

# Exertional Heat Stroke and American Football: What the Team Physician Needs to Know

Jillian E. Sylvester, MD, Luke N. Belval, MS, ATC, CSCS, Douglas J. Casa, PhD, ATC, FNATA, FACSM, and Francis G. O'Connor, MD, MPH

## Abstract

Football is recognized as a leading contributor to sports injury secondary to the contact collision nature of the endeavor. While direct deaths from head and spine injury remain a significant contributor to the number of catastrophic injuries, indirect deaths (systemic failure) predominate. Exertional heat stroke has emerged as one of the leading indirect causes of death in high school and collegiate football. This review details for the team physician the unique challenge of exercising in the heat to the football player, and the prevention, diagnosis, management, and return-to-play issues pertinent to exertional heat illnesses.

(systemic failure) predominate. Exertional heat stroke (EHS) has emerged as one of the leading indirect causes of death in high school and collegiate football. Boden and colleagues<sup>2</sup> reported that high school and college football players sustain approximately 12 fatalities annually, with indirect systemic causes being twice as common as direct blunt trauma.<sup>2</sup> The most common indirect causes identified included cardiac failure, heat illness, and complications of sickle cell trait (SCT). It was also noted that the risk of SCT, heat-related, and cardiac deaths increased during the second decade of the study, indicating these conditions may require a greater emphasis on diagnosis, treatment, and prevention. This review details for the team physician the unique challenge of exercising in the heat to the football player, and the prevention, diagnosis, management and return-to-play issues pertinent to exertional heat illness (EHI).

Football, one of the most popular sports in the United States, is additionally recognized as a leading contributor to sports injury secondary to the contact collision nature of the endeavor. There are an estimated 1.1 million high school football players with another 100,000 participants combined in the National Football League (NFL), college, junior college, Arena Football League, and semipro levels of play.<sup>1</sup> USA Football estimates that an additional 3 million youth participate in community football leagues.<sup>1</sup> The National Center for Catastrophic Sports Injury Research recently calculated a fatality rate of 0.14 per 100,000 participants in 2014 for the 4.2 million who play football at all levels—and 0.45 per 100,000 in high school.<sup>1</sup> While direct deaths from head and spine injury remain a significant contributor to the number of catastrophic injuries, indirect deaths

## The Challenge

EHS represents the most severe manifestation of EHI—a gamut of diseases commonly encountered during the hot summer months when American football season begins. The breadth of EHI includes several important clinical diagnoses: exercise-associated muscle cramps (heat cramps); heat exhaustion with and without syncope; heat injury with evidence of end organ injury (eg, rhabdomyolysis); and EHS. EHS is defined as “a form of hyperthermia associated with a systemic inflammatory response leading to a syndrome of multi-organ dysfunction in which encephalopathy predominates.”<sup>3</sup> EHS, if left untreated, or even if clinical treatment is delayed, may result in significant end organ morbidity and/or mortality.

During exercise, the human thermoregulatory system mitigates heat gain by increasing skin blood flow and sweating, causing an increased dissipation

**Authors' Disclosure Statement:** The authors report no actual or potential conflict of interest in relation to this article. The opinions and assertions contained herein are private views and are not to be construed as official or as reflecting the views of the US Army Medical Department, US Air Force Medical Corps, Uniformed Services University, or the Department of Defense at large.

of heat to the surrounding environment by leveraging conduction, convection, and evaporation.<sup>4,5</sup> Elevated environmental temperatures, increased humidity, and dehydration can impede the body's ability to dissipate heat at a rate needed to maintain thermoregulation. This imbalance can result in hyperthermia secondary to uncompensated heat stress,<sup>5</sup> which in turn can lead to EHI. Football players have unique challenges that make them particularly vulnerable to EHI. The summer heat during early-season participation and the requirement for equipment that covers nearly 60% of body surfaces pose increased risk of volume losses and hyperthermia that trigger the onset of EHI.<sup>6</sup> Football athletes' body compositions and physical size are additional contributing risk factors; the relatively high muscle and fat content increase thermogenicity, which require their bodies to dissipate more heat.<sup>7</sup>

An estimated 9000 cases of EHI occur annually across all high school sports,<sup>8</sup> with an incidence

of 1.6:100,000 athlete-exposures.<sup>8,9</sup> Studies have demonstrated, however, that EHI occurs in football 11.4 times more often than in all other high school sports combined.<sup>10</sup> The incidence of nonfatal EHI in all levels of football is 4.42-5:100,000.<sup>8,9</sup> Between 2000 and 2014, 41 football players died from EHS.<sup>1</sup> In football, approximately 75% of all EHI events occurred during practices, while only 25% of incidents occurred during games.<sup>8</sup>

Given these potentially deadly consequences, it is important that football team physicians are not only alert to the early symptoms of heat illness and prepared to intervene to prevent the progression to EHS, but are critical leaders in educating coaches and players in evidence-based EHI prevention practices and policies.



**Table 1. Risk Factors For Developing Exertional Heat Illness<sup>9,10,29</sup>**

**Extrinsic Risk Factors**

- High WBGT
- Poor medical supervision and emergency medical plan
- Delay in recognition of early signs of EHI
- Use of excessive clothing, pads/equipment
- Incongruent work-to-rest ratios based on WBGT and heat acclimatization
- Lack of acclimatization

**Intrinsic Risk Factors**

- Previous episode of heat illness or exertional cramping; history of malignant hyperthermia
- Obesity or poor physical conditioning
- Hypohydration
- Sleep loss
- Skin conditions that inhibit sweating
- Genetic predisposition
- Febrile illness
- Overzealousness of athlete
- Medications/supplements
  - Antihistamines
  - Anticholinergics
  - Calcium channel blockers
  - Beta blockers
  - Diuretics
  - Capsaicin
  - Female reproductive hormones
  - Nutritional supplements
  - Illicit drugs
  - Alcohol use

Abbreviations: EHI, exertional heat illness; WBGT, wet bulb globe temperature.

**Prevention**

EHS is a preventable condition, arguably the most common cause of preventable nontraumatic exertional death in young athletes in the United States. Close attention to mitigating risk factors should begin prior to the onset of preseason practice and continue through the early season, where athletes are at the highest risk of developing heat illness.

**Primary Prevention**

Primary prevention is fundamental to minimizing the occurrences of EHI. It focuses on the following methods: recognition of inherent risk factors, acclimatization, hydration, and avoidance of inciting substances (including supplements).

*Pre-Participation Examination.* The purpose of the pre-participation examination (PPE) is to maximize an athlete's safety by identifying medical conditions that place the athlete at risk.<sup>11,12</sup> The *Preparticipation Physical Evaluation, 4th edition*, the most widely used consensus publication, specifically queries if an athlete has a previous history of heat injury. However, it only indirectly addresses intrinsic risk factors that may predispose an athlete to EHI who has never had an EHI before. Therefore, providers should take the opportunity of the PPE to inquire about additional risk factors that may make an athlete high risk for sustaining a heat injury. Common risk factors for EHI are listed in **Table 1**. While identifying at-risk athletes is important in mitigating the risk of developing EHI, it will

not identify all possible cases: a study of military recruits found that up to 50% of Marines who developed EHI lacked an identifiable risk factor.<sup>13</sup>

**Wet Bulb Globe Temperature.** Humidity can heighten a player's risk of developing thermogenic dysregulation during hot temperatures. As ambient temperature nears internal body temperature, heat may actually be absorbed by the skin rather than dissipated into the air. As a result, the body must increasingly rely on sweat evaporation to encourage heat loss; this process is hindered in very humid climates. Wet bulb globe temperature (WBGT) is a measure of heat stress that accounts for temperature, humidity, wind speed, and cloud cover. WBGT should be utilized to determine the relative risk of EHI based on local environmental conditions, as there is a direct correlation between elevated WBGT and risk of EHI.<sup>11,14</sup> The greatest risk for EHS is performing high-intensity exercise (>75% VO<sub>2</sub>max) when WBGT >28°C (82°F).<sup>7</sup> A study of hyperthermia-related deaths in football found that a majority of fatalities occurred on days classified as high risk (23°C-28°C) or extreme risk (>28°C) by WBGT.<sup>14</sup> Consensus guidelines recommend that activities be modified based on WBGT (**Table 2**).<sup>7,12</sup> The impact of WBGT does not end solely on the day of practice. Athletes who exercise in elevated WBGT environments on 2 consecutive days are at increased risk of EHI due

to cumulative effects of exercise in heat.<sup>11</sup>

**Clothing.** In football, required protective equipment may cover up to 60% of body surfaces. Studies have shown that wearing full uniform with pads increases internal body temperature and decreases time to exhaustion when compared to light clothing.<sup>5,15</sup> In addition, athletic equipment traps heat close to the body and inhibits evaporation of sweat into the environment, thereby inhibiting radiant and evaporative heat dissipation.<sup>5,11</sup> Likewise, wearing dark clothing encourages radiant absorption of heat, further contributing to potential thermal dysregulation.<sup>5</sup> Use of a helmet is a specific risk factor for EHI, as significant heat dissipation occurs through the head.<sup>11</sup> To mitigate these risk factors, the introduction of padded equipment should occur incrementally over the heat acclimatization period (see below). In addition, athletes should be encouraged to remove their helmets during rest periods to promote added heat dissipation and recovery.

**Heat Acclimatization.** The risk of EHI escalates significantly when athletes are subjected to multiple stressors during periods of heat exposure, such as sudden increases in intensity or duration of exercise; prolonged new exposures to heat; dehydration; and sleep loss.<sup>5</sup> When football season begins in late summer, athletes are least conditioned as temperatures reach their seasonal peak,

Table 2. **Activity Recommendations Based on Wet Bulb Globe Temperature**<sup>a11,13,30</sup>

Heat Category		High-Risk Athletes	Low-Risk Athletes
1	<65°F	Normal activity, no restrictions	Normal activity, no restrictions
1	65°F -72°F	Monitor athletes, encourage increased fluid intake and decreased work:rest ratio	Normal activity, no restrictions
1	72°F -78°F	Increase rest intervals, consider decreasing total practice time	Normal activity, encourage hydration
1	78°F -82°F	Increase rest intervals, consider decreasing total practice time	Normal activity, encourage hydration
2 (Green)	82°F -86°F	Work:rest ratio 1:1. Limit intensity and overall duration of practice. Monitor closely for early symptoms of EHI. At least 3 hydration/rest breaks for ≥4 minutes	Consider activity modification, limiting intense/prolonged exercise periods. At least 3 hydration/rest breaks for ≥4 minutes
3/4 (Yellow/Red)	86°F -90°F	All outdoor activities should stop	Limit practice to 2 hours. Limit periods of intense exercise; no pads during conditioning workouts Closely monitor for early symptoms of EHI
5 (Black)	>90°F	All outdoor activities should stop	All outdoor activities should stop

<sup>a</sup>Risk determined by degree of acclimatization, pre-existing risk factors outlined in Table 1. Abbreviation: EHI, exertional heat illness.

causing increased risk of EHI.<sup>15</sup> Planning for heat acclimatization is vital for all athletes who exercise in hot environments. Acclimatization procedures place progressively mounting physiologic strains on the body to improve athletes' ability to dissipate heat, diminishing thermoregulatory and cardiovascular exertion.<sup>4,5</sup> Acclimatization begins with expansion of plasma volume on days 3 to 6, causing improvements in cardiac efficiency and resulting in an overall decrease in basal internal body temperature.<sup>4,5,15</sup> This process results in improvements in heat tolerance and exercise performance, evolving over 10 to 14 days of gradual escalation of exercise intensity and duration.<sup>5,10,11,16</sup> However, poor fitness levels and extreme temperatures can prolong this period, requiring up to 2 to 3 months to fully take effect.<sup>5,7</sup>

The National Athletic Trainers Association (NATA) and National Collegiate Athletic Association (NCAA)

have released consensus guidelines regarding heat acclimatization protocols for football athletes at the high school and college levels (**Tables 3 and 4**). Each of these guidelines involves an initial period without use of protective equipment, followed by a gradual addition of further equipment.<sup>11,16</sup>

**Secondary Prevention**

Despite physicians' best efforts to prevent all cases of EHI, athletes will still experience the effects of exercise-induced hyperthermia. The goal of secondary prevention is to slow the progression of this hyperthermia so that it does not progress to more dangerous EHI.

*Hydration.* Dehydration is an important risk factor for EHI. Sweat maintains thermoregulation by dissipating heat generated during exercise; however, it also contributes to body water losses. Furthermore, intravascular depletion decreases stroke

**Table 3. NCAA Heat Acclimatization Guidelines<sup>11</sup>**

Day	Practice Guideline	Equipment
1-2	Single, 3-hour practice OR 2-hour practice and 1-hour field session	Helmet only
3-4	Single, 3-hour practice OR 2-hour practice and 1-hour field session	Helmet and shoulder pads
5	Single, 3-hour practice OR 2-hour practice and 1-hour field session	Full pads
6-14	1 day between days with multiple practices <5 hours total practice time daily Walk-throughs <2 hours	Full pads

Abbreviation: NCAA, National Collegiate Athletic Association.

**Table 4. NATA 2009 Heat Acclimatization Guidelines for Secondary School Athletes<sup>16</sup>**

Day	Practice Guideline	Equipment
1-2	No more than 1 practice per day Total practice time <3 hours 1-hour maximum walk-through, spaced at least 3 hours between sessions No contact	Helmet only
3-5	No more than 1 practice per day Total practice time <3 hours 1-hour maximum walk-through, spaced at least 3 hours between sessions Initiate contact with blocking dummies and sleds	Helmets and shoulder pads
6-14	2-a-day practices must be followed by a single practice day No single practice should exceed 3 hours; 2-a-day practices should not exceed 5 hours in total On single practice days, 1-hour maximum walk-through may occur spaced at least 3 hours from practice	Full pads

<sup>16</sup>Due to the increased risk of heat illness during initial acclimatization, an athletic trainer or trained medical professional should be present for all practices. Abbreviation: NATA, National Athletic Trainers Association.

volume, thereby increasing cardiovascular strain. It is estimated that for every 1% loss in body mass from dehydration, body temperature rises 0.22°C in comparison to a euhydrated state.<sup>6</sup> Dehydration occurs more rapidly in hot environments, as fluid is lost through increased sweat production.<sup>7</sup> After approximately 6% to 10% body weight volume loss, cardiac output cannot be maintained, diminishing sweat production and blood flow to both skin and muscle and causing diminished performance and a significant risk of heat exhaustion.<sup>7</sup> If left unchecked, these physiologic changes result in further elevations in body temperature and increased cardiovascular strain, ultimately placing the athlete at significant risk for development of EHS.

Adequate hydration to maintain euolemia is an important step in avoiding possible EHI. Multiple studies have shown that football players experience a baseline hypovolemia during their competition season,<sup>6</sup> a deficit that is most marked during the first week of practices.<sup>17</sup> This deficit is multifactorial, as football players expend a significant amount of fluid through sweat, are not able to adequately replace these losses during practice, and do not appropriately hydrate off the field.<sup>6,18</sup> Some players, especially linemen, sweat at a higher rate than their teammates, posing a possible risk of significant dehydration.<sup>6</sup> Coaches and players alike should be educated on the importance of adequate hydration to meet their fluid needs.

The goal of hydration during exercise is to prevent large fluid losses that can adversely affect performance and increase risk of EHI;<sup>6</sup> it may be unrealistic to replace all fluid losses during the practice period. Instead, athletes should target complete volume replacement over the post-exercise period.<sup>6</sup> Some recommend hydrating based upon thirst drive; however, thirst is activated following a volume loss of approximately 2% body mass, the same degree of losses that place athletes at an increased risk for performance impairment and EHI.<sup>4,6,11,12</sup> Individuals should have access to fluids throughout practice and competition and be encouraged to hydrate as needed.<sup>6,12,15</sup> Furthermore, staff should modify their practices based upon WBGT and acclimatization status to provide more frequent hydration breaks.

*Hyperhydration and Salt Intake.* Of note, there are inherent risks to hyperhydration. Athletes with low sweat rates have an increased risk of overhydration and the development of exercise-associated hyponatremia (EAH),<sup>6</sup> a condition whose presentation is very similar to EHS. In

addition, inadequate sodium intake and excessive sweating can also contribute to the development of EAH. EAH has been implicated in the deaths of 2 football players in 2014.<sup>1,6</sup> Establishing team hydration guidelines and educating players and staff on appropriate hydration and dietary salt intake is essential to reduce the risk of both dehydration and hyperhydration and their complications.<sup>6</sup>

*Intra-Event Cooling.* During exercise, team physicians can employ strategies for cooling athletes during exertion to mitigate their risk of EHI by decreasing thermal and cardiovascular strain.<sup>4,19</sup> Cooling during exercise is hypothesized to allow for accelerated heat dissipation, where heat is lost from the body more effectively. This accelerated loss enables athletes to maintain a higher heat storage capacity over the duration of exercise, avoiding uncompensated heat stresses that ultimately cause EHI.<sup>19</sup>

Some intra-event cooling strategies include the use of cooling garments, cooling packs, and cold water/slurry ingestion. Cooling garments lower skin temperature, which in turn can decrease thermoregulatory strains;<sup>4</sup> a recent meta-analysis of intra-event cooling modalities revealed that wearing an ice vest during exercise resulted in the greatest decrease in thermal heat strain.<sup>19</sup> Internal cooling strategies—namely ingestion of cold fluids/ice slurry—have shown some mild benefit in decreasing internal temperatures; however, some studies have demonstrated some decrease in sweat production associated with cold oral intake used in isolation.<sup>19</sup> Overall, studies have shown that combining external (cooling clothing, ice packs, fanning) and internal (cold water, ice slurry) cooling methods result in a greater cooling effect than a use of a single method.<sup>4</sup>

### Tertiary Prevention

The goal of tertiary prevention is to mitigate the risk of long-term adverse outcomes following an EHS event. The most effective means of reducing risk for morbidity and mortality is rapid identification and treatment of EHS as well as close evaluation of an athlete's return to activity in heat. This process is spearheaded by an effective and well-rehearsed emergency action plan.

### Diagnosis and Management

Rapid identification and treatment of EHS is crucial to minimizing the risk of poor outcomes.<sup>7</sup> Any delay in the treatment of EHS can dramatically increase the likelihood of associated morbidity and mortality.<sup>20</sup>

EHS is diagnosed by an elevated rectal temperature  $\geq 40^{\circ}\text{C}$  ( $104^{\circ}\text{F}$ ) and associated central nervous system (CNS) dysfunction.<sup>21</sup> EHS should be strongly suspected in any athlete exercising in heat who exhibits signs of CNS dysfunction, including disorientation, confusion, dizziness, erratic behavior, irritability, headache, loss of coordination, delirium, collapse, or seizures.<sup>7,12,15</sup> EHS may also present with symptoms of heat exhaustion, including fatigue, hyperventilation, tachycardia, vomiting, diarrhea, and hypotension.<sup>7,12,15</sup>

Rectal temperature should be taken for any athlete with suspected EHS, as other modalities—oral, skin, axillary, and aural—can be inaccurate and easily modified by ambient confounders such as ambient and skin temperature, athlete hyperventilation, and consumption of liquids.<sup>7,11,12</sup> Athletes exhibiting CNS symptoms with moderately elevated rectal temperatures that do not exceed  $40^{\circ}\text{C}$  should also be assumed to be suffering from EHS and treated with rapid cooling.<sup>11</sup> On the other hand, athletes with CNS symptoms who are normothermic should be assumed to have EAH until ruled out by electrolyte assessment;

IV fluids should be at no more than keep vein open (KVO) pending this determination.<sup>11</sup> In some cases, an athlete may initially present with altered mental status but return to “normal.” However, this improvement may represent a “lucid period”; evaluation should continue with rectal temperature and treatment, as EHS in these cases may progress quickly.<sup>15</sup>

Treatment is centered on rapid, whole body cooling initiated at the first sign of heat illness.<sup>7,22</sup> The goal of treatment is to achieve a rectal temperature  $<38.9^{\circ}\text{C}$  within 30 minutes of the onset of EHS.<sup>15</sup> Upon diagnosis, the athlete should be quickly placed in a tub of ice water to facilitate cold water immersion (CWI) therapy. Some guidelines suggest the athlete’s clothing be removed to potentiate evaporative cooling during CWI;<sup>12</sup> however, cooling should not be delayed due to difficulties in removing equipment. CWI, where a heat stroke victim is submerged in ice water up to their neck while water is continuously circulated, is generally considered to be the gold standard treatment as it is the modality with the highest recorded cooling rates and the lowest rate of morbidity and

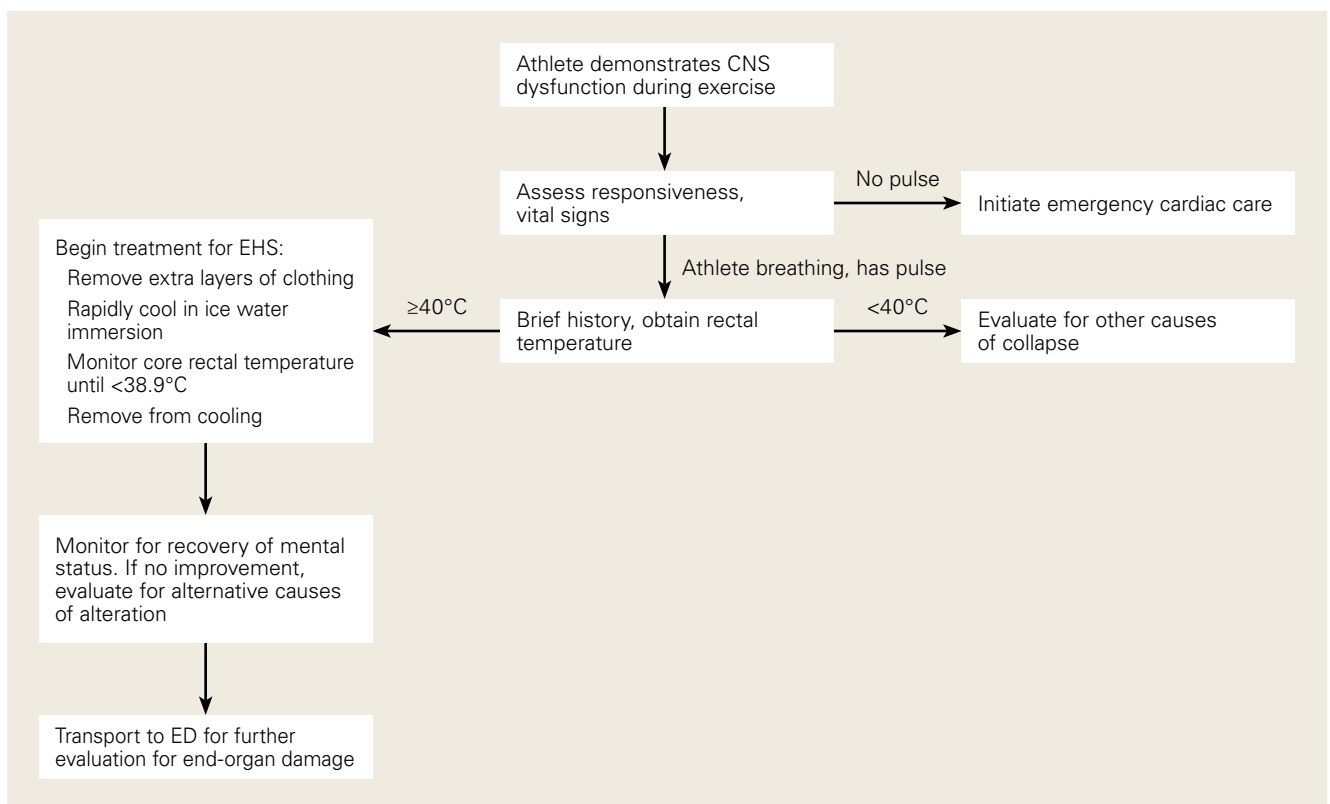


Figure. Algorithm for treatment of exertional heat stroke.<sup>6,9</sup>

Abbreviations: CNS, central nervous system; ED, emergency department; EHS, exertional heat stroke.

mortality.<sup>7,20,21</sup> Multiple studies of CWI have shown that survival nears 100% when aggressive cooling starts within 5 minutes of collapse or identification of EHS.<sup>20,21,22</sup>

If whole body CWI is unavailable, alternative methods of rapid cooling should be employed. Partial CWI, with torso immersion being preferable to the extremities, has been shown to achieve an acceptable rate of cooling to achieve sufficient drops in internal body temperature.<sup>20,23</sup> However, one popular treatment—applying ice packs to the whole body, in particular to the groin and axillae—has not been shown to be sufficient to achieve standard cooling goals.<sup>20</sup> None of these methods have been shown to be as effective as CWI.<sup>23</sup>

Intravenous access should be initiated with fluid resuscitation dictated by the provider's assessment. Normal saline is recommended as the resuscitative fluid of choice, with the rate dictated by clinical judgment and adjusted as guided by electrolyte determination and clinical response. It cannot be overstated that in normothermic patients with confusion, EAH is the diagnosis of

exclusion and aggressive fluid resuscitation should be withheld until electrolyte determination.

Once rectal temperature descends appropriately (~38.9°C), the cooling process should stop and the individual should be transported to a hospital for further observation<sup>20</sup> and evaluation of possible sequelae, including rhabdomyolysis and renal injury, cardiac dysfunction and arrhythmia, severe electrolyte abnormalities, acute respiratory distress syndrome, lactic acidosis, and other forms of end-organ failure (**Figure**).

Rapid cooling is more crucial than transport; transport poses a risk of delayed cooling, which can dramatically increase an individual's risk of morbidity and mortality.<sup>20,23</sup> In situations where a patient can be cooled on-site, physicians should pursue cooling before transporting the patient to a medical treatment facility.

### Emergency Action Plan

Team physicians should be proactive in developing an emergency action plan to address possible EHS events. These plans should be site-specific, addressing procedures for all practice and home competition locations.<sup>12</sup> All competition venues should have a CWI tub on-site in events where there is an increased risk of EHS.<sup>12,15,20</sup> This tub should be set up and functional for all high-risk activities, including practices.<sup>12</sup>

Following recognition of a potential case of EHS, treatment teams should have procedures in place to transport athletes to the treatment area, obtain rectal temperature, initiate rapid cooling, and stabilize the athlete for transport to an emergency department (ED) for further evaluation.<sup>12,15</sup> A written record of treatments and medications provided during athlete stabilization should be maintained and transported with the athlete to the ED.<sup>15</sup> A list of helpful equipment and supplies for treatment of EHS can be found in **Table 5**.

EHS is a unique life-threatening situation where it is best to treat the patient on the sideline before transport.<sup>15</sup> Those athletes transported before cooling risk spending an increased amount of time above critical temperatures for cell damage, which has been associated with increased morbidity and mortality. This mantra of “cool first, transport second” cannot be overemphasized, as those individuals with EHS who present to the ED with a persisting rectal temperature >41°F may risk up to an 80% mortality rate.<sup>24</sup> Conversely, a recent large, retrospective study of 274 EHS events sustained during the Falmouth Road Race found a 100% sur-

**Table 5. Suggested Equipment for Diagnosis and Management of Exertional Heat Stroke<sup>15,28</sup>**

Wet bulb globe temperature (WBGT) device
Rectal thermometer and/or in-dwelling thermistor
Poleless litter for transport into/out of cooling tub
Cots
Lubricating gel
Tub for cold water immersion
Water supply and coolers with ice (crushed or cubed)
Cooling fans
Supplies for drying and rewarming (blankets, towels)
Plastic bags
Oral rehydration fluids with cups
Intravenous tubing and cannulation needles, normal saline intravenous fluids, sharps and biohazard disposal containers
Disposable gloves
Stethoscope
Blood pressure cuff
Automated external defibrillator
Consider iStat for point-of-care electrolyte analysis

vival rate when athletes were rapidly identified via rectal thermometry and treated with aggressive, rapid cooling through CWI.<sup>21</sup>

### Return to Play

Perhaps the most challenging and important role the team physician has is determining an athlete's return to play following EHI, as there currently are no evidence-based guidelines for return to activity for these athletes.<sup>7</sup> The decisions surrounding return to play are highly individualized, as recovery from EHS and heat injury is associated with the duration of internal body temperature elevation above the critical level (40°C).<sup>7,20</sup> Guidelines for return to activity following recovery from EHI differ among experts and institutions.<sup>7,25</sup> The general consensus from these guidelines is that, at minimum, athletes should not participate in any physical activity until they are asymptomatic and all blood tests have normalized.<sup>11</sup> Following this asymptomatic period, most guidelines advocate for a slow, deliberate return to activity.<sup>11</sup> The American College of Sports Medicine (ACSM) offers one reasonable approach to the returning athlete following EHS:<sup>7</sup>

- No exercise for at least 7 days following release from medical care.
- Follow-up with a physician 1 week after release from medical care for physical examination and any warranted lab or radiologic studies (based upon organ systems affected during EHS).
- Once cleared to return to activity, the athlete begins exercise in a cool environment, gradually increasing the duration, intensity, and heat exposure over 2 weeks to demonstrate heat tolerance and acclimatization.
- Athletes who cannot resume vigorous activity due to recurrent symptoms (eg, excessive fatigue) should be reevaluated after 4 weeks. Laboratory exercise-heat tolerance testing may be useful in this setting.
- The athlete may resume full competition once they are able to participate in full training in the heat for 2 to 4 weeks without adverse effects. Heat tolerance testing (HTT) in these athletes remains controversial.<sup>5,26</sup> The ACSM recommends that HTT be considered only for those unable to return to vigorous activity after a suitable period (approximately 4 weeks). In contrast, the Israeli Defense Force (IDF) uses HTT to evaluate soldiers following EHS to guide decision-making about return to duty.<sup>27</sup> The IDF HTT assumes that individuals will respond differently to heat stresses. They identify individuals who are "heat intolerant" as being un-

able to tolerate specific heat challenges, indicated by increases in body temperature occurring more rapidly than normal responders under identical environmental and exercise conditions. However, despite being used for more than 30 years, there is no clear evidence that HTT adequately predicts who will experience subsequent episodes of EHS.

### Conclusion

While the recognized cornerstone of being a team physician is the provision of medical care, the ACSM Team Physician Consensus Statement<sup>28</sup> further delineates the medical and administrative responsibilities as both (1) understanding medical management and prevention of injury and illness in athletes; and (2) awareness of or involvement in the development and rehearsal of an emergency action plan. These tenets are critical for the team physician who accepts the responsibility to cover sports at the high school level or higher. Football team physicians play an essential role in mitigating risk of EHI in their athletes. Through development and execution of both comprehensive prevention strategies and emergency action plans, physicians can work to minimize athletes' risk of both developing and experiencing significant adverse outcomes from an EHI.

---

Dr. Sylvester is a Resident Physician, Department of Family Medicine, National Capitol Consortium-Fort Belvoir Community Hospital, Fort Belvoir, Virginia. Mr. Belval is Director of Research and Director of Military and Occupational Safety, Korey Stringer Institute, Department of Kinesiology, University of Connecticut, Storrs. Dr. Casa is Chief Executive Officer, Korey Stringer Institute, Department of Kinesiology, University of Connecticut, Storrs. Dr. O'Connor is Professor and Chair of Military and Emergency Medicine, and Associate Director of the Consortium for Health and Military Performance, Uniformed Services University of the Health Sciences, Bethesda, Maryland.

Address correspondence to: Francis G. O'Connor, MD, MPH, Military and Emergency Medicine, Uniformed Services University of the Health Sciences, 4301 Jones Bridge Road, Bethesda, MD 20814 (tel, 301-295-0220; email, francis.oconnor@usuhs.edu).

*Am J Orthop.* 2016;45(6):340-348. Copyright Frontline Medical Communications Inc. 2016. All rights reserved.

### References

1. Kucera KL, Klossner D, Colgate B, Cantu RC. Annual Survey of Football Injury Research: 1931-2014. National Center for Catastrophic Sport Injury Research Web site. <https://nccsir.unc.edu/files/2013/10/Annual-Football-2014-Fatalities-Final.pdf>. Accessed May 31, 2016.
2. Boden BP, Breit I, Beachler JA, Williams A, Mueller FO. Fatalities in high school and college football players. *Am J Sports Med.* 2013;41(5):1108-1116.
3. Bouchama A, Knochel JP. Heat stroke. *N Engl J Med.*



- 2002;346(25):1978-1988.
4. Racinais S, Alonso JM, Coutts AJ, et al. Consensus recommendations on training and competing in the heat. *Scand J Med Sci Sports*. 2015;25 Suppl 1:6-19.
  5. Pryor RR, Casa DJ, Adams WM, et al. Maximizing athletic performance in the heat. *Strength Cond J*. 2013;35(6):24-33.
  6. Adams WM, Casa DJ. Hydration for football athletes. *Sports Sci Exchange*. 2015;28(141):1-5.
  7. American College of Sports Medicine, Armstrong LE, Casa DJ, et al. American College of Sports Medicine position stand. Exertional heat illness during training and competition. *Med Sci Sports Exerc*. 2007;39(3):556-572.
  8. Yard EE, Gilchrist J, Hailayesus T, et al. Heat illness among high school athletes—United States, 2005-2009. *J Safety Res*. 2010;41(6):471-474.
  9. Huffman EA, Yard EE, Fields SK, Collins CL, Comstock RD. Epidemiology of rare injuries and conditions among United States high school athletes during the 2005-2006 and 2006-2007 school years. *J Athl Train*. 2008;43(6):624-630.
  10. Kerr ZY, Casa DJ, Marshall SV, Comstock RD. Epidemiology of exertional heat illness among U.S. high school athletes. *Am J Prev Med*. 2013;44(1):8-14.
  11. 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.
  12. Casa DJ, Almquist J, Anderson SA. 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.
  13. Gardner JW, Kark JA, Karnei K, et al. Risk factors predicting exertional heat illness in male Marine Corps recruits. *Med Sci Sports Exerc*. 1996;28(8):939-944.
  14. Gundstein 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.
  15. 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.
  16. Casa DJ, Csillan D; Inter-Association Task Force for Preseason Secondary School Athletics Participants, et al. Preseason heat-acclimatization guidelines for secondary school athletics. *J Athl Train*. 2009;44(3):332-333.
  17. 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.
  18. Stover EA, Zachwieja J, Stofan J, Murray R, Horswill CA. Consistently high urine specific gravity in adolescent American football players and the impact of an acute drinking strategy. *Int J Sports Med*. 2006;27(4):330-335.
  19. Bongers CC, Thijssen DH, Veltmeijer MTW, Hopman MT, Eijssvogels TM. Precooling and percooling (cooling during exercise) both improve performance in the heat: a meta-analytical review. *Br J Sports Med*. 2015;49(6):377-384.
  20. 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.
  21. 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.
  22. Casa DJ, Kenny GP, Taylor NA. Immersion treatment for exertional hyperthermia: cold or temperate water? *Med Sci Sports Exerc*. 2010;42(7):1246-1252.
  23. 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):115-122.
  24. Argaud L, Ferry T, Le QH, et al. Short- and long-term outcomes of heat stroke following the 2003 heat wave in Lyon, France. *Arch Intern Med*. 2007;167(20):2177-2183.
  25. 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.
  26. Kazman JB, Heled Y, Lisman PJ, Druyan A, Deuster PA, O'Connor FG. Exertional heat illness: the role of heat tolerance testing. *Curr Sports Med Rep*. 2013;12(2):101-105.
  27. 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.
  28. Herring SA, Kibler WB, Putukian M. Team Physician Consensus Statement: 2013 update. *Med Sci Sports Exerc*. 2013;45(8):1618-1622.
  29. Heat stroke treatment. Korey Stringer Institute University of Connecticut Web site. <http://ksi.uconn.edu/emergency-conditions/heat-illnesses/exertional-heat-stroke/heat-stroke-treatment/>. Accessed June 14, 2016.
  30. Headquarters, Department of the Army and the Air Force. Heat Stress Control and Heat Casualty Management. Technical Bulletin Medical 507. [http://www.dir.ca.gov/oshsb/documents/Heat\\_illness\\_prevention\\_tmed507.pdf](http://www.dir.ca.gov/oshsb/documents/Heat_illness_prevention_tmed507.pdf). Published March 7, 2003. Accessed June 14, 2016.