goal is to lower core body temperature to less than 38.9°C (102°F) within 30 minutes of collapse.<sup>20</sup> Body cooling serves 2 purposes: returning blood flow from the skin to the heart and lowering core body temperature by reducing the hypermetabolic state of the organs. The length of time the core body (and particularly the brain) is above the critical temperature threshold (40.5°C [105°F]) dictates morbidity and the risk of death from EHS (Figure 2).<sup>57,58</sup> *Strength of recommendation: B*
- 37. When EHS is suspected, the patient’s body (trunk and extremities) should be quickly immersed in a pool or tub

of cold water. Removing excess clothing and equipment will enhance cooling by maximizing the surface area of the skin. However, because removing excess clothing and equipment can be time consuming, CWI should begin immediately and equipment should be removed while the patient is in the tub (or while temperature is being assessed or the tub is being prepared).<sup>59</sup> Rectal temperature and other vital signs should be monitored during cooling every 5 to 10 minutes if a continuous monitoring device is not available.<sup>20,60</sup> *Strength of recommendation: B*

- 38. Cold-water immersion up to the neck is the most effective cooling modality for patients with EHS.<sup>57</sup> The water should be approximately 1.7°C (35°F) to 15°C (59°F) and stirred continuously to maximize cooling. The patient should be removed when core body temperature reaches 38.9°C (102°F) to prevent overcooling (Table 4).<sup>60</sup> *Strength of recommendation: A*
- 39. Although cooling rates may vary, the cooling rate for CWI will be approximately 0.2°C/min (0.37°F/min) or about 1°C every 5 minutes (or 1°F every 3 minutes) when considering the entire immersion period from postcollapse to 38.9°C (102°F).<sup>20,57,58</sup> *Strength of recommendation: B*
- 40. If full-body CWI is not available, partial-body immersion (ie, torso) with a small pool or tub and other modalities, such as wet ice towels rotated and placed over the entire body or cold-water dousing with or without fanning, may be used but are not as effective as CWI.<sup>61,62</sup> *Strength of recommendation: B*
- 41. If a physician is onsite (as in a mass medical tent situation) and can manage the EHS, then transportation to a medical facility may not be necessary if cooling occurred immediately (ie, if the duration above 40°C [104°F] was less than 30 minutes) and the patient is asymptomatic 1 hour postcooling. If a physician is not present but other medical staff (eg, AT, EMS, nurse) are onsite, aggressive cooling should continue until the patient’s temperature is 39°C (102.8°F). When medical staff is onsite, all patients with EHS should be cooled first and transported second. However, when medical staff is not present and EHS is suspected, then the coaching



**Figure 2. Relationship between severity of hyperthermia and rat survivability.** Reprinted with permission. Casa DJ, Kenny JP, Taylor NAS. Immersion treatment for exertional hyperthermia: cold or temperate water? *Med Sci Sports Exerc.* 2010;42(7):1246–1252. Promotional and commercial use of the material in print, digital, or mobile-device format is prohibited without the permission from publisher Wolters Kluwer Health. Please contact [lwjournalpermissions@wolterskluwer.com](mailto:lwjournalpermissions@wolterskluwer.com) for further information.

staff/supervisors should implement cooling until medical assistance arrives. *Strength of recommendation: B*

42. Policies and procedures for cooling patients before transport to the hospital must be explicitly stated in an emergency action plan and shared with potential EMS responders so that treatment of EHS by all medical professionals is coordinated (Figure 3). *Strength of recommendation: B*

### Return to Activity

43. In cases of EAMCs or heat syncope, the athletic trainer should monitor the patient's condition until signs and symptoms are no longer present. *Strength of recommendation: C*
44. In patients with heat exhaustion, same-day return to activity is not recommended and should be avoided.<sup>15,56</sup> *Strength of recommendation: C*
45. Many patients with EHS are cooled effectively and sent home the same day<sup>63</sup>; they may be able to resume modified activity within 1 month with a physician's clearance. However, when treatment is delayed (ie, not provided within 30 minutes), patients may experience residual complications for months or years after the event. *Strength of recommendation: C*
46. Most guidelines suggest that a patient recovering from EHS be asymptomatic with normal blood-work results (renal and hepatic panels, electrolytes, and muscle enzyme levels) before a gradual return to activity is initiated.<sup>64</sup> Unfortunately, few evidence-based strategies have been developed to determine recovery of the

**Table 4. Guidelines for Implementing Cold-Water Immersion for a Patient With Exertional Heat Stroke**

- Initial response. Once exertional heat stroke is suspected, prepare to cool the patient and contact emergency medical services.
- Prepare for ice-water immersion. On the playing field or in close proximity, half-fill a stock tank or wading pool with water and ice (make sure there is a sufficient water source).
  - The tub can be filled with ice and water before the event begins (or have the tub half-filled with water and keep 3 to 4 coolers of ice next to the tub; this prevents having to keep the tub cold throughout the day.
  - Ice should cover the surface of the water at all times.
  - If the athlete collapses near the athletic training room, a whirlpool tub or cold shower may be used.
- Determine vital signs. Immediately before immersing the patient, obtain vital signs.
  - Assess core body temperature with a rectal thermistor.
  - Check airway, breathing, pulse, and blood pressure.
  - Assess the level of central nervous system dysfunction.
- Begin ice-water immersion. Place the patient in the ice-water-immersion tub. Medical staff, teammates/coaches, and volunteers may be needed to assist with entry to and exit from the tub.
- Total-body coverage. Cover as much of the body as possible with ice water while cooling.
  - If full-body coverage is not possible due to the tub size, cover the torso as much as possible.
  - To keep the patient's head and neck from going under water, an assistant may hold him or her under the axillae with a towel or sheet wrapped across the chest and under the arms.
  - Place an ice/wet towel over the head and neck while body is being cooled in the tub.
  - Use a water temperature under 15°C (60°F).
- Vigorously circulate the water. During cooling, water should be continuously circulated or stirred to enhance the water-to-skin temperature gradient, which optimizes cooling. Have an assistant stir the water during cooling.
- Continue medical assessment. Vital signs should be monitored at regular intervals.
- Fluid administration. If a qualified medical professional is available, an intravenous fluid line can be placed for hydration and support of cardiovascular function.
- Cooling duration. Continue cooling until the patient's rectal temperature lowers to 38.9°C (102°F).
  - If rectal temperature cannot be measured and cold-water immersion is indicated, cool for 10–15 min and then transport to a medical facility.
  - An approximate estimate of cooling via cold-water immersion is 1°C for every 5 min and 1°C for every 3 min (if the water is aggressively stirred). For example, someone in the tub for 15 min would cool approximately 3°C or 5°C during that time.
- Patient transfer. Remove the patient from the immersion tub only after rectal temperature reaches 38.9°C (102°F) and then transfer to the nearest medical facility via emergency medical services as quickly as possible.

thermoregulatory system,<sup>65</sup> so the medical professional must use clinical cues such as ongoing signs and symptoms, responses to a standard exercise heat-tolerance test, responses to gradually increasing exercise demands, and ability to acclimatize to the heat to make return-to-play decisions. *Strength of recommendation: C*

47. In all cases of EHS, after the patient has completed a 7- to 21-day rest period, demonstrated normal blood-work results, and obtained physician clearance, he or she may begin a progression of physical activity, supervised by the athletic trainer or other medical professional with knowledge of EHS treatment and care, from low intensity

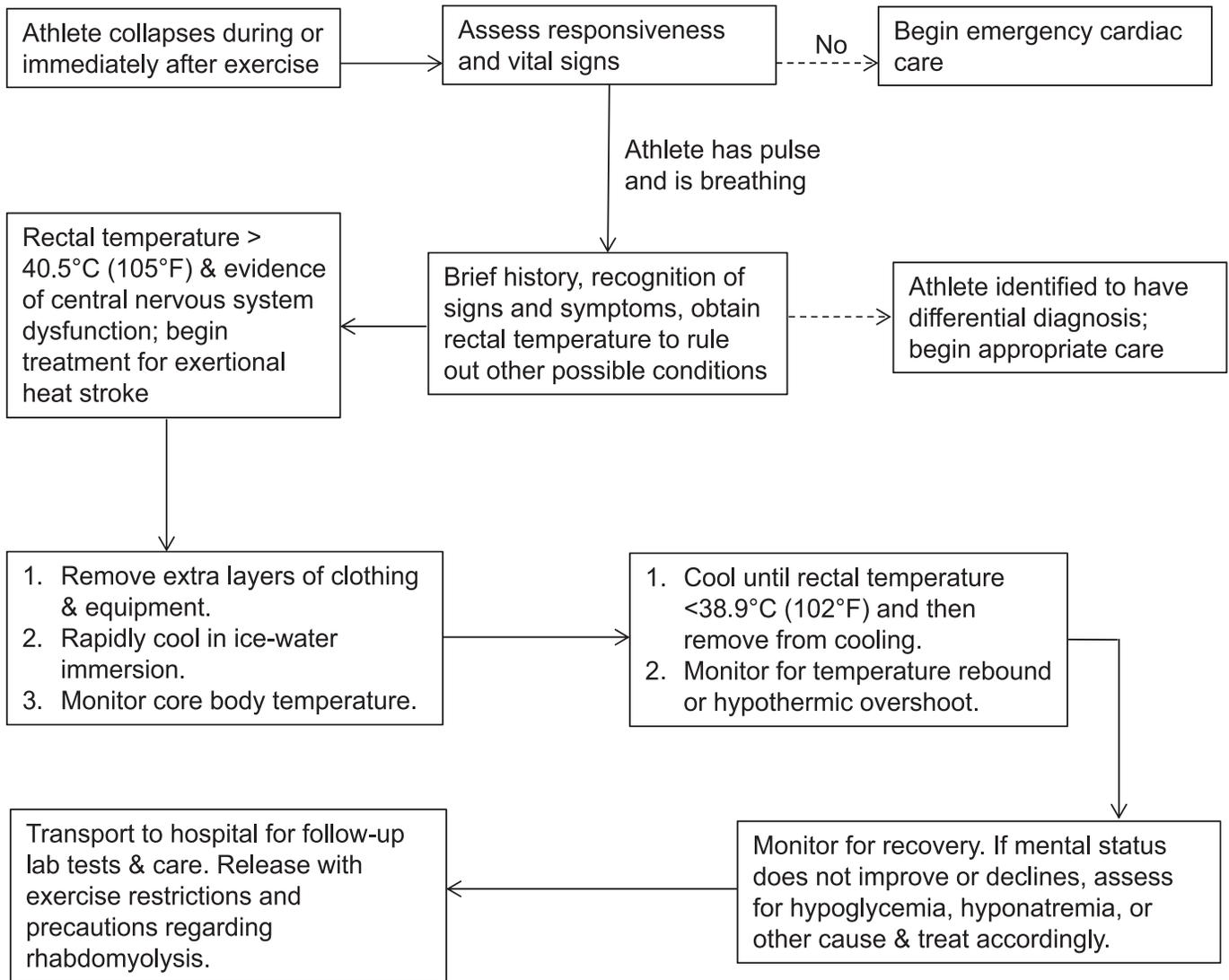


Figure 3. Algorithm for treatment of exertional heat stroke.

to high intensity and increasing duration in a temperate environment, with equipment added gradually where indicated. Also, a graded progression of heat acclimatization, while monitoring for signs and symptoms of EHI, should be completed. The ability to progress depends largely on the treatment provided, and in some rare cases, full recovery may not be possible. Rectal temperature and heart rate should be monitored during these activities, and if the patient experiences any side effects or negative symptoms with training, the progression should be slowed, delayed, or stopped.<sup>65,66</sup> *Strength of recommendation: C*

48. Although structured guidelines for return to play after EHS in athletics are lacking, the US military has adopted effective recommendations for the proper progression of return to duty after an episode of EHS. The main considerations are treating any associated sequelae and, if possible, identifying the cause of EHS, so that future episodes can be prevented.<sup>65-67</sup> As evidence-based medicine research has advanced, the role of exercise heat-tolerance testing has gained favor as a common-sense approach: a patient who has a poor test result should not increase activity at that point. However, the

significance of a normal test result and its relationship with clearance to return to play still need to be refined and evaluated. In either circumstance, monitoring the physiologic response to series of challenging exercise heat exposures is a large step forward in our delivery of health care to the EHS patient who is recovering and working toward a return to physical activity as a laborer, soldier, or athlete. This method has proved effective within the Israeli military<sup>68</sup> and the US military and at the Korey Stringer Institute, and it supports many of the considerations put forth by the American College of Sports Medicine and US military.<sup>65,67,69</sup> *Strength of recommendation: C*

## BACKGROUND AND REVIEW OF THE LITERATURE

### Thermoregulation

Thermoregulation is a complex interaction of the CNS, the cardiovascular system, and the skin to maintain a core body temperature of approximately 37°C (98.6°F).<sup>17,34,70,71</sup> The CNS temperature-regulation center, located in the

hypothalamus, is where the core temperature setpoint is determined. The hypothalamus receives information regarding core body temperature and skin temperature from peripheral skin receptors and the circulating blood. This interaction regulates core body temperature via an open-ended feedback loop similar to a home thermostat system. Based on the peripheral feedback sent to the hypothalamus, the body adjusts accordingly to initiate the appropriate heat-transfer responses. If core temperature falls below the normal setpoint, peripheral vasoconstriction and shivering responses increase core body temperature, whereas if core temperature rises above the normal setpoint, cutaneous vasodilation and increased sweating occur to dissipate heat.<sup>70,71</sup>

Core body temperature is determined by metabolic heat production and the transfer of body heat to and from the surrounding environment by the following heat-balance equation<sup>71</sup>:

$$S = M(\pm \text{work}) - E \pm R \pm C \pm K, \quad (1)$$

where S is the amount of stored heat, M is the metabolic heat production, E is the evaporative heat loss, R is the heat gained or lost by radiation, C is the heat lost or gained by convection, and K is the heat lost or gained by conduction. Basal metabolic heat production while fasting and at absolute rest is approximately 60 to 70 kcal/h for an average adult, with 50% of the heat being produced by internal organs.<sup>72</sup> Metabolic heat produced by intense exercise may approach 1000 kcal/h, with more than 90% of the heat resulting from metabolism in muscles.<sup>72</sup> Heat is further gained or lost by 1 or more of the following mechanisms.<sup>72</sup>

**Radiation:** Heat is transferred to or from an object or body via electromagnetic radiation (ie, sunlight) from higher to lower energy surfaces.

**Conduction:** Heat is transferred from warmer to cooler objects through direct physical contact (eg, ice packs).

**Convection:** Heat is transferred to or from the body to surrounding fluid or air (eg, moving air from a fan or immersion in water).

**Evaporation:** Heat is transferred via the vaporization of sweat. This is the most efficient means of heat transfer. The evaporation of sweat from the skin depends on the water saturation of the air (ie, humidity level) and the velocity of the moving air (ie, wind speed).<sup>17,30,70-73</sup> The effectiveness of evaporation for heat loss from the body diminishes rapidly when the humidity level is high.

### Exercise-Associated Muscle Cramps

The most common EHI experienced by athletes is EAMCs.<sup>74</sup> They afflict adolescents,<sup>9</sup> adult athletes,<sup>7,51,74,75</sup> soldiers, and industrial workers. They are seemingly unpredictable, though affected athletes often report muscle twinges before they experience full-blown, debilitating muscle cramping. Probably because spinal inhibition is weakest when a muscle contracts forcefully while shortened, EAMCs usually occur when muscles are in this position.<sup>76</sup> Although they may occur in any muscle, EAMCs related to muscle overload or fatigue tend to affect exercising (or constantly loaded) muscles, especially those that cross 2 joints in the lower extremities (eg, gastrocnemius, hamstrings).<sup>7</sup> Patients experiencing EAMCs

typically display a transient inability to continue normal activity and may have muscle soreness for days postcramping.<sup>50</sup>

Their cause is controversial,<sup>4,10</sup> but a growing body of experimental,<sup>76-79</sup> quasiexperimental,<sup>6-8,51,75</sup> and case<sup>49</sup> studies suggest that EAMCs are not the result of dehydration or electrolyte losses. Although athletes prone to EAMCs may have substantial fluid (2 to 3.4 L/h) and sodium (up to 5 g or far more in a single session) losses,<sup>9,11</sup> the volume of fluid ingested,<sup>11</sup> postexercise body weights,<sup>7,8,11</sup> and gross sweat losses<sup>11</sup> are often comparable with those of noncramping athletes. These findings underscore that such measures (or blood sodium concentration) do not necessarily indicate a whole-body sodium deficit. Potential risk factors for EAMCs consistent across several prospective cohort studies<sup>7,51,75,80</sup> include a history of EAMCs; faster competition performance times; and prior muscle, tendon, or ligament injury. In contrast, stretching history, muscle flexibility, training frequency or volume, height, age, body mass index, weight, and sex tend not to be adequate predictors of EAMC occurrence.<sup>7,51,75,80</sup>

The most effective treatment for acute EAMCs is static stretching of the affected muscle until the cramp subsides.<sup>7,51,53</sup> Stretching reduces the activity of cramping muscles<sup>53</sup> and may relieve cramps by increasing the inhibition produced by the Golgi tendon organs<sup>4,76</sup> or the physical separation of contractile proteins (or both).<sup>53</sup> The effectiveness of chronic or acute static stretching as prophylaxis against future EAMC episodes is unknown. Those patients with EAMCs related to a whole-body sodium deficit must be promptly treated with a high-salt solution, either orally or intravenously. However, it is important to note that this treatment will not result in immediate relief of muscle cramping because of the time necessary to properly absorb sodium.

No well-designed, controlled cohort or experimental studies have compared the effectiveness of EAMC prophylactics, yet some case studies suggest that fluid and electrolyte monitoring and replacement<sup>9</sup> or neuromuscular reeducation<sup>49</sup> may effectively reduce EAMC recurrence.

### Hyperthermia and EHS

Signs and symptoms of hyperthermia include dizziness, confusion, behavioral changes, coordination difficulties, decreased cognitive function, reduced physical performance, and collapse.<sup>15,52,69,71,72,81,82</sup> The residual effects of elevated core body temperature depend on the duration, not necessarily the degree, of the hyperthermia.<sup>46,57,69,82</sup> Moderate exercise-induced hyperthermia is normal and even protective in that it triggers the body's thermoregulatory system. However, with EHS (core body temperature greater than 40.5°C [105°F]), long-term neurologic deficits are possible if the condition is not quickly recognized and treated. When EHS is immediately treated via rapid whole-body cooling and core body temperature is normalized within 30 minutes of collapse, a 100% survival rate with limited or no sequelae has been reported.<sup>57,69</sup>

The fastest way to decrease core body temperature is full-body CWI in a pool or tub (with a water temperature between 1°C [35°F] and 15°C [59°F]).<sup>57,61,69</sup> Timely (less than 30 minutes from the time of collapse) CWI therapy was associated with a 0% fatality rate in more than 2000

**Table 5. Example of Wet-Bulb Globe Temperature (WBGT) Guidelines<sup>a</sup>**

WBGT Reading	Activity Guidelines and Rest-Break Guidelines
Under 82.0°F (27.8°C)	Normal activities: provide ≥3 separate rest breaks/h of minimum duration 3 min each during workout.
82.0–86.9°F (27.8°C–30.5°C)	Use discretion for intense or prolonged exercise. Watch at-risk players carefully. Provide ≥3 separate rest breaks/h of minimum duration 4 min each.
87.0°F–89.9°F (30.5°C–32.2°C)	Maximum practice time = 2 h. For football: players restricted to helmet, shoulder pads, and shorts during practice. All protective equipment must be removed for conditioning activities. For all sports: provide ≥4 separate rest breaks/h of minimum duration 4 min each.
90.0–92.0°F (32.2°C–33.3°C)	Maximum length of practice = 1 h. No protective equipment may be worn during practice and there may be no conditioning activities. There must be 20 min of rest breaks provided during the hour of practice.
Over 92.1°F (33.4°C)	No outdoor workouts, cancel exercise, delay practices until a cooler WBGT reading occurs.

**Guidelines for hydration and rest breaks**

1. Rest time should involve both unlimited hydration intake (water or electrolyte drinks) and rest without any activity involved.
2. For football, helmets should be removed during rest time.
3. The site of the rest time should be a “cooling zone” and not in direct sunlight.
4. When the WBGT reading is greater than 86°F (30°C):
  - a. Ice towels and spray bottles filled with ice water should be available at the “cooling zone” to aid the cooling process.
  - b. Cold-immersion tubs must be available for practices for the benefit of any player showing early signs of heat illness.

**Definitions**

1. Practice: The period of time that a participant engages in a coach-supervised, school-approved sport or conditioning-related activity. Practices are timed from the time the players report to the field until they leave the field.
2. Walk-through: This period of time shall last no more than 1 h, is not considered to be a part of the practice-time regulation, and may not involve conditioning or weight-room activities. Players may not wear protective equipment.

<sup>a</sup> Example originates from Georgia High School Athletics Association wet-bulb globe temperature guidelines and is only applicable to those who practice, condition, train, or compete under similar environmental conditions. Guidelines should be region specific and based on the following criteria: (1) environmental conditions, (2) intensity of activity, (3) heat-acclimatization status, (4) equipment and clothing, (5) fitness of individual, and (6) age of participants.

EHS patients in athletics and military settings. Other forms of cooling (eg, cold-water dousing with fans, ice-water towels) may be used if CWI is not available, but these methods decrease core body temperature at a slower rate than does CWI.<sup>61,62,69</sup>

**Environmental Risk Factors**

**Environmental Conditions.** Hot and humid environmental conditions can more readily predispose an individual to EHS.\* When the environmental temperature is higher than the body’s skin temperature, individuals absorb heat from the environment, and their heat loss depends entirely on evaporation.<sup>17,30,71,72</sup> Yet when humidity is also high, evaporative heat loss is severely diminished, which can lead to a rapid rise in core body temperature and an extreme risk for EHS (Table 5).

The environmental factors that influence the risk of heat illness include the ambient temperature, relative humidity (amount of water vapor in the air), air motion (wind speed), and amount of radiant heat from the sun. The relative risk of heat illness can be calculated using the WBGT equation:

$$\begin{aligned} \text{WBGT} = & (\text{wet-bulb temperature} \times 0.7) \\ & + (\text{black-globe temperature} \times 0.2) \\ & + (\text{dry-bulb temperature} \times 0.1). \end{aligned} \quad (2)$$

This equation is used to estimate the risk associated with exercise based on environmental conditions and can be useful for setting local policies regarding environmental heat. The WBGT index has long been used in athletics and by the US military. Using the WBGT index to modify activity in high-risk settings has greatly diminished the occurrence of EHS cases in US Marine Corps recruits. However, due to geographical differences among athletic

teams and schools across the United States, the WBGT index may not be the most appropriate tool in determining a universal policy for activity modifications and cancellations.<sup>84</sup> Therefore, caution is necessary when setting protocols based solely on climate due to differences among the various regions of the country. It should be noted that an EHI could occur in seemingly “normal” environmental conditions and, therefore, all appropriate precautions should be taken, especially in the first week of practice (Table 6).

**Barriers to Evaporative Heat Loss.** Athletic equipment and rubber or plastic suits used for weight loss do not allow water vapor to pass from the skin to the environment and, as a result, inhibit evaporative, convective, and radiant heat loss.<sup>27,42,86,87</sup> Participants who wear equipment that does not allow for heat dissipation are at an increased risk for heat illness. Wearing a helmet is also a risk factor because a significant amount of heat is dissipated through the head. Individuals are most susceptible to EHI during the first week of preseason practices.<sup>29,88,89</sup> Thus, it is important to include a phase-in of equipment as part of the heat-acclimatization period.

**Wet-Bulb Globe Temperature the Previous Day and Night.** When individuals compete in high WBGT conditions, the risk of EHI increases the following day.<sup>91</sup> This factor appears to be one of the best predictors of EHI and should be considered when planning successive practice sessions. Additionally, individuals who sleep in warm or non-air-conditioned quarters are also at greater risk due to the cumulative effects of heat exposure.

**Excessive Clothing or Equipment.** Excessive clothing or equipment decreases the body’s ability to thermoregulate and may cause greater absorption of radiant heat from the environment.

\* References 17, 22, 23, 30, 33, 71, 72, 83, 85

**Table 6. Risk Factors for Exertional Heat Stroke**<sup>69,90</sup>

Extrinsic Risk Factors	Intrinsic Risk Factors
High ambient temperature, solar radiation, and high humidity	High intensity of exercise and/or poor physical conditioning
Athletic gear or uniforms	Sleep loss
Peer or organizational pressure	Dehydration or inadequate water intake
Inappropriate work-to-rest ratios based on intensity, wet-bulb globe temperature, clothing, equipment, fitness, and athlete's medical condition	Use of diuretics or certain medications (ie, antihistamines, diuretics, antihypertensives, attention-deficit hyperactive disorder drugs)
Predisposing medical conditions	Overzealousness or reluctance to report problems, issues, or illnesses
Lack of education and awareness of heat illnesses among coaches, athletes, and medical staff	Inadequate heat acclimatization
No emergency plan to identify and treat exertional heat illnesses	High muscle mass-to-body fat ratio
Minimal access to fluids before and during practice and rest breaks	Presence of a fever
Delay in recognition of early warning signs	Skin disorder

### Nonenvironmental Risk Factors

**Heat Acclimatization.** Heat acclimatization is a physiologic response to repeated heat exposure during exercise over the course of 10 to 14 days.<sup>24,25,92</sup> This response enables the body to cope more effectively with thermal stressors and consists of increases in stroke volume and sweat rate and decreases in heart rate, core body temperature, skin temperature, and sweat salt losses.<sup>17,93,94</sup> Athletes should be allowed to acclimatize to the heat sufficiently before stressful conditions such as full equipment, multiple practices within a day, or performance trials are implemented.<sup>16,23,26,27,88</sup> Individual differences affect the onset and decay of heat acclimatization.<sup>24,25</sup> The rate of acclimatization is related to aerobic conditioning and fitness; in general, a better conditioned athlete will acclimatize to the heat more quickly.

**Exercise Intensity.** The rate of metabolic heat production is clearly a function of the intensity of physical exertion. The relative intensity of exercise, which is based in part on individual physical fitness, has the greatest influence on the rate of increase in core body temperature.<sup>94</sup> From a physiologic standpoint, high-intensity exercise results in a substantial amount of metabolic heat production, which then produces a rapid rise in core body temperature.<sup>95-97</sup> This rapid rise in temperature often exceeds the ability of the body to dissipate heat, ultimately overwhelming the thermoregulatory system. From a behavioral standpoint, individuals will often use an anticipatory defense mechanism and behavioral modifications (eg, slowing their pace) to protect themselves against dangerous levels of hyperthermia.<sup>98,99</sup> However, during competition, the will to win or to accomplish a personal best may trump this internal cue. In addition, external pressure from coaches or teammates may force athletes to ignore this protective instinct.<sup>54,88</sup>

**Overzealousness.** Overzealous athletes are at higher risk for EHI because they tend to override the normal behavioral adaptations to heat and ignore early warning signs of EHI.<sup>42,88</sup>

**Poor Physical Condition.** Untrained individuals are more susceptible to EHI than trained individuals because, as aerobic power ( $\dot{V}O_2\text{max}$ ) improves, the ability to withstand heat stress generally also improves.<sup>42,44-46</sup> High-intensity exercise can readily produce 1000 kcal/h and elevate the core temperature of at-risk athletes (those who are unfit, overweight, or unacclimatized) to a dangerous level in less than 30 minutes.<sup>94</sup>

**Increased Body Mass Index.** Obese people are at increased risk for EHI because they are less efficient in dissipating heat and produce more metabolic heat during exercise. Conversely, those who are muscle bound produce increased metabolic heat and have a lower ratio of surface area to mass, contributing to a decreased ability to dissipate heat.<sup>42,100</sup>

**Dehydration.** Excess sweat loss, inadequate fluid intake, vomiting, diarrhea, certain medications, and alcohol can lead to a measurable fluid deficit. Proper hydration can help to reduce exercise heart rate,<sup>15,34,101-103</sup> fatigue,<sup>12,104</sup> and core body temperature,<sup>105,106</sup> while improving performance<sup>105-107</sup> and cognitive functioning.<sup>81,108-111</sup> Dehydration of as little as 2% of body weight can negatively affect performance and thermoregulation.<sup>32,34</sup> Caution should be taken to ensure that athletes arrive at practice euhydrated (ie, having reestablished their weight since the last practice) and replace body water that is lost during practice. Measuring body-weight change before, during, and after a practice or an event and across successive days is the preferred method for monitoring dehydration in the field. Using a clinical refractometer is another effective method of estimating hydration status: specific gravity should be no more than 1.020 at the start of the activity.<sup>16,31,32,42</sup> Hydration status can also be identified by monitoring the first-void morning urine color via a urine color chart (urine color should be no more than 4).<sup>31,42</sup>

Water loss that is not sufficiently regained by the next practice increases the risk for EHI.<sup>11,27,31,32</sup> Cumulative dehydration develops insidiously over several days and is typically observed during the first few days of preseason practices<sup>112</sup> and in tournament competition. Cumulative dehydration can be detected by monitoring daily prepractice and postpractice body weights and morning urine color. During intense exercise in the heat, sweat rates can be as high as 2 L/h; if the fluid is not replaced, large deficits will result.<sup>27</sup> Therefore, the rehydration rate may have to be increased during exercise periods of this nature in order to minimize fluid deficits.

**Illness.** Individuals who are currently or were recently ill may be at increased risk for EHI because of fever, dehydration, or medications (eg, decongestants or anti-diarrheal agents).<sup>27,42</sup>

**History of Exertional Heat Illness.** Athletes with a history of heat illness are often at greater risk for recurrent heat illness during strenuous physical activity due to the potential for widespread debilitation involving the thermoregulatory, central nervous, cardiovascular,

musculoskeletal, renal, and hepatic systems.<sup>20,33,42,65–68</sup> However, these long-term effects are markedly reduced if proper treatment is initiated within 10 minutes of collapse.<sup>57</sup> Identifying the cause of the heat illness and making appropriate decisions to correct the cause will decrease the risk of subsequent heat illnesses.<sup>66</sup> Therefore, the clinician's thorough understanding of the common causes and predisposing factors of EHI is extremely important. Addressing these common causes and implementing proper strategies to mitigate their harmful effects may be the most important approach in avoiding EHIs.

**Medications and Drugs.** Individuals who take certain medications or drugs, particularly those with a dehydrating effect or those that increase metabolic rate, are at increased risk for EHI.<sup>113–116</sup> Medications that have been suggested to have an adverse effect on thermoregulation include stimulants, antihistamines, anticholinergics, and antipsychotics.<sup>116</sup> Approximately one-third of high school football players reportedly used dietary supplements, most for the purpose of increasing muscle mass.<sup>113</sup> Although such substances do not preclude participation, clinicians should recognize that these athletes are at higher risk and ensure adherence to acclimatization and hydration strategies and observe and intervene to protect if the athlete appears to be struggling.

**Electrolyte Imbalance.** Electrolyte imbalances can occur even in trained, acclimatized individuals who engage in regular physical activity and eat a normal diet. Most sodium and chloride losses occur through the urine, but people with high sweat rates (eg, >2 L/h) and sodium concentrations and those who are not heat acclimatized can lose significant amounts of sodium during physical activity. It is important to emphasize that athletes' meals should replace electrolyte losses and thereby allow them to avoid salt-depletion dehydration. Electrolyte imbalances also commonly arise with the use of diuretics.<sup>117,118</sup>

### Hospitalization and Recovery

After an episode of EHS, the patient may experience impaired thermoregulation, persistent CNS dysfunction, hepatic insufficiency, and renal insufficiency.<sup>65–67,119</sup> For persons with EHS and associated multisystem tissue damage, the rate of recovery is highly individualized, ranging up to more than 1 year.<sup>65,119–121</sup> However, evidence indicates that the degree of morbidity is negatively associated with proper recognition and treatment.<sup>69</sup> If EHS is quickly recognized and immediately treated, morbidity and mortality are significantly decreased.

A patient who experiences EHS may have compromised heat tolerance and heat acclimatization even after physician clearance. Additional heat stress may reduce the patient's ability to train and compete due to impaired cardiovascular and thermoregulatory responses.<sup>65,67</sup> After recovery from EHS, an athlete's physical activity should be restricted and the gradual return to sport individualized by a physician. The patient should be monitored daily by the clinician during exercise. During the return to exercise, the patient may experience some detraining and deconditioning not directly related to the EHS. He or she should be evaluated over time to determine if there has been complete recovery of exercise and heat tolerance.

Certified athletic trainers and other allied health care providers must be able to differentiate EAMCs, heat syncope, heat exhaustion, exertional heat injury, and EHS in order to treat these conditions appropriately in athletes. This position statement outlines the NATA's current recommendations to reduce the incidence, improve the recognition, and optimize the treatment of these heat illnesses in athletes. Education and increased awareness will help to reduce both the frequency and severity of heat illnesses in athletes.

### ACKNOWLEDGMENTS

We gratefully acknowledge the efforts of William M. Adams, MS, ATC; Chad Asplund, MD, MPH; Michele Benz, MS, LAT, ATC, CSCS; Yuri Hosokawa, MAT, ATC; Glen P. Kenny, PhD; Brendon P. McDermott, PhD, ATC, and the Pronouncements Committee in the review of this document.

### DISCLAIMER

The NATA and NATA Foundation publish position statements as a service to promote the awareness of certain issues to members. The information contained in the position statement is neither exhaustive nor exclusive to all circumstances or individuals. Variables such as institutional human resource guidelines, state or federal statutes, rules, or regulations, as well as regional environmental conditions, may impact the relevance and implementation of these recommendations. The NATA and NATA Foundation advise members and others to carefully and independently consider each of the recommendations (including the applicability of same to any particular circumstance or individual). The position statement should not be relied upon as an independent basis for care but rather as a resource available to NATA members or others. Moreover, no opinion is expressed herein regarding the quality of care that adheres to or differs from the NATA and NATA Foundation position statements. The NATA and NATA Foundation reserve the right to rescind or modify its position statements at any time.

### REFERENCES

1. Binkley HM, Beckett J, Casa DJ, Kleiner DM, Plummer PE. National Athletic Trainers' position statement: exertional heat illnesses. *J Athl Train*. 2002;37(3):329–343.
2. Casa DJ, Armstrong LE, Carter R, Lopez R, McDermott B, Scriber K. Historical perspectives on medical care for heat stroke, part 1: ancient times through the nineteenth century: a review of the literature. *Athl Train Sports Health Care*. 2010;2(3):132–138.
3. Casa DJ, Armstrong LE, Carter R, Lopez R, McDermott B, Scriber K. Historical perspectives on medical care for heat stroke, part 2: 1850 through the present: a review of the literature. *Athl Train Sports Health Care*. 2010;2(4):178–190.
4. Schweltnus M. Cause of exercise associated muscle cramps (EAMC)—altered neuromuscular control, dehydration or electrolyte depletion? *Br J Sports Med*. 2009;43(6):401–408.
5. Bergeron MF. Muscle cramps during exercise—is it fatigue or electrolyte deficit? *Curr Sports Med Rep*. 2008;7(4):S50–S55.
6. Maughan R. Exercise-induced muscle cramp: a prospective biochemical study in marathon runners. *J Sports Sci*. 1986;4(1):31–34.
7. Schweltnus M, Drew N, Collins M. Increased running speed and previous cramps rather than dehydration or serum sodium changes predict exercise-associated muscle cramping: a prospective cohort

- study in 210 Ironman triathletes. *Br J Sports Med.* 2011;45(8):650–656.
8. Schweltnus M, Nicol J, Laubscher R, Noakes T. Serum electrolyte concentrations and hydration status are not associated with exercise associated muscle cramping (EAMC) in distance runners. *Br J Sports Med.* 2004;38(4):488–492.
  9. Bergeron M. Heat cramps: fluid and electrolyte challenges during tennis in the heat. *J Sci Med Sport.* 2003;6(1):19–27.
  10. Parisi L, Pierelli F, Amabile G, et al. Muscular cramps: proposals for a new classification. *Acta Neurol Scand.* 2003;107(3):176–186.
  11. Stofan JR, Zachwieja JJ, Horswill CA, Murray R, Anderson SA, Eichner ER. Sweat and sodium losses in NCAA football players: a precursor to heat cramps? *Int J Sport Nutr Exerc Metab.* 2005;15(6):641–652.
  12. Carter R, Chevront SN, Vernieuw CR, Sawka MN. Hypohydration and prior heat stress exacerbates decreases in cerebral blood flow velocity during standing. *J Appl Physiol (1985).* 2006;101(6):1744–1750.
  13. Poh PY, Armstrong LE, Casa DJ, et al. Orthostatic hypotension after 10 days of exercise-heat acclimation and 28 hours of sleep loss. *Aviat Space Environ Med.* 2012;83(4):403–411.
  14. Nybo L, Rasmussen P, Sawka MN. Performance in the heat-physiological factors of importance for hyperthermia-induced fatigue. *Compr Physiol.* 2014;4(2):657–689.
  15. Kenefick RW, Sawka MN. Heat exhaustion and dehydration as causes of marathon collapse. *Sports Med.* 2007;37(4–5):378–381.
  16. Armstrong LE, Casa DJ, Millard-Stafford M, Moran DS, Pyne SW, Roberts WO. American College of Sports Medicine position stand: exertional heat illness during training and competition. *Med Sci Sports Exerc.* 2007;39(3):556–572.
  17. Sawka MN, Leon LR, Montain SJ, Sonna LA. Integrated physiological mechanisms of exercise performance, adaptation, and maladaptation to heat stress. *Compr Physiol.* 2011;1(4):1883–1928.
  18. Winkenwerder W, Sawka MN. Disorders due to heat and cold. In: Goldman L, Schafer AI, eds. *Goldman-Cecil Medicine.* 24th ed. Philadelphia, PA: Elsevier, Inc; 2011:666–670.
  19. Casa DJ, Armstrong LE, Ganio MS, Yeargin S. Exertional heat stroke in competitive athletes. *Curr Sports Med Rep.* 2005;4(6):309–317.
  20. Adams WM, Hosokawa Y, Casa DJ. The timing of exertional heat stroke survival starts prior to collapse. *Med Sci Sports Exerc.* 2015;47(4):273–274.
  21. Ebell MH, Siwek J, Weiss BD, et al. Strength of recommendation taxonomy (SORT): a patient-centered approach to grading evidence in the medical literature. *Am Fam Physician.* 2004;69(3):548–556.
  22. Casa DJ, Anderson SA, Baker L, et al. The Inter-Association Task Force for Preventing Sudden Death in Collegiate Conditioning Sessions: best practices recommendations. *J Athl Train.* 2012;47(4):477–480.
  23. Casa DJ, Almquist J, Anderson SA, et al. The Inter-Association Task Force for Preventing Sudden Death in Secondary School Athletics Programs: best-practices recommendations. *J Athl Train.* 2013;48(4):546–553.
  24. Armstrong LE, Maresh CM. The induction and decay of heat acclimatization in trained athletes. *Sports Med.* 1991;12(5):302–312.
  25. Pandolf KB. Time course of heat acclimation and its decay. *Int J Sports Med.* 1998;19(suppl 2):S157–S160.
  26. Casa DJ, Csillan D, Armstrong LE, et al. Preseason heat-acclimatization guidelines for secondary school athletics. *J Athl Train.* 2009;44(3):332–333.
  27. Bergeron MF, McKeag DB, Casa DJ, et al. Youth football: heat stress and injury risk. *Med Sci Sports Exerc.* 2005;37(8):1421–1430.
  28. Yeargin SW, Casa DJ, Armstrong LE, et al. Heat acclimatization and hydration status of American football players during initial summer workouts. *J Strength Cond Res.* 2006;20(3):463–470.
  29. Kerr ZY, Casa DJ, Marshall SW, Comstock D. Epidemiology of exertional heat illness among U.S. high school athletes. *Am J Prev Med.* 2013;44(1):8–14.
  30. Kenny GP, Journeay WS. Human thermoregulation: separating thermal and nonthermal effects on heat loss. *Front Biosci (Landmark Ed).* 2010;15:259–290.
  31. Casa DJ, Armstrong LE, Hillman SK, et al. National Athletic Trainers' Association position statement: fluid replacement for athletes. *J Athl Train.* 2000;35(2):212–224.
  32. Casa DJ, Clarkson PM, Roberts WO. American College of Sports Medicine roundtable on hydration and physical activity: consensus statements. *Curr Sports Med Rep.* 2005;4(3):115–127.
  33. Casa DJ, Almquist J, Anderson S. Inter-Association Task Force on Exertional Heat Illnesses consensus statement. *NATA News.* June;2003:24–29.
  34. Chevront SN, Kenefick RW, Montain SJ, Sawka MN. Mechanisms of aerobic performance impairment with heat stress and dehydration. *J Appl Physiol (1985).* 2010;109(6):1989–1995.
  35. Courson R. Preventing sudden death on the athletic field: the emergency action plan. *Curr Sports Med Rep.* 2007;6(2):93–100.
  36. Anderson J, Courson RW, Kleiner DM, McLoda TA. National Athletic Trainers' Association position statement: emergency planning in athletics. *J Athl Train.* 2002;37(1):99–104.
  37. Casa DJ, Guskiewicz KM, Anderson SA, et al. National Athletic Trainers' Association position statement: preventing sudden death in sports. *J Athl Train.* 2012;47(1):96–118.
  38. Casa DJ, Becker SM, Ganio MS, et al. Validity of devices that assess body temperature during outdoor exercise in the heat. *J Athl Train.* 2007;42(3):333–342.
  39. Gagnon D, Lemire BB, Jay O, Kenny GP. Aural canal, esophageal, and rectal temperatures during exertional heat stress and the subsequent recovery period. *J Athl Train.* 2010;45(2):157–163.
  40. Huggins R, Glaviano N, Negishi N, Casa DJ, Hertel J. Comparison of rectal and aural core body temperature thermometry in hyperthermic, exercising individuals: a meta-analysis. *J Athl Train.* 2012;47(3):329–338.
  41. Ronneberg K, Roberts WO, McBean AD, Center BA. Temporal artery temperature measurements do not detect hyperthermic marathon runners. *Med Sci Sports Exerc.* 2008;40(8):1373–1375.
  42. Cleary M. Predisposing risk factors on susceptibility to exertional heat illness: clinical decision-making considerations. *J Sport Rehabil.* 2007;16(3):204–214.
  43. Shibolet S, Coll R, Gilat T, Sohar E. Heatstroke: its clinical picture and mechanism in 36 cases. *Q J Med.* 1967;36(144):525–548.
  44. Wallace RF, Kriebel D, Punnett L, et al. Risk factors for recruit exertional heat illness by gender and training period. *Aviat Space Environ Med.* 2006;77(4):415–421.
  45. Rav-Acha M, Hadad E, Epstein Y, Heled Y, Moran DS. Fatal exertional heat stroke: a case series. *Am J Med Sci.* 2004;328(2):84–87.
  46. Epstein Y, Moran DS, Shapiro Y, Sohar E, Shemer J. Exertional heat stroke: a case series. *Med Sci Sports Exerc.* 1999;31(2):224–228.
  47. Green JM, Yang Z, Laurent CM, et al. Session RPE following interval and constant-resistance cycling in hot and cool environments. *Med Sci Sports Exerc.* 2007;39(11):2051–2057.
  48. Roelands B, Meeusen R. Caffeine, dopamine and thermoregulation. *Eur J Appl Physiol.* 2012;112(5):1979–1980.
  49. Wagner T, Behnia N, Ancheta W, Shen R, Farrokhi S, Powers C. Strengthening and neuromuscular reeducation of the gluteus maximus in a triathlete with exercise-associated cramping of the hamstrings. *J Orthop Sports Phys Ther.* 2010;40(2):112–119.

50. Maquirriain J, Merello M. The athlete with muscular cramps: clinical approach. *J Am Acad Orthop Surg.* 2007;15(7):425–431.
51. Shang G, Collins M, Schweltnus M. Factors associated with a self-reported history of exercise-associated muscle cramps in Ironman triathletes: a case-control study. *Clin J Sport Med.* 2011;21(3):204–210.
52. Casa DJ. *Preventing Sudden Death in Sport and Physical Activity.* Sudbury, MA: Jones and Bartlett Learning; 2012.
53. Bertolasi L, De Grandis D, Bongiovanni L, Zanette G, Gasperini M. The influence of muscular lengthening on cramps. *Ann Neurol.* 1993;33(2):176–180.
54. Allen S, Miller K, Albrecht J, Garden-Robinson J, Blodgett-Salafia E. Ad libitum fluid intake and plasma responses following pickle juice, hypertonic saline, and deionized water ingestion. *J Athl Train.* 2013;48(6):734–740.
55. Miller KC. Electrolyte and plasma responses after pickle juice, mustard, and deionized water ingestion in dehydrated humans. *J Athl Train.* 2014;49(3):360–367.
56. Armstrong LE, Lopez RM. Return to exercise training after heat exhaustion. *J Sport Rehabil.* 2007;16(3):182–189.
57. Casa DJ, McDermott BP, Lee EC, Yeargin SW, Armstrong LE, Maresh CM. Cold water immersion: the gold standard for exertional heatstroke treatment. *Exerc Sport Sci Rev.* 2007;35(3):141–149.
58. Casa DJ, Kenny GP, Taylor NA. Immersion treatment for exertional hyperthermia: cold or temperate water? *Med Sci Sports Exerc.* 2010;42(7):1246–1252.
59. Miller KC, Swartz EE, Long BC. Cold-water immersion for hyperthermic humans wearing American football uniforms. *J Athl Train.* 2015;50(8):792–799.
60. Gagnon D, Lemire BB, Casa DJ, Kenny GP. Cold-water immersion and the treatment of hyperthermia: using 38.6°C as a safe rectal temperature cooling limit. *J Athl Train.* 2010;45(5):439–444.
61. Costrini A. Emergency treatment of exertional heatstroke and comparison of whole body cooling techniques. *Med Sci Sports Exerc.* 1990;22(1):15–18.
62. McDermott BP, Casa DJ, O'Connor FG. Cold-water dousing with ice massage to treat exertional heat stroke: a case series. *Aviat Space Environ Med.* 2009;80(8):720–722.
63. DeMartini JK, Casa DJ, Stearns R, et al. Effectiveness of cold water immersion in the treatment of exertional heat stroke at the Falmouth Road Race. *Med Sci Sports Exerc.* 2015;47(2):240–245.
64. Pryor RR, Casa DJ, Holschen JC, O'Connor FG, Vandermark LW. Exertional heat stroke: strategies for prevention and treatment from the sports field to the emergency department. *Clin Pediatr Emerg Med.* 2013;14(4):267–278.
65. O'Connor FG, Casa DJ, Bergeron MF, et al. American College of Sports Medicine roundtable on exertional heat stroke – return to duty/return to play: conference proceedings. *Curr Sports Med Rep.* 2010;9(5):314–321.
66. McDermott BP, Casa DJ, Yeargin SW, Ganio MS, Armstrong LE, Maresh CM. Recovery and return to activity following exertional heat stroke: considerations for the sports medicine staff. *J Sport Rehabil.* 2007;16(3):163–181.
67. O'Connor FG, Williams AD, Blivin S, Heled Y, Deuster P, Flinn SD. Guidelines for return to duty (play) after heat illness: a military perspective. *J Sport Rehabil.* 2007;16(3):227–237.
68. Moran DS, Heled Y, Still L, Laor A, Shapiro Y. Assessment of heat tolerance for post exertional heat stroke individuals. *Med Sci Monit.* 2004;10(6):CR252–CR257.
69. Casa DJ, Armstrong LE, Kenny GP, O'Connor FG, Huggins RA. Exertional heat stroke: new concepts regarding cause and care. *Curr Sports Med Rep.* 2012;11(3):115–123.
70. Sawka MN, Castellani JW, Cheuvront SN, Young AJ. Physiological systems and their responses to conditions of heat and cold. In: Farrell PA, Joyner MJ, Caiozzo VJ, eds. *ACSM's Advanced Exercise Physiology.* 2nd ed. Philadelphia, PA: Lippincott, Williams & Wilkins; 2012:567–602.
71. Epstein Y, Roberts WO. The pathophysiology of heat stroke: an integrative view of the final common pathway. *Scand J Med Sci Sports.* 2011;21(6):742–748.
72. Armstrong LE. *Exertional Heat Illnesses.* Champaign, IL: Human Kinetics; 2003.
73. Santee W, Gonzalez R. Characteristics of the thermal environment. In: Pandolf KB, Sawka MN, Gonzalez RR, eds. *Human Performance Physiology and Environmental Medicine at Terrestrial Extremes.* Indianapolis, IN: Benchmark Press; 1988:1–44.
74. Cooper E, Ferrara M, Broglio S. Exertional heat illness and environmental conditions during a single football season in the southeast. *J Athl Train.* 2006;41(3):332–336.
75. Schweltnus M, Allie S, Derman W, Collins M. Increased running speed and pre-race muscle damage as risk factors for exercise-associated muscle cramps in a 56 km ultra-marathon: a prospective cohort study. *Br J Sports Med.* 2011;45(14):1132–1136.
76. Khan S, Burne J. Reflex inhibition of normal cramp following electrical stimulation of the muscle tendon. *J Neurophysiol.* 2007;98(3):1102–1107.
77. Jung A, Bishop P, Al-Nawwas A, Dale R. Influence of hydration and electrolyte supplementation on incidence and time to onset of exercise-associated muscle cramps. *J Athl Train.* 2005;40(2):71–75.
78. Müller K, Mack G, Knight K, et al. Three percent hypohydration does not affect the threshold frequency of electrically induced cramps. *Med Sci Sports Exerc.* 2010;42(11):2056–2063.
79. Braulick K, Miller K, Albrecht J, Tucker J, Deal J. Significant and serious dehydration does not affect skeletal muscle cramp threshold frequency. *Br J Sports Med.* 2013;47(11):710–714.
80. Summers K, Snodgrass S, Callister R. Predictors of calf cramping in rugby league. *J Strength Cond Res.* 2014;28(3):774–783.
81. Lieberman HR, Bathalon GP, Falco CM, Kramer FM, Morgan CA, Niro P. Severe decrements in cognition function and mood induced by sleep loss, heat, dehydration, and undernutrition during simulated combat. *Biol Psychiatry.* 2005;57(4):422–429.
82. Bouchama A, Knochel JP. Heat stroke. *N Engl J Med.* 2002;346(25):1978–1988.
83. DeMartini JK, Casa DJ, Belval L, et al. Environmental conditions and the occurrence of exertional heat illnesses and exertional heat stroke at the Falmouth Road Race. *J Athl Train.* 2014;49(4):478–485.
84. Roberts WO. Determining a “do not start” temperature for a marathon on the basis of adverse outcomes. *Med Sci Sports Exerc.* 2010;42(2):226–232.
85. Bergeron MF, Bahr R, Bartsch P, et al. International Olympic Committee consensus statement on thermoregulatory and altitude challenges for high-level athletes. *Br J Sports Med.* 2012;46(11):770–779.
86. Armstrong LE, Johnson EC, Casa DJ, et al. The American football uniform: uncompensable heat stress and hyperthermic exhaustion. *J Athl Train.* 2010;45(2):117–127.
87. Kulka TJ, Kenney WL. Heat balance limits in football uniforms how different uniform ensembles alter the equation. *Phys Sportsmed.* 2002;30(7):29–39.
88. Lopez RM, Casa DJ, McDermott BP, Stearns RL, Armstrong LE, Maresh CM. Exertional heat stroke in the athletic setting: a review of the literature. *Athl Train Sports Health Care.* 2011;3(4):189–200.
89. Grundstein AJ, Ramseyer C, Zhao F, et al. A retrospective analysis of American football hyperthermia deaths in the United States. *Int J Biometeorol.* 2012;56(1):11–20.
90. Tokizawa K, Sawada S, Tai T, et al. Effects of partial sleep restriction and subsequent daytime napping on prolonged exertional heat strain. *Occup Environ Med.* 2015;72(7):521–528.

91. Wallace RF, Kriebel D, Punnett L, et al. The effects of continuous hot weather training on risk of exertional heat illness. *Med Sci Sports Exerc.* 2005;37(1):84–90.
92. Armstrong LE, Stoppani J. Central nervous system control of heat acclimation adaptations: an emerging paradigm. *Rev Neurosci.* 2002;13(3):271–285.
93. Lorenzo S, Halliwill JR, Sawka MN, Minson CT. Heat acclimation improves exercise performance. *J Appl Physiol (1985).* 2010;109(4):1140–1147.
94. Noakes TD, Myburgh KH, du Plessis J, et al. Metabolic rate, not percent dehydration, predicts rectal temperature in marathon runners. *Med Sci Sports Exerc.* 1991;23(4):443–449.
95. Mora-Rodriguez R, Del Coso J, Estevez E. Thermoregulatory responses to constant versus variable-intensity exercise in the heat. *Med Sci Sports Exerc.* 2008;40(11):1945–1952.
96. Kenny GP, Niedre PC. The effect of exercise intensity on the post-exercise esophageal temperature response. *Eur J Appl Physiol.* 2002;86(4):342–346.
97. Fowkes Godek S, Godek JJ, Bartolozzi AR. Thermal responses in football and cross-country athletes during their respective practices in a hot environment. *J Athl Train.* 2004;39(3):235–240.
98. Tucker R, Noakes T. The physiological regulation of pacing strategy during exercise: a critical review. *Br J Sports Med.* 2009;43(6):E1.
99. Marino F. Anticipatory regulation and avoidance of catastrophe during exercise-induced hyperthermia. *Comp Biochem Physiol B Biochem Mol Biol.* 2004;139(4):561–569.
100. Chung NK, Pin CH. Obesity and the occurrence of heat disorders. *Mil Med.* 1996;161(12):739–742.
101. Montain SJ, Sawka MN, Latzka WA, Valeri CR. Thermal and cardiovascular strain from hypohydration: influence of exercise intensity. *Int J Sports Med.* 1998;19(2):87–91.
102. Sawka MN, Cheuvront SN, Kenefick RW. High skin temperature and hypohydration impair aerobic performance. *Exp Physiol.* 2012;97(3):327–332.
103. Adams WM, Ferraro EM, Huggins RA, Cass DJ. Influence of body mass loss on changes in heart rate during exercise in the heat: a systematic review. *J Strength Cond Res.* 2014;28(8):2380–2389.
104. Hubbard RW, Armstrong LE. The heat illnesses: biochemical, ultrastructural, and fluid-electrolyte considerations. In: Pandolf KB, et al. *Human Performance Physiology and Environmental Medicine at Terrestrial Extremes.* Indianapolis, IN: Benchmark Press; 1988:305.
105. Lopez RM, Casa DJ, Jensen KA, et al. Examining the influence of hydration status on physiological responses and running speed during trail running in the heat with controlled exercise intensity. *J Strength Cond Res.* 2011;25(11):2944–2954.
106. Casa DJ, Stearns RL, Lopez RM, et al. Influence of hydration on physiological function and performance during trail running in the heat. *J Athl Train.* 2010;45(2):147–156.
107. Stearns RL, Casa DJ, Lopez RM, et al. Influence of hydration status on pacing during trail running in the heat. *J Strength Cond Res.* 2009;23(9):2533–2541.
108. Cheuvront SN, Kenefick RW. Dehydration: physiology, assessment, and performance effects. *Compr Physiol.* 2014;4(1):257–285.
109. Lieberman HR. Hydration and cognition: a critical review and recommendations for future research. *J Am Coll Nutr.* 2007;26(suppl 5):S555–S561.
110. Ganio MS, Armstrong LE, Casa DJ, et al. Mild dehydration impairs cognitive performance and mood of men. *Br J Nutr.* 2011;106(10):1535–1543.
111. Armstrong LE, Ganio MS, Casa DJ, et al. Mild dehydration affects mood in healthy young women. *J Nutr.* 2012;142(2):382–388.
112. Godek SF, Godek JJ, Bartolozzi AR. Hydration status in college football players during consecutive days of twice-a-day preseason practices. *Am J Sports Med.* 2005;33(6):843–851.
113. Swirzenski L, Latin RW, Berg K, Grandjean A. A survey of sport nutrition supplements in high school football players. *J Strength Cond Res.* 2000;14(4):464–469.
114. Roelands B, Hasegawa H, Watson P, et al. The effects of acute dopamine reuptake inhibition on performance. *Med Sci Sports Exerc.* 2008;40(5):879–885.
115. Goekint M, Roelands B, Heyman E, Njemini R, Meeusen R. Influence of citalopram and environmental temperature on exercise-induced changes in BDNF. *Neurosci Lett.* 2011;494(2):150–154.
116. Watson P, Hasegawa H, Roelands B, Piacentini MF, Loooverie R, Meeusen R. Acute dopamine/noradrenaline reuptake inhibition enhances human exercise performance in warm, but not temperate conditions. *J Physiol.* 2005;565(pt 3):873–883.
117. Jung AP, Bishop PA, Al-Nawwas A, Dale RB. Influence of hydration and electrolyte supplementation on incidence and time to onset of exercise-associated muscle cramps. *J Athl Train.* 2005;40(2):71–75.
118. Kenefick RW, Cheuvront SN, Montain SJ, Carter R, Sawka MN. Human water and electrolyte balance. In: Brown-Bowman BA, Russell RM, eds. *Present Knowledge in Nutrition.* Washington, DC: International Life Sciences Institute; 2012:493–505.
119. Coris EE, Walz S, Konin J, Pescasio M. Return to activity considerations in a football player predisposed to exertional heat illness: a case study. *J Sport Rehabil.* 2007;16(3):260–270.
120. Carter R, Cheuvront S, Sawka M. A case report of idiosyncratic hyperthermia and review of U.S. Army heat stroke hospitalizations. *J Sport Rehabil.* 2007;16(3):238–243.
121. Carter R, Cheuvront SN, Williams JO, et al. Epidemiology of hospitalizations and deaths from heat illness in soldiers. *Med Sci Sports Exerc.* 2005;37(8):1338–1344.

---

Address correspondence to Douglas J. Casa, PhD, ATC, FNATA, FACSM, Department of Kinesiology, University of Connecticut, 2095 Hillside Road, Box U-1110, Storrs, CT 06269-1110. Address e-mail to douglas.casa@uconn.edu.

In the September 2015 issue of the *Journal of Athletic Training*, in the article,

Casa DJ, DeMartini JK, Bergeron MF, et al. National Athletic Trainers' Association position statement: exertional heat illnesses. *J Athl Train*. 2015;50(9):986–1000,

Table 4, item 9b should read as follows:

"An approximate estimate of cooling via cold-water immersion is 1°C for every 5 min and 1°F for every 3 min (if the water is aggressively stirred). For example, someone in the tub for 15 min would cool approximately 3°C or 5°F during that time."

We regret the error.