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

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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. USA Football estimates that an additional 3 million youth participate in community football leagues. 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. 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 (EHS) has emerged as one of the leading indirect causes of death in high school and collegiate football. Boden and colleagues 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. 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).

**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.” 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 of heat to the surrounding environment by leveraging conduction, convection, and evaporation. 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, 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. 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.

An estimated 9000 cases of EHI occur annually across all high school sports, with an incidence of 1.6:100,000 athlete-exposures. Studies have demonstrated, however, that EHI occurs in football 11.4 times more often than in all other high school sports combined. The incidence of nonfatal EHI in all levels of football is 4.42-5:100,000. Between 2000 and 2014, 41 football players died from EHS. In football, approximately 75% of all EHI events occurred during practices, while only 25% of incidents occurred during games.

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.

**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. 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.
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. \(^{13}\) **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. \(^{11,14}\) The greatest risk for EHS is performing high-intensity exercise (>75% VO2max) when WBGT >28°C (82°F). \(^7\) 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. \(^{14}\) Consensus guidelines recommend that activities be modified based on WBGT (Table 2). \(^{7,12}\)

![Table 2.](ajo045090340_t2.jpg)

Table 2.

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. \(^11\) **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. \(^5,15\) 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. \(^5,11\) Likewise, wearing dark clothing encourages radiant absorption of heat, further contributing to potential thermal dysregulation. \(^5\) Use of a helmet is a specific risk factor for EHI, as significant heat dissipation occurs through the
head. 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. When football season begins in late summer, athletes are least conditioned as temperatures reach their seasonal peak, causing increased risk of EHI. 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. 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. 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. However, poor fitness levels and extreme temperatures can prolong this period, requiring up to 2 to 3 months to fully take effect.

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.

**Table 3.**

### 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.

**Table 4.**
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 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. Dehydration occurs more rapidly in hot environments, as fluid is lost through increased sweat production. 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. 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 euvoemia is an important step in avoiding possible EHI. Multiple studies have shown that football players experience a baseline hypovolemia during their competition season, a deficit that is most marked during the first week of practices. 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. Some players, especially linemen, sweat at a higher rate than their teammates, posing a possible risk of significant dehydration. 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; 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. 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. Individuals should have access to fluids throughout practice and competition and be encouraged to hydrate as needed. 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), 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. 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.

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. 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.

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; 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. 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. 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.
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. Any delay in the treatment of EHS can dramatically increase the likelihood of associated morbidity and mortality.

EHS is diagnosed by an elevated rectal temperature ≥40°C (104°F) and associated central nervous system (CNS) dysfunction. 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. EHS may also present with symptoms of heat exhaustion, including fatigue, hyperventilation, tachycardia, vomiting, diarrhea, and hypotension.

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. Athletes exhibiting CNS symptoms with moderately elevated rectal temperatures that do not exceed 40°C should also be assumed to be suffering from EHS and treated with rapid cooling. 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. 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.

Treatment is centered on rapid, whole body cooling initiated at the first sign of heat illness. The goal of treatment is to achieve a rectal temperature <38.9°C within 30 minutes of the onset of EHS. 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; 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 mortality. Multiple studies of CWI have shown that survival nears 100% when aggressive cooling starts within 5 minutes of collapse or identification of EHS.

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. 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. None of these methods have been shown to be as effective as CWI.

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 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).

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. 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. All competition venues should have a CWI tub on-site in events where there is an increased risk of EHS. This tub should be set up and functional for all high-risk activities, including practices.

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. A written record of treatments and medications provided during athlete stabilization should be maintained and transported with the athlete to the ED. A list of helpful equipment and supplies for treatment of EHS can be found in Table 5.
Table 5. Suggested Equipment for Diagnosis and Management of Exertional Heat Stroke

<table>
<thead>
<tr>
<th>Item</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heat bank</td>
<td>For gentle heat therapy</td>
</tr>
<tr>
<td>Rectal thermometer and/or dual-channel rectal thermometer</td>
<td>For temperature monitoring</td>
</tr>
<tr>
<td>Ice Rolls</td>
<td>For application to the rectum</td>
</tr>
<tr>
<td>Cooling fan</td>
<td>For air circulation</td>
</tr>
<tr>
<td>Supplies for drying and rehydrating blankets, towels</td>
<td>For drying and rehydrating the athlete</td>
</tr>
<tr>
<td>Plastic bags</td>
<td>For transport and cooling</td>
</tr>
<tr>
<td>Oral rehydration fluids with cups</td>
<td>For oral rehydration</td>
</tr>
<tr>
<td>Intravenous tubing and connection needles, normal saline intravenous fluids, sterile and biohazard disposal containers</td>
<td>For intravenous therapy</td>
</tr>
<tr>
<td>Disposable gloves</td>
<td>For contamination control</td>
</tr>
<tr>
<td>Stethoscope</td>
<td>For auscultation</td>
</tr>
<tr>
<td>Blood pressure cuff</td>
<td>For monitoring blood pressure</td>
</tr>
<tr>
<td>Automated external defibrillator</td>
<td>For cardiac arrest intervention</td>
</tr>
<tr>
<td>Cold Pack for post-care follow-up analysis</td>
<td>For monitoring core temperature</td>
</tr>
</tbody>
</table>

EHS is a unique life-threatening situation where it is best to treat the patient on the sideline before transport. 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. Conversely, a recent large, retrospective study of 274 EHS events sustained during the Falmouth Road Race found a 100% survival rate when athletes were rapidly identified via rectal thermometry and treated with aggressive, rapid cooling through CWI.

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. 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). Guidelines for return to activity following recovery from EHI differ among experts and institutions. 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. Following this asymptomatic period, most guidelines advocate for a slow, deliberate return to activity. The American College of Sports Medicine (ACSM) offers one reasonable approach to the returning athlete following EHS:

- 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. 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. The IDF HTT assumes that individuals will respond differently to heat stresses. They identify individuals who are “heat intolerant” as being unable 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 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.

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Key Info

Figures/Tables

References

References


Multimedia

Product Guide

Product Guide

- STRATAFIX™ Symmetric PDS™ Plus Knotless Tissue Control Device
- STRATAFIX™ Spiral Knotless Tissue Control Device
- BioComposite SwiveLock Anchor
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