

Heat-Related Illness in Athletes

Allyson S. Howe,[†] MD, and Barry P. Boden,[‡] MD

From the [†]Malcolm Grow Medical Center Family Medicine Residency, Andrews Air Force Base, Maryland, and [‡]The Orthopaedic Center, Rockville, Maryland

Heat stroke in athletes is entirely preventable. Exertional heat illness is generally the result of increased heat production and impaired dissipation of heat. It should be treated aggressively to avoid life-threatening complications. The continuum of heat illness includes mild disease (heat edema, heat rash, heat cramps, heat syncope), heat exhaustion, and the most severe form, potentially life-threatening heat stroke. Heat exhaustion typically presents with dizziness, malaise, nausea, and vomiting, or excessive fatigue with accompanying mild temperature elevations. The condition can progress to heat stroke without treatment. Heat stroke is the most severe form of heat illness and is characterized by core temperature $>104^{\circ}\text{F}$ with mental status changes. Recognition of an athlete with heat illness in its early stages and initiation of treatment will prevent morbidity and mortality from heat stroke. Risk factors for heat illness include dehydration, obesity, concurrent febrile illness, alcohol consumption, extremes of age, sickle cell trait, and supplement use. Proper education of coaches and athletes, identification of high-risk athletes, concentration on preventative hydration, acclimatization techniques, and appropriate monitoring of athletes for heat-related events are important ways to prevent heat stroke. Treatment of heat illness focuses on rapid cooling. Heat illness is commonly seen by sideline medical staff, especially during the late spring and summer months when temperature and humidity are high. This review presents a comprehensive list of heat illnesses with a focus on sideline treatments and prevention of heat illness for the team medical staff.

Keywords: heat stroke; heat exhaustion; dehydration; prevention

Heat illness has received substantial public attention in the United States after the recent deaths of collegiate and professional athletes in the National Collegiate Athletic Association (NCAA), National Football League, and Major League Baseball from heat stroke. Heat stroke is currently the third leading cause of death in athletes behind cardiac disorders and head and neck trauma.^{4,30} Early recognition and prompt treatment are keys to the prevention of morbidity and mortality from heat illness. Because exertional heat stroke is entirely preventable, this article focuses on prevention tactics that may help reduce the incidence of catastrophic heat stroke events. These prevention strategies include proper acclimatization to the heat; proper fluid replacement before, during, and after exertion; wearing proper clothing during certain environmental conditions; and early recognition of heat illness via direct monitoring of athletes by other players, coaches, and medical staff.

It is unclear why some athletes progress to heat illness while others with similar risk factors are spared when exposed to the same environmental stressors. Studies examining high-risk individuals for early signs and symptoms of heat illness are ongoing. The ability to predict who is most susceptible to heat injury and thus offer appropriate early intervention treatments would be a major breakthrough in prevention of this condition.

*Address correspondence to Allyson S. Howe, Family Medicine Clinic, 1075 W. Perimeter Road, Andrews AFB, MD 20762 (e-mail: allyhowe@hotmail.com).

No potential conflict of interest declared.

DEFINITION OF HEAT-RELATED ILLNESS

The graded continuum of heat illness progresses from very mild to more serious disease to a life-threatening condition known as heat stroke (Table 1). There is no evidence that mild heat illness (heat edema, heat rash, heat cramps, or heat syncope) will progress to severe disease if untreated. However, the development of heat exhaustion is significant. Without treatment, heat exhaustion has the potential to progress to heat stroke.

Heat Edema

Very mild forms of heat illness occur in the form of heat edema and heat rash (also known as prickly heat or miliaria rubra). Heat edema appears as dependent soft tissue swelling, usually in the lower extremities, in a person lacking acclimatization. Peripheral vasodilation to produce heat loss leads to pooling of interstitial fluid in the distal extremities. This leads to an increase in vascular hydrostatic pressure and resultant third spacing of intravascular fluid into the surrounding soft tissue. The condition is more commonly seen in older adults who enter a tropical climate without proper acclimatization.

Heat Rash

Miliaria rubra (ie, heat rash or prickly heat) presents as a pinpoint papular erythematous, often intensely pruritic, eruption in areas covered with clothing. It commonly presents in the waist or over highly sweaty areas such as the trunk or groin. Profuse sweating saturates

TABLE 1
Criteria for Diagnosis of Heat Illness^a

Condition	Core Temperature °F (°C)	Associated Symptoms	Associated Signs
Heat edema	Normal	None	Mild edema in dependent areas (ankles, feet, hands)
Heat rash	Normal	Pruritic rash	Papulovesicular skin eruption over clothed areas
Heat syncope	Normal	Dizziness, generalized weakness	Loss of postural control, rapid mental status recovery once supine
Heat cramps	Normal or elevated but <104°F (40°C)	Painful muscle contractions (calf, quadriceps, abdominal)	Affected muscles may feel firm to palpation.
Heat exhaustion	98.6°F-104°F (37°C-40°C)	Dizziness, malaise, fatigue, nausea, vomiting, headache	Flushed, profuse sweating, cold clammy skin, normal mental status
Heat stroke	>104°F (40°C)	Possible history of heat exhaustion symptoms before mental status change	Hot skin with or without sweating, CNS disturbance (confusion, ataxia, irritability, coma)

^aCNS, central nervous system.

the skin surface and clogs the sweat ducts. Obstruction of the ducts results in leakage of eccrine sweat into the epidermis. Secondary infection with staphylococcus may produce prolonged symptoms.

Heat Syncope

Heat syncope occurs with orthostatic hypotension resulting from peripheral vasodilation (physiologic response to heat production) and venous pooling. Prolonged standing after significant exertion and rapid change in body position after exertion, such as from sitting to standing, may lead to heat syncope.^{31,40} Athletes with heat syncope tend to recover their mental status quickly once supine, as blood flow to the central nervous system returns.

Heat Cramps

One of the earliest indications of heat illness presents in the form of muscle spasm or muscle cramps. This typically results after excessive heat exposure that leads to profuse sweating coupled with inadequate fluid and electrolyte intake; the muscles may begin to spasm, causing painful contractions. Often this results in the inability to continue activity for a short time. Sodium loss is thought to play a significant role in exacerbating heat cramps.²¹ Evidence for magnesium, potassium, or calcium abnormalities contributing to heat cramps is not yet clear.¹⁷ Heat cramps may occur alone or concurrently with symptoms of heat exhaustion. Athletes with heat cramps have not been shown to be predisposed to serious heat illness such as heat stroke.

Heat Exhaustion

Heat exhaustion may be the initial presentation of heat illness. Typically a condition in which core body temperature is between 37°C (98.6°F) and 40°C (104°F), heat exhaustion often presents with malaise, fatigue, and dizziness. Heavy sweating is classically noted as well as nausea, vomiting, headache, fainting, weakness, and cold or

clammy skin. Critical to the diagnosis of heat exhaustion is normal mentation and stable neurologic status.

Heat Stroke

Heat exhaustion can progress to heat stroke when unrecognized or untreated. Heat stroke is characterized by both an elevated core temperature of 40°C or greater and central nervous system (CNS) disturbance (irritability, ataxia, confusion, coma). In the setting of suspected heat illness with a temperature below 40°C and mental status changes, heat stroke should still be considered a likely diagnosis as some cooling could have taken place en route to medical treatment. Treatment for heat stroke should be initiated while evaluating for other conditions. With this potentially fatal condition, prompt recognition and treatment offer the best chance of survival.

Classic and Exertional Heat Stroke

Two types of heat stroke have been described: classic, in which the environment plays a major role in an individual's ability to dissipate heat, and exertional, in which intrinsic heat production is the primary cause for hyperthermia. Necessary with both conditions is the dysfunction of the thermoregulatory system to dissipate heat created by or absorbed by the body. The idea of intrinsic heat production with exertional heat stroke is supported by the observations that most classic heat stroke events are linked to environmental heat waves, while exertional-type heat strokes can and have occurred in all types of weather.

The definition of classic versus exertional heat stroke offers no utility in management of the patient with heat stroke. Classic heat stroke has been defined in some texts by anhidrotic skin. It should be noted that exertional heat stroke victims often demonstrate profuse sweating. The presence or absence of sweating is an inconsequential diagnostic criterion for heat stroke. Treatment of the 2 conditions is the same: reduce heat as quickly as possible and monitor for complications of heat exposure.

INCIDENCE

Approximately 400 deaths can be attributed to all types of heat-related illness in the United States annually.¹⁰ From 1979 to 1999, about half (48%) of these deaths were related to weather conditions.¹⁰ With current concerns regarding global warming, it is likely that there will be a rise in the frequency and length of heat waves, including heat waves occurring in previously temperate environments.⁴⁶ Heat-related illnesses are likely to continue to rise if prevention techniques are not employed.

Exertional heat stroke is the third leading cause of death in athletes.³⁰ Obtaining the actual incidence of these events is difficult, as heat stroke is not reportable in any state.⁴⁶

Football has been identified as the sport with the greatest number of associated catastrophic injuries for male athletes and the greatest number of heat stroke fatalities.³⁵ The National Center for Catastrophic Sport Injury Research has been collecting details about heat stroke deaths in football players since 1931. In their most recent survey, it was noted that there were 26 deaths in high school, collegiate, and professional football from 1995 to 2005.³⁵

THERMOREGULATION

At a cellular level in a healthy person, heat stress produces a predictable cascade of events. Peripheral vasodilation at the skin level will produce heat loss and shunt blood from the central circulation. Sweating will occur, and vaporization of the sweat releases heat. Sodium loss in sweat can be significant and can play a role in dehydration from this process if fluids containing water and salt are not replaced. Cardiopulmonary responses include tachycardia, increased cardiac output, and increased minute ventilation. These responses can be impaired by dehydration and excess salt loss.

Acclimatization to a hotter environment may take several weeks. Modifications to the renal and cardiovascular systems by way of improved sodium retention, increased renal glomerular filtration rate, and enhanced cardiovascular performance occur and help to prevent organ damage. Nearly all cells in the body possess the ability to make heat shock proteins, which serve to assist the cell in tolerating the heat.⁹

The basis for heat exchange from a human body to the environment occurs in 4 ways—conduction, convection, radiation, and evaporation. All methods are dependent on the presence of a heat gradient. Heat will transfer from a hotter object to a cooler one. Loss of this heat gradient by certain environmental conditions can inhibit appropriate thermoregulation.

Conduction occurs with direct transfer of heat during contact with a cooler object. Convection is the cooling of the air around the body by way of cooler air passing over the warmer exposed skin. This method depends on wind current to bring cooler air to the body or movement of the body through the environment to produce a heat gradient (eg, with cycling). Lack of wind will reduce heat lost by convection. Radiation is a direct release of heat from a body into

TABLE 2
Summary of Risk Factors for Heat Illness

Internal Factors	External (Environmental) Factors
Age (<15 years or >65 years)	Activity level
Alcohol consumption	Excessive clothing wear
Comorbid medical conditions— respiratory, cardiovascular, hematologic	Lack of water or sufficient shade
Dehydration	Temperature (ambient)
History of heat-related illness	Humidity
Lack of air conditioning	Wet bulb globe temperature
Lack of appropriate sleep	
Medications or supplements	
Obesity	
Overmotivation	
Poor acclimatization	
Poor cardiovascular fitness	
Recent febrile illness	
Sickle cell trait	
Skin condition—eczema, psoriasis, burns, etc	
Social isolation	
Sunburn	
Use of psychiatric medications	

the environment.²⁰ This works well if the body temperature exceeds the ambient temperature. In the case of high ambient temperature, the heat gradient does not allow for heat loss from the body to the environment. Evaporation, via perspiration, is our most effective way to release heat. It has been demonstrated that up to 600 kcal/h of heat can be dissipated by this method.^{22,46}

RISK FACTORS

Risk factors for the development of heat illness can generally be classified into 1 of 2 areas: internal (related to the athlete) or external (environmental) factors. Internal factors include prescription and over-the-counter medications as well as medical conditions in the athlete (sickle cell trait, dehydration, recent febrile illness, sleep deprivation, sunburn, obesity, etc).⁴ External factors are temperature, humidity, excessive clothing or equipment, and activity level of the athlete (Table 2).

Dehydration is a key precursor to heat illness. Dehydration is determined by both inadequate fluid intake and excessive fluid loss primarily through sweating. Many sports medicine disciplines believe that proper hydration can reduce the incidence of heat injuries.²³

Populations at high risk of heat illness include the elderly, children, and those with comorbid medical conditions that may inhibit their thermoregulatory ability. Alcoholism, living on the higher floors of multistory buildings, and the use of psychiatric medications, such as tricyclic antidepressants and typical antipsychotics, contribute to an increased risk of developing heat stroke.²⁸ Other medications have also

TABLE 3
Medication Classes That May Predispose
to Heat Illnesses^a

Alcohol
Alpha-adrenergic agents
Amphetamines
Anticholinergics
Antihistamines
Antihypertensives (ie, beta blockers, calcium channel blockers, diuretics)
Benzodiazepines
Dietary supplements (ie, ephedra, diet pills)
Illicit drugs (ie, cocaine, heroin, PCP, LSD)
Laxatives
Monoamine oxidase inhibitors
Thyroid agonists
Tricyclic antidepressants
Typical antipsychotics (phenothiazines, thioxanthines, butyrophenones)

^aPCP, phencyclidine; LSD, lysergic acid diethylamide.

been shown to increase the risk of heat illness. Many of these medications may be taken by athletes to improve their performance or to treat common medical conditions. The mechanism by which these medications contribute to heat illness varies. Stimulant medications (ie, amphetamines, ephedra, thyroid agonists) can cause increased heat production. Anticholinergic medications (ie, antihistamines, antidepressants, antipsychotics) decrease sweat production. Medications that affect the cardiovascular system (ie, antihypertensives, diuretics) may inhibit natural cardioprotective responses to dehydration and heat illness.⁴⁰ The team medical staff should be aware of any athletes taking medications or supplements. The common types of medications associated with heat illness are summarized in Table 3.^{5,22}

Military recruits and athletes share a common theme in heat illness. In these populations, illness is often related more to the intrinsic heat production during relative overexertion (for a specific body type, physical fitness level, hydration status) than to ambient temperature as a primary source of heat. In a case series of heat stroke events in the Israeli army, 50% of cases were seen in the first 6 months of service.¹⁸ Overmotivation, the desire of a recruit to push physical limits of exercise, was often linked to heat illness events. Sixty percent of the soldiers with exertional heat stroke were overweight, and 30% of the events occurred in the spring months.¹⁸

Environmental factors that may be responsible for heat illness include ambient temperature, level of humidity, and the type of clothing a person wears. In addition, access to adequate water and shade may play a role. As the ambient temperature rises, the basal metabolic rate increases proportionately.

An interesting phenomenon to explain heat illness in cooler environments, where heat stroke is less expected, is known as the "penguin effect." The idea stems from actions typical of Antarctic penguins for heat conservation. As a crowd forms, people in the middle of the crowd tend to

absorb the radiant heat given off by others around them and cannot shed their own radiant heat because of the higher ambient temperature created around them. They are also subjected to poor convection by wind-shielding. An example of this occurs commonly during running races, when large groups of people pace together and spend a good deal of the race running in a pack. The runners in the middle of the pack are at higher risk of heat illness because they cannot dissipate their heat as efficiently as those at the outside of the pack. This may explain why heat illness can occur in situations in which the ambient temperature is relatively cool.⁷

DIAGNOSIS

Heat Edema

Mild swelling of the hands and feet without history of injury may be related to heat illness. Heat edema often occurs in a hot environment where full acclimatization has not occurred. There should be no concurrent systemic symptoms to suggest heart, liver, or kidney failure as possible cause. Heat edema is rare in conditioned athletes, but it must be considered in the aging athletic population.

Heat Rash

Commonly termed "prickly heat," heat rash usually is pruritic and appears papulovesicular (Figure 1). It occurs when a person is exposed to high heat and humidity that lead to excessive sweating. Obstruction of sweat glands allows leakage of eccrine sweat into the epidermis or dermis. The rash will be found in locations that have been occluded by clothing and areas of friction (neck, trunk, axilla, groin, and waist).

The differential diagnosis for such a rash may include viral exanthem, rhus dermatitis, or urticaria. Distinguishing heat rash from these conditions can be difficult on physical examination alone. Generally, heat rash will have a rapid onset, is located over sweaty areas, and is associated with a history of excessive heat exposure and sweating. The rash may have a stinging or "prickling" sensation.²⁵ A viral exanthem generally follows a period of viral illness, most often involving the respiratory tract. The rash tends to be generalized and maculopapular in character. Rhus dermatitis produces fairly discrete vesicular lesions that are intensely pruritic. Exposure to a wooded area has usually occurred. Urticaria can result after exercise and usually presents with elevated erythematous wheals in the skin that are pruritic and may coalesce into large lesions. The athlete may have a history of atopic conditions. They can be generalized but usually begin on the neck and trunk.³²

Heat Syncope

Athletes with heat syncope will most commonly present after they have stopped exercising. Venous pooling and peripheral vasodilation (a cardiovascular method to increase heat loss) can lead to hypotension and syncope,



Figure 1. Miliaria rubra (heat rash, or prickly heat).

particularly if the athlete stops and stands still shortly after exercise.⁴⁰ The loss of muscular contraction in the lower extremities reduces venous return. An athlete with heat syncope should recover rapidly once supine, as cerebral blood flow is restored.

Any syncopal event in an athlete should prompt evaluation of the cardiorespiratory and central nervous systems for serious causes of syncope. These may include cardiomyopathy, myocardial infarction, arrhythmia, asthma, and seizure. In addition, injuries may result from a fall with any syncopal event and should be considered when examining the patient.³¹

Heat Cramps

Painful muscle cramps most commonly involve the quadriceps, hamstrings, gastrocnemius, and abdominal musculature. Cramps often occur in these active muscle groups when they have been challenged by a prolonged exercise event of more than 2 hours.²² Likely a result of fluid and sodium depletion, heat cramps are more common in individuals with heavy amounts of salt in their sweat.^{5,43} Tennis players, American football players, steel mill workers, and military members who deploy to hot environments have a high incidence of heat cramps.²¹

Heat Exhaustion

Athletes with heat exhaustion frequently complain of fatigue, malaise, muscle cramps, nausea, vomiting, and dizziness. The patient should, by definition, be alert and oriented and have normal cognition. There may be evidence of circulatory compromise seen as tachycardia or hypotension. Orthostatic syncope can occur but should be followed by rapid return to normal CNS function. Often the skin is profusely diaphoretic. Identification of heat exhaustion is of utmost importance in order to avoid progression to heat stroke. If there is any question regarding the mental status, it is prudent to treat for heat stroke and continue evaluation for other conditions such as hyponatremia, hypoglycemia, seizure, or closed head trauma.

Heat Stroke

Heat stroke should be considered in any athlete with a change in mental status or alteration in consciousness during or after an athletic endeavor. The differential diagnosis of heat stroke includes life-threatening emergencies such as myocardial infarctions, hyponatremia, cerebrovascular accidents, and anaphylaxis.

The diagnosis of heat stroke is dependent on accurate core body temperature recordings $>40^{\circ}\text{C}$ (104°F) and CNS dysfunction. In situations in which cooling has already begun en route, temperature criteria may not be met. When CNS changes are present but core temperature is below 40°C , it is important to initiate treatment for heat stroke while exploring the differential diagnosis of the patient's mental status changes.

The most reliable measurement of core temperature is obtained via the rectal route. This may be uncomfortable to patients, but it is the standard method to measure core body temperature.¹⁹ A handheld electronic digital thermometer can be easily carried in a medical kit and used to obtain the rectal temperature. In an adult patient, laying the patient on his or her side and pulling the shorts down so that the rectum can be easily accessed is optimal. Shielding for privacy with a sheet or other people can be considered but should not delay measurement or treatment. The provider should place a plastic sheath over the thermometer and cover it with sufficient lubrication. The thermometer should be inserted into the rectum approximately 1 to 2 inches so that the metal tip is no longer exposed. Research evaluating a swallowed pill that provides temperature recording is promising but is only advantageous when the athlete has ingested the pill before presentation with heat illness.

Athletes with heat stroke have often progressed through heat exhaustion without recognition of the condition. Their teammates or coaches may have observed vomiting, fatigue, or loss of athletic ability that progressed to confusion, ataxia, or agitation. Although in the setting of classic heat stroke a person's skin may be identified as dry and hot (anhidrosis), this is often not the case with exertional heat stroke. Recognition of profuse sweating should not eliminate the diagnosis of heat stroke.

Hyponatremia

A condition especially important to mention in the differential diagnosis of exertional heat stroke is exertional hyponatremia. Defined by serum sodium levels <130 mmol/L, this type of hyponatremia can present with a clinical appearance similar to heat stroke, with mental status changes and an altered level of consciousness. Exertional hyponatremia is distinguished from heat illness by a normal core body temperature.^{21,31,40}

Exertional hyponatremia is caused by the inappropriate, excessive intake of free water before, during, and after endurance events. These athletes typically consume more fluid (usually water) than they lose in sweat and may gain weight over the course of an event.^{26,40} As distance athletes have become more educated about hydration, many athletes

may overhydrate, thinking they are providing good hydration to their body. As they race, they may begin to feel lethargic (nausea, malaise, vomiting) and misinterpret this to mean they are not well hydrated, thus prompting the intake of more fluid.

Risk factors for hyponatremia differ somewhat from those for heat stroke (see Table 2). These athletes are typically female, have slower race times, lower body weights, and have a high availability of fluids. Severe hyponatremia (serum sodium <120 mmol/L) can precipitate seizures, coma, and death. Treatment of the condition is beyond the scope of our article, but often begins with oral sodium solutions if mild and progresses to intravenous hypertonic saline for severe cases.

TREATMENT

Treatment protocols for heat illness follow a critical common theme—lower the core body temperature to an acceptable level (37.5–38°C) as quickly as possible. A major determinant of outcome in heat stroke is the duration of hyperthermia. The human critical thermal maximum is 41.6°C to 42°C for 45 minutes to 8 hours.⁹ Beyond this time frame, lethal or near-lethal injury occurs and is irreversible.

Treatment of all heat illness should begin with an assessment of airway, breathing, and circulation (ABCs), and transfer of the patient to a cooler environment. With exertional heat illness, this may be as simple as taking a player off the field to sit still on the bench or bringing the athlete to a shaded area. Most beneficial, of course, would be to move the patient to an air-conditioned building if available at the time of evaluation. These treatments should be universally employed in the setting of heat illness.

Heat Edema

Edema of the hands and feet should be mild and improve with elevation and relative rest. Compressive stockings may be helpful in cases that are slow to resolve. Ensuring that the athlete is well hydrated and has adequate salt intake is important as these conditions may delay resolution. Diuretics are not helpful as they further reduce intravascular volume and can exacerbate the condition. Generally, this condition improves in 7 to 14 days as acclimatization occurs or sooner if the athlete returns to his or her home climate.

Heat Rash

Cooling the area and reducing clothing coverage where possible will help resolution. The rash is benign but often takes a week or more to resolve completely.⁴⁰ Application of a mild anti-inflammatory lotion such as desonide may relieve symptoms and shorten the duration of the rash.²⁵

Heat Syncope

The treatment of heat syncope involves safely moving the patient into a supine position in a cool location. Often this

alone will resolve the condition as cerebral blood flow is restored. Elevating the patient's legs will aid in venous return of blood flow. Intravenous fluids may be necessary to correct volume depletion that likely contributed to the syncopal event.^{12,40}

Heat Cramps

Stretching of affected muscles, cooling with ice, massage of cramped muscles, and removal from activity are generally effective. Oral replenishment of fluid and electrolytes must be initiated for prevention of subsequent cramping but are generally not effective acutely for treatment. In severe cases or when the symptoms continue to rebound, intravenous hydration with 0.9% normal saline is indicated. This is often rapidly curative.⁴⁰

Heat Exhaustion

An athlete with heat exhaustion often presents with several concomitant symptoms. It is important to consider heat illness in the athlete who complains of nausea, vomiting, headache, or dizziness. Left untreated, this condition can progress to heat stroke. Core temperature readings, ideally with a rectal thermometer, are necessary to accurately identify athletes at risk of permanent injury and need for higher levels of care. If an athlete has mild illness and normal vital signs, cooling the athlete with removal from the heat and oral rehydration with cool salt-containing fluids (ie, sports drinks) will often be sufficient to lower temperature effectively. If there are more serious symptoms present such as abnormal vital signs, vomiting, or failure to improve with the above conservative techniques, intravenous fluids are indicated.³¹ Ice bags applied to the axilla and groin can also produce rapid lowering of body temperature and are recommended when repeat monitoring of core body temperature is available.

Heat Stroke

Heat stroke demands an aggressive approach to lowering body temperature. Direct correlation between duration of elevated temperature and morbidity/mortality of a patient has been established.³¹ In one case series of heat stroke occurrences, a trend was noted toward improved survival with cooling below 38.9°C core temperature within 60 minutes.^{31,44} Another report suggested improved survival if cooling to the same level occurs within 30 minutes.^{16,31} As previously stated, all treatment begins with an assessment of the ABCs, movement to a cooler location, and removal of clothing. This is unlikely to be effective alone in heat stroke, which requires more aggressive treatment.

There are many documented heat cooling techniques, but the level of effectiveness is controversial. In a comprehensive review of cooling techniques, it was demonstrated that immersion in ice water is the most effective method to produce total body cooling.⁴¹ There are obvious problems and limitations to this method. Unless heat illness is anticipated, ice water immersion baths and the personnel

required to monitor a patient in a bath are not readily available. If a patient is severely ill, immersion may limit the ability to monitor cardiovascular status and can be dangerous in the setting of reduced consciousness. Treatment with intravenous fluids can be difficult when the patient's body, other than the arm with the intravenous access, is immersed in the ice bath. Concern regarding peripheral vasoconstriction and slower cooling rates in patients immersed has not been proven experimentally.

Evaporative cooling by way of spraying cool to tepid water on a patient and facilitating evaporation and convection by use of a fan over bare skin has demonstrated superior cooling to other techniques in normal subjects.⁴¹ This has not been directly compared with the immersion technique in heat stroke patients.

Another method of cooling that has been used in military recruits involves laying patients over a cool water bath on a mesh stretcher while regularly dousing them with cool water and allowing a fan to evaporatively cool them. Concurrent massage with ice to large muscle groups is performed. The massage method is accomplished with generously sized bags of ice and focuses on the large muscle groups of the extremities, with repeated massage toward the core. Rectal temperatures are monitored with a probe that gives continuous readings. The method has been extrapolated for use at the Marine Corps Marathon in Washington, D.C. This method has yet to be directly compared with immersion or exclusive evaporation techniques.

Treatment of Heat Stroke Complications

Heat stroke may be complicated by seizure activity, hypotension, rhabdomyolysis, liver damage, and/or arrhythmias. Benzodiazepines are recommended for patients suffering from seizure activity. A short-acting benzodiazepine such as lorazepam will likely control the seizure. A starting dose of 2 to 4 mg is reasonable, with repeat doses every 10 to 15 minutes as needed up to 8 mg total for a 12-hour period. Ideally this is accomplished with a controlled airway. In many patients with hypotension, cooling alone will help blood pressure to rise to normotensive levels. Vasopressors may be needed if intravenous fluids alone do not correct hypotension, but should be used with caution because the catecholamines (ie, epinephrine, norepinephrine, dopamine) can lead to increased heat production. In patients with persistent hypotension not responsive to cooling and intravenous fluids, a catheter to measure central venous pressure (ie, Swan-Ganz) is indicated.³³ Rhabdomyolysis can also occur in severe cases of heat illness. Some sources recommend the use of diuretics (eg, mannitol at 0.25g/kg) and intravenous fluids to maintain renal blood flow and help prevent cellular destruction in the face of heat stroke.^{33,39} Myoglobinuria occurs due to destruction of muscle and, if present, maintaining a urine output of 50 mL/h is recommended. Liver damage can occur in severe cases of heat illness and may lead to coagulopathy and hepatitis. Acetaminophen use as an antipyretic should be avoided because it may worsen hepatic damage. Hypotension and cell death can lead to

heart muscle damage and arrhythmia.²² Therefore, it is important to monitor the cardiac status of all patients being treated for heat stroke. Many of the arrhythmias will resolve with cooling and, for this reason, electrical cardioversion should be avoided until the myocardium returns to normal temperature.³³

Field Treatment of Heat Stroke

On-field treatment of heat stroke requires common sense and use of available resources. The initial step in heat illness treatment is to recognize an athlete in trouble. Often teammates or coaches are made aware of athletes who are not feeling well before the medical staff is apprised. Early symptoms such as dizziness, nausea, malaise, and fatigue may not be reported to the medical staff by the athlete. The coaches and athletes should be taught the signs and symptoms of heat illness and be instructed to notify the medical staff if any exist.

Treatment for mild heat illness should be initiated as rapidly as possible to avoid progression to severe heat stroke (Table 4). Evaluation of the ABCs is the first critical step. In addition to moving the patient and removing equipment and clothing, ice packs to the axilla, groin and neck are often the most available resource and should be used. If the athlete is able to drink fluids, cool sports drinks or water should be encouraged. When available, a rectal temperature should be taken. Periodic questioning of the athlete to assess mental status changes will help alert the staff of a worsening condition. If the temperature is >104°F or if the mental status is unstable, elevation of the level of medical care is critical. Accessing the emergency medical system rapidly will allow for faster implementation of advanced care techniques, such as cooling with fans or using intravenous therapy.

Experimental Treatments of Heat Stroke

Dantrolene. Dantrolene impairs calcium release from the sarcoplasmic reticulum and thereby reduces muscle excitement and contractility.⁴¹ Its primary use is for malignant hyperthermia or neuroleptic malignant syndrome as it can reduce the spasticity of muscle seen in these conditions. It is hypothesized that muscle cramp or spasm seen in heat stroke may contribute to core temperature elevation. The use of dantrolene in heat stroke has been studied in 2 randomized trials. Although 1 trial showed benefit, it was criticized for flaws in the study design.¹¹ The second trial did not show any difference in cooling times, complications, length of stay in the hospital, or mortality.⁸ Dantrolene is not considered the standard of care for heat stroke at this time.

Activated Protein C. Heat stroke results in an inflammatory cascade and prothrombotic state. There is a tendency to progress to disseminated intravascular coagulation and organ ischemia. Activated protein C inhibits clotting factors necessary for thrombin formation. It has been used in cases of severe life-threatening sepsis with some success in improving outcomes. Promising results in a study using

TABLE 4
On-Field Treatment of Heat Stroke^a

—Recognize there is an athlete with signs or symptoms of heat illness.
—Initiate cooling methods: move to cool area, ice bags to groin/axilla/neck, ice water tub immersion, fan-sprayed mist.
—Assess for mental status changes.
—Assess need for rectal temperature (and repeat during cooling every 3-5 min).
—Encourage liberal oral fluid intake with cool sports drinks or water if able to tolerate.
—Check blood glucose and sodium levels if available.
Access EMS immediately if the athlete has any of the following:
Altered mental status
Temperature elevated >104°F
Persistent vomiting (unable to rehydrate orally)

^aEMS, emergency medical system.

activated protein C in rats with heat stroke¹² offers optimism that the agent may be helpful in humans, but no trials have been completed at this time.

PREVENTION AND RISK FACTORS

Education

Knowledge of the signs and symptoms of heat illness is important for athletes, parents, and coaches as well as medical staff. Early recognition of a problem and simple treatments initiated at the onset of symptoms may be life-saving measures. It has been argued that the most important precursor to heat illness is relative dehydration. Athletes should be offered ample amounts of water and salt-containing solutions, such as a sports drink, to hydrate during exertion. Nutritional supplements containing ephedra or other stimulants should be strongly discouraged. Athletes should be evaluated before competing with respiratory, gastrointestinal, or other febrile illness as these conditions have been shown to increase the risk of heat illness. If allowed to play, close monitoring is indicated. Maintenance of healthy body weights or, at the very least, more intense monitoring of obese patients may help prevent heat illness. Each individual athlete must acclimatize to the heat and attain an appropriate fitness level for the sport being played to prevent heat illness. The NCAA guidelines for acclimatization for football, the sport with the highest risk of heat illness, are listed in Table 5.^{6,37} Acclimatization typically takes 7 to 10 days. A gradual increase in exertion, environmental exposure time, and equipment wear is necessary to gain fitness and heat tolerance.

The American College of Sports Medicine (ACSM) has modified these guidelines slightly for adolescent athletes given the fact that adolescents are more susceptible to heat illness. Adolescents tend to begin their practice sessions underhydrated, ingest insufficient fluids during exertion, and take longer to acclimatize to hot conditions.⁶ In addition, they have a greater surface body area to body mass ratio than adults. This leads to greater heat gain

TABLE 5
Acclimatization Guidelines for Football^{6,37a}

NCAA Guidelines:
5-day acclimatization period at the beginning of the season—restricted to no more than 1 practice session a day lasting <3 hours
Helmet wear only for days 1, 2
Helmet plus shoulder pads only days 3, 4
Full equipment on day 5
After day 5, multipractice days are allowed with specific guidelines
—the total practice time per day must be <5 hours
—a single practice may not last longer than 3 hours
—at least 3 hours of rest between practices must occur
—the multipractice days must not occur on consecutive days
ACSM Guidelines: ^b
6-day acclimatization period at the beginning of the season—no more than 1 practice lasting <3 hours during this time
—Days 1, 2: helmet only
—Days 3-5: helmet and shoulder pads only
—Day 6: full equipment
—No contact drills during acclimatization period
—Limit consecutive practice days to 6
—Day 8: multiple practice sessions with same restrictions as above

^aNCAA, National Collegiate Athletic Association; ACSM, American College of Sports Medicine.

^bDiffer slightly from NCAA Guidelines, for high school athletes.

from the environment on a hot day. Children and adolescents have a lower sweating capacity than adults and produce more metabolic heat per unit of mass during physical activities (walking or running).¹ The acclimatization modifications for adolescents are described in Table 5. It should be noted that these guidelines have not yet been validated to be effective at reducing the number of athletes with heat illness.

Evaporative heat loss is critical to thermoregulation for the athlete. Uniform wear contributes to the formula of overall heat tolerance for an individual by decreasing the amount of skin surface available for evaporation.²⁹ Clothing should be light-colored, loose-fitting, and made from a lightweight open-weave material.¹³ Allowing time for acclimatization to heat conditions during preseason by limiting uniform wear, attention to the cardiovascular conditioning level of individual athletes, and awareness of environmental conditions are all important. Players should be offered frequent breaks during practice sessions and encouraged to seek shade and remove equipment to facilitate body cooling.¹⁵

A small population of college-age males was studied to evaluate the ability to tolerate various heat and humidity levels with differing degrees of uniform coverage. The results demonstrated the ability of an athlete to tolerate greater temperature and humidity while wearing fewer items of clothing, thus allowing for better evaporative heat loss. Intuitively, the study supported the theory that full football uniforms would cause a faster increase in core body temperature and thus reduce the critical heat balance limit. This information may be helpful to football coaches to determine the safety of dressing in full gear during hot, humid days.²⁹

TABLE 6
Fluid Management During Exertion:
Specific Guidelines^a

NCAA Sports Medicine Handbook ³⁶	8 to 16 oz of water 1 hour before exertion. Continue drinking during activity every 15-20 min. At the end of exercise, replace fluids lost (1 qt [32 oz] for every 2 pounds lost)
NATA	16-20 oz of fluid 2-3 hours before exertion. Just before exercise, take in another 6-10 oz. Take 6-10 oz every 15-20 min during exercise. After the event, consume fluid in excess of what was lost.

^aNCAA, National Collegiate Athletic Association; NATA, National Athletic Trainers Association.

Fluid Management

Dehydrated athletes are more likely to suffer heat illness. Mild dehydration (<2% body weight loss) occurs commonly in athletics and may be unavoidable. Dehydration levels can be approximated by weighing athletes before and after practices and competition. An athlete should be able to compete with a weight loss less than 3% of pre-exertion body weight.^{1,4} For weight loss greater than 3%, athletes should be restricted until body weight recovers with hydration. In athletes with weight loss greater than 3% dehydration, muscular strength and endurance decreases, plasma and blood volume decreases, cardiac output is compromised, thermoregulation is impaired, kidney blood flow and filtration decreases, liver glycogen stores decrease, and electrolytes are lost.³⁶ In contrast, body weight gains greater than 3% may predispose athletes to exertional hyponatremia from excessive water intake.

Prevention of dehydration is a reasonable goal for all who monitor athletes as well as the athletes themselves. Unfortunately, many athletes do not realize they are becoming dehydrated. Perhaps this is due to a lack of education or because of their intense concentration on the sport they are playing. In either case, a coach, athletic trainer, or parent may need to intervene to ensure that adequate hydration occurs. Ideally hydration starts before a practice session or game competition. Different sports organizations offer specific guidelines as to the amount of fluid needed (Table 6). We recommend taking 16 ounces of water or a sports drink 1 hour before exertion and continued hydration with 4 to 8 ounces of fluid every 15 to 20 minutes as long as exertion continues. If weight loss can be assessed after the event, replacement of 16 ounces of sports fluid for every pound lost is prudent. Teaching athletes to monitor their urine color and output may be prudent to assist in the process of hydration. The goal for the athlete is copious output of clear to light yellow urine.

Including salty foods in the diet may be helpful to athletes who are "salty sweaters" or have a history of a condition

along the continuum of heat illness.⁴⁰ College football players undergoing 2-a-day practices have been shown to have significant difficulty replacing their salt losses overnight.²⁴

Overweight/Obesity

Increased risk of heat illness in overweight or obese athletes or military members has been clearly demonstrated.^{18,30,45} Body mass index (BMI) is calculated to define weight classes for individuals. A BMI of 18.5 to 25 is normal; above 25 is considered overweight, and a BMI greater than 30 is obese.

Increased body mass leads to relatively low surface area necessary for heat dissipation via evaporation. The amount of metabolic heat production increases with body weight.⁴⁵ Overweight or obese athletes should be monitored closely for signs of heat illness.

Sickle Cell Trait

Hemoglobin S is an inherited type of hemoglobin that is unstable and can cause red blood cells (RBCs) to sickle during times of stress. A patient who inherits 2 hemoglobin S genes has sickle cell disorder. These patients are unlikely to participate in athletics because of their high likelihood of having painful crises related to the sickling of their RBCs during activity. Athletes are more commonly found to have 1 hemoglobin S gene and 1 normal (hemoglobin A) gene, a condition termed sickle cell trait. The incidence of this condition is about 8% in African Americans.⁴²

Athletes with sickle cell trait do not usually have painful crises at rest. During times of stress with exercise, however, they can be predisposed to sickling of their RBCs. Several reports of increased risk of sudden death in athletes with sickle cell trait have been reported.^{27,38} Some of these deaths have been related to exertional heat stroke. Dehydration, extreme heat, and exercise at high altitudes have been shown to be risk factors related to these events. We recommend evaluating African-American athletes for sickle cell trait when any condition along the spectrum of heat illness occurs or if there is a family history of sickle cell disorder or trait. Once diagnosed, it is clear that sickle cell trait individuals should be monitored closely to maintain hydration, especially in high heat or at altitude.³⁴

Heat Illness Symptom Index

The Heat Illness Symptom Index (HISI) has been proposed to identify mild forms of heat illness based on an athlete's subjective symptoms. This symptom-based index was recently validated in Division I football players in South Florida. The players were asked a series of questions related to symptoms they experienced during the current day's practice. The athletes' symptoms were shown to correlate proportionately with other known risks such as ambient temperature, level of dehydration (measured by weight changes), and perceived level of exertion. Unfortunately, core body temperature data on these athletes was not available, but an ongoing study is evaluating this risk factor and its

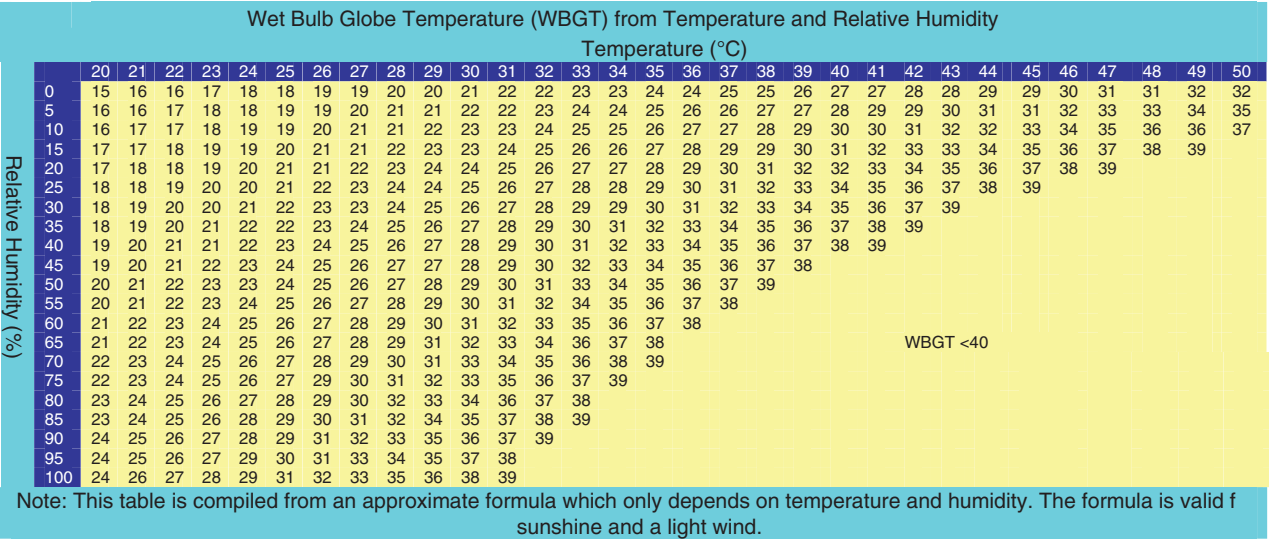


Figure 2. Wet Bulb Globe Temperature instrument. Courtesy of Australian Government Bureau of Meteorology; Richard de Dear, Macquarie University.



Figure 3. Wet bulb temperature measuring device. Photograph taken by SSG Paul R. Nieman, courtesy of Fort Drum Range Control.

correlation with HISI scores. No cases of heat stroke were identified during the study.¹⁴

Wet Bulb Globe Temperature

Environmental heat stress can most reliably be predicted by using the wet bulb globe temperature (WBGT) index (Figure 2). Variables measured are ambient heat, humidity, and radiant stress from direct sunlight. It is defined by the formula:

WBGT index = (0.7)T_{WB} + (0.2)T_{BG} + (0.1)T_{DB},

where T_{WB} is the wet bulb temperature, T_{BG} is the black globe temperature, and T_{DB} is the dry bulb temperature. The measuring device (Figure 3) is commercially available,

TABLE 7
Risk Categories in Wet Bulb Globe Temperature Readings

Risk Category	Temperature °F	Temperature °C
Low risk	<64°F	<18°C
Moderate risk	64-73°F	18-23°C
High risk	73-82°F	23-28°C
Hazardous risk	>82°F	>28°C

but difficult to use unless trained. Coaches, athletic directors, and athletic trainers can obtain WBGT readings from their local weather service during hot weather months.

Humidity plays the largest role in affecting the heat stress formula.³¹ The ACSM recommends canceling sporting events when the WBGT is above 28°C (82.4°F).² Risk categories in WBGT readings are as shown in Table 7.

Return to Play After Heat Illness

For mild forms of heat illness, proper hydration will allow an athlete to return to play within a 24-hour period. In the case of exertional heat stroke, further monitoring is warranted before returning to competition. A physician should evaluate any athlete with exertional heat stroke. Risk factors for heat stroke should be thoroughly addressed. Before returning to play, an athlete must be asymptomatic and all laboratory tests and vital signs should have normalized.³⁴ It is also prudent to monitor body weight until normalization. After treatment of the acute heat stroke event, it has been suggested that an athlete wait at least 1 week to return to play.^{3,34} The task force recommends a graduated and monitored return to exercise including progressive exposure to heat and level of sports equipment. Waiting for a period of 48 to 72 hours until return to duty has been

used in a military setting when the heat stroke was relatively mild (ie, rapid CNS recovery and normal laboratory testing). Each exertional heat stroke case must be considered independently. Overall, the severity of heat stroke illness should dictate the delay in return to play for an individual athlete.

CONCLUSION

Perhaps the most tragic fact surrounding heat stroke deaths in athletes is that the condition is entirely preventable. At the same time, the preventable nature of heat stroke offers the opportunity to prepare for these events and decrease the incidence.

Heat illness occurs along a spectrum that begins with relatively mild disease and can progress to life-threatening heat stroke. Recognition of heat illness and initiation of early treatment may prevent progression to heat stroke.

Heat stroke in athletes occurs as a result of intrinsic body heat production and impaired heat dissipation. It is commonly seen in hot and humid weather but has occurred in the setting of mild weather conditions. Diagnosis of heat stroke includes elevated core body temperature ($>104^{\circ}\text{F}$) and CNS dysfunction. Any athlete with CNS dysfunction during or after exertion should be evaluated for heat stroke even in the setting of core body temperature $<104^{\circ}\text{F}$ as cooling may have already begun. Paramount to the treatment of heat stroke is rapid temperature cooling and access to higher levels of care via the emergency medical system.

Proper education of coaches and athletes, identification of high-risk athletes, concentration on preventative hydration and acclimatization techniques, and appropriate monitoring of athletes for heat-related events are important ways to prevent heat stroke.

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