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Exertional heat illness in adolescents and adults: Epidemiology, thermoregulation, risk factors, and diagnosis

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INTRODUCTION

Exertional heat illness (EHI) is among the leading causes of death in young athletes each year [1,2]. A report by the United States Centers for Disease Control (CDC) found that EHI occurs both during practice and competition and noted a disturbing trend of increasing incidence [3]. Clinicians who care for athletes, both young and old, and others who exert themselves in the heat (eg, firefighters, soldiers, construction workers) need to be aware of the basic physiologic principles of thermoregulation, the spectrum of heat illness, strategies for prevention and treatment, and current guidelines for determining safe return to play or work.

The process of thermoregulation and the epidemiology, clinical presentation, and diagnosis of the different types of exertional heat illness, including exertional heat stroke, are reviewed here. The management of exertional heat stroke and other forms of exertional heat illness is discussed separately, as are exercise-associated hyponatremia, nonexertional heat stroke, malignant hyperthermia and other causes of severe hyperthermia, and heat illness in children. (See "Exertional heat illness in adolescents and adults: Management and prevention" and "Exercise-associated hyponatremia" and "Severe nonexertional hyperthermia (classic heat stroke) in adults" and "Malignant hyperthermia: Diagnosis and

management of acute crisis" and "Neuroleptic malignant syndrome" and "Heat stroke in children" and "Heat illness (other than heat stroke) in children".)

EPIDEMIOLOGY

Exertional heat illness (EHI) is an ever present danger when athletes, military personnel, or laborers perform intense exercise in the heat. A table summarizing important functional, acquired, and congenital risk factors for EHI is provided (table 1).

Despite great progress educating athletes, coaches, and clinicians, deaths related to exertional heat stroke (EHS), the most severe form of EHI, appear to be on the rise [2]. Deaths from EHS were higher during the period from 2005 to 2009 than any other five year period over the past 35 years. The United States Centers for Disease Control report a weighted average of 9237 cases of EHI among high school athletes per year for the period 2005 to 2009 [3]. The United States military, despite a continued focus on prevention, reported an increase in exertional heat stroke cases (344) in 2014 compared with 2013 [4].

In the United States, the highest incidence of EHS is found among participants in American football, in whom the condition occurs at a rate of 4.5 cases per 100,000 athlete exposures. According to an annual survey of catastrophic American football injuries presented in 2008, 31 players have died from EHS since 1995 [2]. Most cases occurred during summer practice when players are less fit, and temperatures and humidity are often high. According to our data, only one death from EHS occurred among American collegiate football players between 2003 and 2011 during traditional August practices due to the adoption of heat acclimatization policies in 2003. However, problems persist in high school football and collegiate strength and conditioning training, during which a disproportionate number of deaths have occurred [5].

A comprehensive study of fatal episodes of EHS among military personnel provides further insight into important risk factors [6]. According to this study, the absence of appropriate medical triage and physical effort beyond the fitness capacities of the victim were found in all deaths. Training in extreme heat was also common.

Other studies emphasize the importance of adverse environmental conditions in heat illness. Data from large-scale endurance events, such as marathons, show a strong correlation between the severity of environmental conditions and the incidence of heat illness, especially EHS [7]. According to annual surveillance studies of the Falmouth (Massachusetts, USA) Road Race (7 mile/11 km event), elevations in the temperature and heat index correlate with an

increase in the incidence of exertional heat stroke [8]. A four-year study of collegiate American football players found the incidence of heat illness to be closely associated with rising wet bulb globe temperature (WBGT) and lack of heat acclimatization [9]. A study of high school football players reported similar results [10,11]. (See 'Wet bulb globe temperature (WBGT) and other heat indices' below.)

RISK FACTORS

According to several large reviews and reports, common risk factors for all types of exertional heat illness (EHI) include the following [1-3,12,13]:

- Strenuous exercise in high ambient temperature and humidity
- Lack of acclimatization (see 'Thermotolerance and acclimatization' below)
- Poor physical fitness
- Obesity
- Dehydration
- Acute illness
- External load, including clothing, equipment, and protective gear

A table summarizing important functional, acquired, and congenital risk factors for EHI is provided (table 1).

Congenital disorders include ectodermal dysplasia and anhidrosis, which impair the ability to sweat, thereby limiting thermotolerance. Other congenital disorders that may increase the risk for EHI include malignant hyperthermia and sickle cell trait. However, the mechanisms whereby these disorders increase risk remain unknown, and these traits may be surrogates for heretofore undetermined risk factors [14,15].

In addition to the risk factors listed above, a number of acquired factors, including infection, certain medications, and dietary supplements predispose to EHI [16-18]. Infection not only impairs function through fever, but may increase risk through systemic activation of cytokines, which appears to impair thermotolerance. Consecutive days in the heat has been demonstrated in both military and civilian populations to compromise exercise heat tolerance and increase the risk for EHI [19,20].

Drugs and dietary supplements may increase the risk for EHI through a number of mechanisms, including impaired sweating, cardiovascular disturbances (eg, peripheral vasoconstriction or impaired cardiovascular performance), increased heat production,

disturbances in water and electrolyte balance, and decreased perception of fatigue, which might hinder the voluntary termination of exercise. Drugs and supplements associated with an increased risk for EHI include but are not limited to [18,21,22]:

- Anticholinergic agents
- Antiepileptic agents
- Angiotensin-converting enzyme inhibitors
- Angiotensin II receptor blockers
- Antihistamines
- Decongestants
- Phenothiazines
- Tricyclic antidepressants
- Amphetamines
- Ergogenic stimulants (eg, ephedrine, dimethylamylamine)
- Lithium
- Diuretics
- Beta blockers
- Ethanol

Not all commonly available supplements are associated with an increased risk of heat illness, however, those with sympathomimetic properties can be problematic [23]. A systematic review of 10 studies concluded that there is no evidence to support the notion that creatine supplementation impairs an athlete's ability to dissipate heat or disturbs their fluid balance [23]. (See "Nutritional and non-medication supplements permitted for performance enhancement".)

Data from the United States military confirms the role of the risk factors for EHI listed above and has identified several other factors, including [7,24-26]:

- Asian/Pacific Islander ethnicity
- Raised in a temperate climate (ie, higher incidence of EHI among recruits from Northern states)
- Male gender

Nevertheless, approximately 50 percent of EHI cases during basic training occur in recruits without these risk factors [9,27]. According to observational data, an episode of EHI sustained during basic training does not increase the risk for further episodes [28]. Retrospective studies of military cases of EHI report an association between EHI and

increased long-term mortality from organ failure (kidney, heart, liver) [29]. However, such studies are limited in that the timing and types of treatment for EHS were not considered, nor were individual risk factors.

THERMOREGULATION IN THE HEAT

Regulation of body temperature — Body temperature is regulated in the preoptic nucleus of the anterior hypothalamus, which carefully maintains a core temperature of 37°C±1° (98.6˚F±1.8°). The pathophysiology of heat illness is discussed separately. (See "Severe nonexertional hyperthermia (classic heat stroke) in adults", section on 'Pathophysiology'.)

While the human body has remarkable resilience against cold, it can tolerate only minor temperature elevations above normal (4.5°C, 9°F) without developing systemic dysfunction, which ultimately leads to multiorgan failure and death if body temperature cannot be lowered. Accordingly, the human body has multiple mechanisms to dissipate heat [21,30,31]:

- **Evaporation** occurs when water vaporizes from the skin and respiratory tract. This is the body's most effective mechanism for dissipating excess heat and is the primary means for athletes exercising in hot environments.
- **Radiation** is the emission of electromagnetic heat waves. This energy transfer does not require direct contact or air motion.
- **Convection** is the transfer of heat to a gas or liquid moving over the body. Heat transfer occurs when the gas or liquid is colder than the body.
- **Conduction** is direct heat transfer to an adjacent, cooler object.

During exercise, the human body acts to dissipate the excess heat generated by skeletal muscle. This requires an intact cardiovascular system that uses blood to transfer heat from the body core to the skin, where the mechanisms for dissipating heat can take effect. During high heat loads, blood flow to the skin increases many fold. However, when the ambient temperature is higher than the body's core temperature, convection, conduction, and radiation are no longer effective.

Environmental conditions also effect evaporative cooling. A water vapor pressure gradient must exist for sweat to evaporate and release heat into the environment. In high humidity (relative humidity >75 percent), evaporation becomes ineffective for transferring heat. Thus, in hot and humid conditions, athletes become susceptible to exertional heat illness.

Limitations on heat dissipation in hot and humid weather are exacerbated during intense exercise by a finite supply of blood that must fulfill multiple functions, including meeting the metabolic demands of active skeletal muscle and transporting heat to the skin surface for cooling. Further complicating matters is the dehydration that develops in most individuals during intense exercise in the heat, which decreases plasma volume.

Studies suggest that during intense exercise in the heat, for every one percent of body mass lost from dehydration, there is a concomitant increase in core body temperature of 0.22°C (0.4°F) [32-36]. In other words, other factors being equal, an athlete who lost only 1 percent of body mass from dehydration during intense exercise in the heat would be 1°C cooler compared to a teammate who lost 6 percent of body mass. This would equate to a temperature difference of approximately 39°C (102°F) versus 40°C (104°F) at the end of a training session.

A number of additional factors influence the rate at which a person's core body temperature rises during vigorous activity, including fitness level, degree of acclimatization to heat, clothing/equipment, and physiologic response (eg, degree of tachycardia) [34].

Compensated and uncompensated heat stress — Heat stress refers to the environmental and host conditions that increase body temperature. Heat stress is further categorized as compensated or uncompensated. Heat strain is the physiological and psychological consequence of heat stress. Severe heat strain is associated with a decline in athletic performance and increases the risk for EHI [21,37,38].

During exercise, the body elevates its temperature in response to the increase in metabolic heat production; a modest rise in temperature is thought to represent a favorable adjustment that optimizes physiologic functions [30]. With compensated heat stress (CHS), the body achieves a new steady-state core temperature that is proportional to the increased metabolic rate and available means for dissipating heat. Studies in runners describe exercise-induced hyperthermia, including athletes completing events successfully with significantly elevated core temperatures [39,40].

Uncompensated heat stress (UCHS) results when cooling capacity is exceeded and the athlete cannot maintain a steady temperature. Continued exertion in the setting of UCHS increases heat retention, causing a progressive rise in core body temperature and increasing the risk for severe heat illness [38,41].

Thermotolerance and acclimatization — Tolerance of extreme heat and humidity depends upon a number of functional, acquired, and congenital factors, of which acclimatization is of

great importance [30,42-44]. (See 'Risk factors' above.)

Acclimatization is the body's ability to improve its response and tolerance of heat stress over time, and it is the most important factor determining how well an athlete withstands extreme heat. Thus, allowing sufficient time and using optimal training strategies that enable athletes to acclimatize is critical for improving performance and mitigating the risk for EHI. Observational studies have found that the first week of athletic practice in high heat and humidity is the period of greatest risk for developing EHI [3,41,45]. Acclimatization requires at least one to two weeks. However, any improved tolerance of heat stress generally dissipates within two to three weeks of returning to a more temperate environment [21,41]. The attached tables provide guidelines for acclimatization (table 2 and table 3).

The major physiologic adjustments that occur during heat and humidity acclimatization include [41]:

- Plasma volume expansion
- Improved cutaneous blood flow
- Lower threshold for initiation of sweating
- Increased sweat output
- Lower salt concentration in sweat
- Lower skin and core temperatures for a standard exercise

These adaptations allow for better dissipation of heat during exercise and limit increases in body temperature compared to athletes who have not acclimatized.

DETERMINING RISK

It is important to consider environmental and individual factors when assessing the risk for environmental heat illness (EHI). A method for assessing the environment is described here, while individual risk factors are described above. (See 'Risk factors' above.)

Wet bulb globe temperature (WBGT) and other heat indices — One clinical tool commonly used to determine the overall environmental heat load is the wet bulb globe temperature (WBGT). This index was developed by the military to calculate heat stress, thereby enabling commanders to make adjustments in physical activity and fluid requirements in order to maximize performance [41]. The index is employed in the civilian setting to adjust athletic workload (eg, work-to-rest ratios, intensity of exercise, equipment, hydration breaks). In extreme circumstances, the WBGT may serve as the basis for cancelling activities (table 4)

[46]. (See "Exertional heat illness in adolescents and adults: Management and prevention", section on 'Prevention of exertional heat illness'.)

The WBGT integrates radiant heat, ambient temperature, and relative humidity. Equipment and instructions for performance have been published. We strongly prefer the WBGT over the heat index for determining the risk for EHI. The WBGT is calculated as follows:

WBGT = 0.1 x Dry Bulb Temperature (DBT) + 0.7 x Wet Bulb Temperature (WBT) + 0.2 x Globe Temperature (GT)

DBT represents the ambient air temperature, WBT the relative humidity, and GT the radiant heat. The equation for the WBGT reflects the critical importance of evaporative cooling for managing heat stress, as judged by the relative weight given to WBT. Measurements to determine the WBGT should be obtained about three to four feet off the ground on the playing field where the training session or sporting event will take place.

Given the close association between WBGT and exertional heat illnesses, this measurement should be used to guide and modify the intensity and duration of exercise, the use of equipment (eg, football helmets and padding), the frequency of rest breaks, and hydration needs (table 4). Any person or group responsible for these kinds of decisions should establish an accurate method for determining WBGT on site and should not rely upon local weather stations or news reports. However, it is acceptable to use WBGT measurements performed regularly by experts within close proximity (approximately 10 miles or 16 km) of the site of athletic activity (eg, WBGT calculated daily by local airport meteorologists).

The specific WBGT that would warrant modifications and the types of modifications needed vary by region, event, and individual [47]. As an example, the same WBGT might result in minor changes for a fit, well-hydrated individual engaged in light exercise who lives year-round in a semitropical climate but dramatic modifications for an obese adolescent American football player who lives in a temperate climate. Thus, detailed guidelines for modifying activity based upon the WBGT are beyond the scope of this review. A table from the Australian Bureau of Meteorology that provides an approximate indication of risk is provided (figure 1). When knowledgeable personnel are not available, it is best to err on the side of mandating more severe activity restrictions.

As noted, we prefer the WBGT to determine the risk for EHI (table 4); tables showing the heat index are included should they be needed for reference (table 5 and table 6). Other measurement tools to assess the risk for heat-related illness, such as the physiological equivalent temperature (PET), are being studied, but further research is needed to determine

their utility [48].

EXERTIONAL HEAT ILLNESS: BASIC TYPES AND THEIR CLINICAL PRESENTATION

Terminology — The classification of heat illness is controversial, as the precise pathophysiology of these disorders remains largely unknown. Accordingly, experts disagree about the general categories of illness, as well as the temperature and symptoms needed to define specific heat illnesses [18,49,50].

The International Classification of Diseases (ICD) published by the World Health Organization (WHO) offers one reasonable method for categorizing the various forms of exertional heat illnesses [51]. The ICD contains 10 categories of heat disorders. Four of these diagnoses (heat cramps, heat syncope, heat exhaustion, and heat stroke), as well as heat injury, are the most common entities found in athletes and others who engage in vigorous activity in the heat (eg, soldiers, laborers). Although not recognized by the WHO, heat injury is a term used by the United States military to describe a condition that falls between heat exhaustion and heat stroke. These relatively common entities are described below.

"Heat cramps" (exercise associated muscle cramps)

Definition, clinical presentation, and risk factors — Although muscle cramps are common in athletes, their etiology and pathophysiology remain poorly understood. The term "heat cramps" is a misnomer, as heat has not been shown to directly trigger cramping. Nevertheless, nearly all cases of cramping in athletes involve exercise at a high intensity or to exhaustion. Muscle cramps occur more often when athletes perform strenuous exercise in the heat, but they can also occur in cooler environments (eg, ice hockey, swimming). Muscle cramps are also more common in athletes engaged in novel or rarely performed exercise regimens. The treatment of heat cramps is discussed separately. (See "Exertional heat illness in adolescents and adults: Management and prevention", section on '"Heat cramps"'.)

A number of factors are thought to contribute to the development of muscle cramps in athletes [14]. Dehydration, loss of sodium and/or potassium, extreme environmental conditions, and neurogenic fatigue are suspected to play a role [14,52].

As a result of the controversy surrounding the etiology of muscle cramps, sports medicine researchers often refer to cramping that occurs during or after exercise as "exercise associated muscle cramping" (EAMC) [53]. Clinical criteria for establishing the diagnosis of

muscle cramps generally include intense muscle pain (not associated with acute muscle strain or other injury) and spasm, and persistent contractions of the muscles primarily involved in the prolonged exercise. No signs of more severe illness, such as exertional hyponatremia or exertional heat stroke, may be present.

Factors that are thought to predispose to EAMC include [14,53,54]:

- Sweat with high salt concentration (ie, "salty sweaters") [55,56]
- Heavy sweating
- Dehydration
- Insufficient sodium intake prior to and during intense activity
- Lack of heat acclimatization
- Baseline (preactivity) fatigue
- History of heat cramps

Differential diagnosis — It is important to note that muscle cramps are not necessarily related to exercise. The differential diagnosis is extensive and includes medications (eg, diuretics), myopathies, and endocrine disorders. Another possible cause is sickle cell trait, which is thought to have played a role in several cases of exertional sudden death and severe rhabdomyolysis [57]. In some of these cases, reports describe antecedent cramping following brief periods of intense exercise characterized by intense pain and distinguishable from EAMC-related symptoms by the lack of spasm, suggesting the possibility of acute muscle ischemia [16,17]. (See "Myopathies of systemic disease" and "Sickle cell trait", section on 'Rhabdomyolysis and sudden death during strenuous physical activity'.)

Heat syncope and exercise associated collapse

Definitions, clinical presentation, and pathophysiology — Heat syncope is among the more confusing diagnoses identified by the International Classification of Diseases. Like heat cramps, heat syncope is a misnomer as heat does not directly cause the syncopal event (ie, core body temperature is not significantly elevated). The treatment of heat-related syncope is discussed separately. (See "Exertional heat illness in adolescents and adults: Management and prevention", section on 'Heat syncope and exercise associated collapse'.)

The syncopal event that occurs in the exercising athlete is more appropriately termed "exercise associated collapse" (EAC) [15]. EAC occurs when an athlete is unable to stand or walk as a result of lightheadedness or syncope. EAC usually occurs immediately after completing a race or workout and is commonly observed at endurance events (eg, marathon). The mechanism for collapse is an abrupt decrease in venous return once that

athlete completes the event. Given the typical degree of vasodilatation seen with prolonged exertion, the sudden loss of the pressure exerted by the skeletal muscles on the vasculature leads to a precipitous decline in venous return, as well as postural tone, causing the athlete to collapse.

Heat is an indirect contributor to EAC as the body is dually tasked to provide blood to exercising muscle and the periphery, to assist in thermoregulation. In typical EAC, the athlete's core temperature is either normal or only marginally elevated and any alterations in mental status quickly resolve (within approximately 15 to 20 minutes) with appropriate treatment. These features help to distinguish EAC from heat stroke.

Heat syncope in those who are not exercising can be described as a transient loss or near-loss of consciousness due to the indirect effects of high ambient temperature. Heat syncope occurs most often during the first few days that someone is exposed to high environmental temperatures, before acclimatization is complete. Two common scenarios for heat syncope are:

- Prolonged standing in the heat with little movement
- Sudden standing after prolonged sitting in the heat

The signs and symptoms associated with these forms of heat syncope include light-headedness, tunnel vision, pale and sweaty skin, and decreased pulse rate. Most often the core temperature is normal or only mildly elevated. Patients generally recover rapidly with appropriate treatment.

The pathophysiology for each of these nonexertional events is related to the body's competing needs for thermoregulation and maintaining adequate blood pressure for an upright posture [41,58]. Thermoregulation requires increasing blood flow to the periphery through vasodilation in order to facilitate sweating. This increase in peripheral vasodilation can lead to peripheral pooling of blood, causing postural hypotension. Acclimatization eventually results in an increased circulating blood volume capable of accommodating both sweating and activity in the heat. In each scenario, the severity of illness is proportional to the rise in body temperature and the degree of dehydration.

Differential diagnosis — Particularly in older athletes and those with preexisting cardiac disease, all forms of heat-related syncope must be distinguished from general causes unrelated to exercise, including cardiac arrhythmia. The approach to the collapsed athlete and the differential diagnosis and management of syncope generally are reviewed

separately. (See "Evaluation of the collapsed adult athlete" and "Approach to the adult patient with syncope in the emergency department" and "Syncope in adults: Clinical manifestations and initial diagnostic evaluation".)

Heat exhaustion — Heat exhaustion is characterized by the inability to maintain adequate cardiac output due to strenuous physical exercise and environmental heat stress [50,59]. Acute dehydration may be present, but is not required for the diagnosis. The treatment of heat exhaustion is discussed separately. (See "Exertional heat illness in adolescents and adults: Management and prevention", section on 'Heat exhaustion'.)

The clinical criteria for heat exhaustion generally include the following:

- Athlete has obvious difficulty continuing with exercise
- Core body temperature is usually 101 to 104°F (38.3 to 40.0°C) at the time of collapse
- No significant dysfunction of the central nervous system (eg, seizure, altered consciousness, persistent delirium) is present

If any central nervous system dysfunction develops (eg, mild confusion), it is mild and resolves quickly with rest and cooling.

Patients with heat exhaustion may also manifest:

- Tachycardia and hypotension
- Extreme weakness
- Dehydration and electrolyte losses
- Ataxia and coordination problems, syncope, light-headedness
- Profuse sweating, pallor, "prickly heat" sensations
- Headache
- Abdominal cramps, nausea, vomiting, diarrhea
- Persistent muscle cramps

It is important to note that during exercise free water losses exceed electrolyte losses, leading to elevated serum sodium concentrations, unless these losses are replaced. However, sodium concentrations in sweat vary widely among athletes and there may be a subset with high concentrations (so-called "salty sweaters") [56,60]. Some researchers speculate that the carrier trait for cystic fibrosis may lead to higher sodium concentrations in sweat, but this has not been clearly established [61]. (See "Etiology and evaluation of hypernatremia in adults", section on 'Unreplaced water losses' and "Cystic fibrosis: Genetics and pathogenesis".)

Heat injury — Heat injury is defined as an exertional heat illness with evidence of both hyperthermia and end organ damage, but without any significant neurologic manifestations [21]. The absence of neurologic findings distinguishes the diagnosis from exertional heat stroke. The treatment of heat injury is discussed separately. (See "Exertional heat illness in adolescents and adults: Management and prevention", section on 'Heat injury'.)

Organs commonly damaged with heat injury include the muscles, kidneys, and liver; clinical and laboratory manifestations of metabolic acidosis, rhabdomyolysis, acute kidney injury, and liver failure are often seen.

The diagnosis of heat injury is primarily based upon a history of collapse during strenuous activity, a core temperature above 104 to 105°F (40 to 40.5°C), and the absence of neurologic findings. Any alteration in mental function suggests the diagnosis of exertional heat stroke. (See 'Exertional heat stroke' below.)

As noted earlier, exertional heat injury is not officially recognized as a heat illness by the World Health Organization. This term was created by United States military physicians to classify soldiers manifesting signs of severe heat-related injury (ie, more severe than heat exhaustion) but without significant CNS dysfunction, thus precluding use of the term heat stroke [62].

Exertional heat stroke — Heat stroke is a multisystem illness characterized by central nervous system (CNS) dysfunction (encephalopathy) and additional organ and tissue damage (eg, acute kidney injury, liver injury, rhabdomyolysis) in association with high body temperatures. Nonexertional heat stroke is reviewed in detail separately; exertional heat stroke in healthy adults and older adolescents is described here. (See "Severe nonexertional hyperthermia (classic heat stroke) in adults" and "Heat stroke in children".)

The two main criteria for diagnosing exertional heat stroke (EHS) are a core temperature **above** 104°F (40°C), measured immediately following collapse during strenuous activity, and CNS dysfunction [18,21,63]. CNS dysfunction can manifest as a wide range of possible symptoms and signs, including: disorientation, headache, irrational behavior, irritability, emotional instability, confusion, altered consciousness, coma, or seizure.

Other clinical findings vary. Most patients are tachycardic and hypotensive. Symptoms and signs that may be present include hyperventilation, dizziness, nausea, vomiting, diarrhea, weakness, profuse sweating, dehydration, dry mouth, thirst, muscle cramps, loss of muscle function, and ataxia. Some texts describe the absence of sweating with heat stroke but this is incorrect.

The morbidity and mortality due to EHS are a direct result of ischemia and oxidative and nitrosative stress; the prognosis is worse when cooling is delayed and the core temperature is allowed to remain above the critical threshold of 40.5 to 41.0°C (105 to 106°F) for any period of time [2,64-66]. The treatment of EHS is discussed separately. (See "Exertional heat illness in adolescents and adults: Management and prevention", section on 'Management of exertional heat stroke'.)

The majority of deaths from EHS among athletes occur primarily in two settings: high school American football practices and strength and conditioning workouts. (See 'Epidemiology' above.)

DIFFERENTIAL DIAGNOSIS FOR SEVERE EXERTIONAL HEAT ILLNESS

Athletes with potentially severe exertional heat illness (EHI), including heat stroke and heat injury, most commonly present with collapse. However, assessing a collapsed athlete, whether at the field or clinic, can be difficult due to the broad differential diagnosis and the inability to obtain a clear history in many cases. While a comprehensive approach to the collapsed athlete is beyond the scope of this review, the clinician should consider several critical diagnoses in the collapsed athlete or worker presumed to have EHI. These include exertional hyponatremia, malignant hyperthermia, and cardiac arrest, all of which must be recognized and treated quickly to avoid terrible consequences [15,67].

Exertional hyponatremia often occurs in endurance athletes, who may be normothermic, and present with cognitive changes, possibly including seizure [68]. These athletes are typically fluid overloaded, causing a dilutional hyponatremia. Early detection of a low serum sodium concentration and administration of hypertonic (3 percent) saline can prevent a catastrophe [13,69]. Treatment of exertional hyponatremia is discussed separately. (See "Exercise-associated hyponatremia", section on 'Treatment'.)

Individuals susceptible to malignant hyperthermia have abnormal skeletal muscle receptors and calcium channels that can lead to marked temperature elevations [12]. This occurs most often in the operating room following exposure to halogenated anesthetics and/or succinylcholine. However, some researchers postulate that this disorder may manifest with extreme exercise. Individuals with malignant hyperthermia typically are not flaccid, but rather rigid and spastic, which helps to distinguish them from heat stroke victims. Early recognition and treatment with dantrolene can be lifesaving. (See "Malignant hyperthermia: Diagnosis and management of acute crisis" and "Susceptibility to malignant hyperthermia:

Evaluation and management".)

Cardiac arrest is a rare cause of collapse among healthy athletes; EHI is far more common [70,71]. Among younger athletes, hypertrophic cardiomyopathy is the leading cause of cardiac arrest, while acute coronary syndrome is the leading cause among older athletes. Of note, the athlete with severe EHI may present in cardiac arrest. Therefore, the rectal temperature is a critical vital sign in the collapsed athlete. (See "Athletes: Overview of sudden cardiac death risk and sport participation" and "Hypertrophic cardiomyopathy: Clinical manifestations, diagnosis, and evaluation" and "Advanced cardiac life support (ACLS) in adults" and "Initial evaluation and management of suspected acute coronary syndrome (myocardial infarction, unstable angina) in the emergency department".)

SOCIETY GUIDELINE LINKS

Links to society and government-sponsored guidelines from selected countries and regions around the world are provided separately. (See "Society guideline links: Exertional heat illness".)

SUMMARY AND RECOMMENDATIONS

- **Treatment of exertional heat illness (EHI)** – The management of exertional heat stroke (EHS) and other forms of EHI is discussed separately. (See "Exertional heat illness in adolescents and adults: Management and prevention".)

- **Risk factors** – EHI is an ever-present danger when athletes or workers perform intense exercise in the heat. A table summarizing important functional, acquired, and congenital risk factors for EHI is provided (table 1). Important risk factors include high ambient temperature and humidity, lack of acclimatization, dehydration, and poor physical fitness. A number of drugs and supplements, including alcohol and stimulants, increase the risk of EHI and are listed in the text. (See 'Epidemiology' above and 'Risk factors' above.)

- **Body temperature regulation** – High heat and humidity impair the body's capacity for dissipating heat, which is accomplished primarily through evaporation but also involves convection, conduction, and radiation. (See 'Regulation of body temperature' above.)

- **Acclimatization** – Acclimatization is the body's ability to improve its response and

tolerance of heat stress over time, and it is the most important factor determining how well an athlete can withstand extreme heat and humidity. General principles for heat acclimatization are listed in the accompanying table and described in the text (table 2). (See 'Thermotolerance and acclimatization' above.)

- **Heat indices** – The wet bulb globe temperature (WBGT) is an important index for determining the environmental risk for heat illness and the need to modify activity. The specific WBGT that would warrant modifications and the types of modifications needed vary by region and individual (table 4). (See 'Wet bulb globe temperature (WBGT) and other heat indices' above.)

- **Exercise-associated collapse (EAC)** – The syncopal event that occurs in the exercising athlete is more appropriately termed EAC. EAC occurs when an athlete is unable to stand or walk and usually occurs immediately after completing an endurance race or workout. (See 'Heat syncope and exercise associated collapse' above.)

- **Heat exhaustion** – Heat exhaustion is characterized by the inability to maintain adequate cardiac output due to strenuous physical exercise and environmental heat stress. Acute dehydration may be present but is not required for the diagnosis. The clinical criteria for heat exhaustion generally include the following:

  - Athlete has obvious difficulty continuing with exercise

  - Body temperature is usually 101 to 104ºF (38.3 to 40.0°C) at the time of collapse

  - No significant dysfunction of the central nervous system (eg, seizure, altered consciousness, persistent delirium) is present (see 'Heat exhaustion' above)

- **Heat injury** – Heat injury is defined as an EHI with evidence of both hyperthermia (core temperature above 40 to 40.5°C) and end organ damage, but without any significant neurologic manifestations. Clinical and laboratory signs of metabolic acidosis, rhabdomyolysis, acute kidney injury, and/or liver failure are often seen. The absence of neurologic findings distinguishes the diagnosis from EHS. (See 'Heat injury' above.)

- **Exertional heat stroke (EHS; most dangerous type)** – EHS is a multisystem, life-threatening illness characterized by central nervous system (CNS) dysfunction (encephalopathy) and additional organ and tissue damage (eg, acute kidney injury, liver injury, rhabdomyolysis) in association with high body temperatures. The two main diagnostic criteria are a core (eg, rectal) temperature above 40°C and CNS dysfunction. CNS dysfunction can manifest as a wide range of possible symptoms and signs,

including: disorientation, headache, irritability, emotional instability, confusion, altered consciousness, coma, or seizure. (See 'Exertional heat stroke' above.)

- **Differential diagnosis** – The differential diagnosis for an athlete or worker who is presumed to have collapsed from an EHI includes exertional hyponatremia, malignant hyperthermia, and cardiac arrest. (See 'Differential diagnosis for severe exertional heat illness' above.)

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Topic 13788 Version 15.0

GRAPHICS

Factors predisposing to heat intolerance among active adults

Functional:
Low physical fitness
Lack of acclimatization
Low work efficiency
Reduced skin area to mass ratio (eg, large muscle mass, obesity)
Acquired:
Use of alcohol prior to activity
Medications/supplements (eg, stimulants)
Dehydration
Viral or bacterial infection
Previous heat stroke
Sweat gland dysfunction
Large skin area with burn scars
X-ray radiation
Congenital:
Ectodermal dysplasia
Chronic idiopathic anhidrosis

Adapted from: Epstein Y. Heat intolerance: Predisposing factor or residual injury? Med Sci Sports Exerc. 1990; 22:29-35.

Graphic 74567 Version 1.0

Guidelines for optimizing heat acclimatization

Acclimatization should be performed gradually over 10-14 consecutive days	
Environment	Reflects the environment that will be encountered during goal activity
	Warm or hot (WBGT >26°C)
	Relative humidity reflects the humidity that will be encountered
	If necessary environmental conditions not present, add clothing or exercise during hottest/most humid part of the day
Exercise session	Duration: >60 minutes
	Type: aerobic
	Intensity: moderate intensity (should progress from 60-80 percent age-predicted maximum heart rate)
	Frequency: one session per day (may progress to two sessions per day alternating with a single session day)
Hydration	Urine specific gravity: <1.020
	Urine osmolality: <700
	Urine color: <3 (using 8 color scale)
Safety	Monitor temperature with rectal or gastrointestinal thermistor (<40°C)
	Monitor athlete for signs and symptoms of heat illness
	Monitor heart rate for increased intensity (>80 percent age-predicted maximum)
	Avoid interval training at race speeds (may be performed separately in a cooler environment)
	Calculate sweat rate: pre-exercise nude body mass (kg) - post-exercise nude body mass (kg) + fluid ingested (L) per exercise time (hour) = sweat rate (L/hour)
	Keep athlete well hydrated before and during exercise (<2 percent dehydrated)
	Training intensity should correspond with fitness level

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Sample heat acclimatization program for American football[1]

Modification	Practices 1-5		Practices 6-14
	Days 1-2	Days 3-5	
Number of practices permitted per day	1	1	2 (only every other day)
Equipment	Helmets only	Helmets & shoulder pads	Full equipment
Maximum duration of practice session	3 hours	3 hours	3 hours (maximum of 5 hours total on double session days)
Permitted walk through time (in addition to regular practice session)	1 hour (must be separated from regular practice by 3 hours)		1 hour (must be separated from regular practice by 3 hours)
Contact	No contact	Contact only with blocking sleds/dummies	Full contact drills

NOTE: Warm-up, stretching, cool-down, conditioning, and weight-room activities are all included as part of practice time.

Reference:

1. *Preseason Heat-Acclimatization Guidelines for Secondary School Athletics. Journal of Athletic Training 2009; 44:332.*

Graphic 82778 Version 4.0

WBGT activities chart

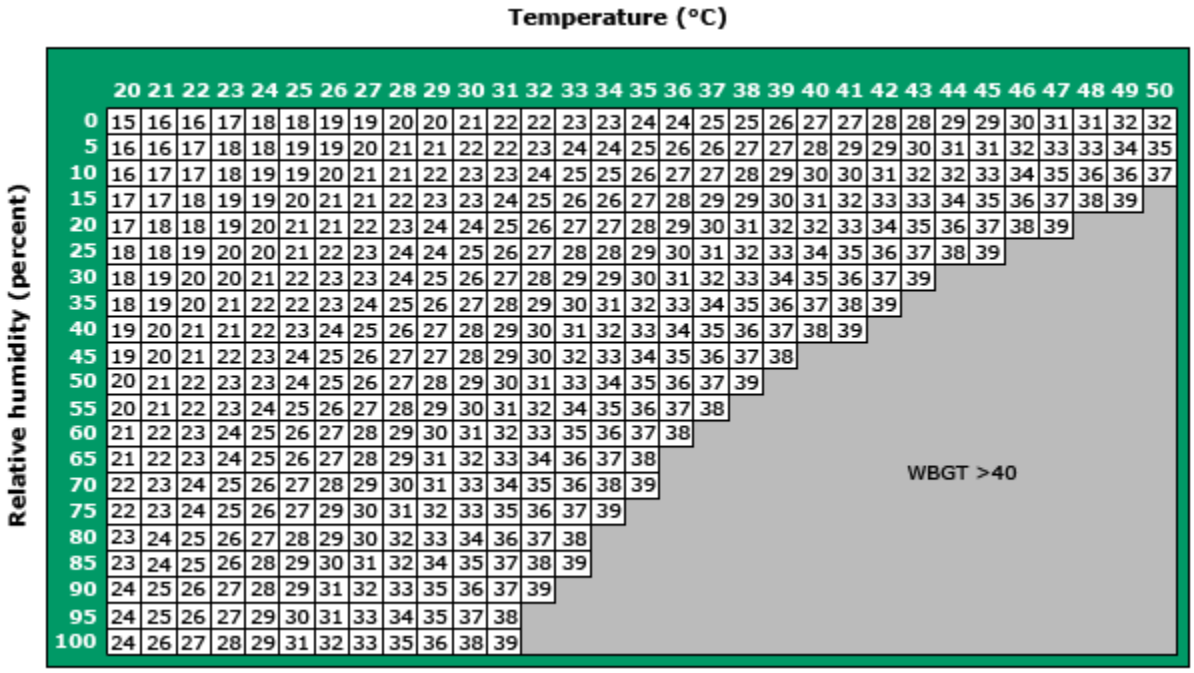
WBGT (°C)	WBGT (°F)	Risks and permitted activities		
		Intermittent activites		Continuous activities
		Non-acclimatized, unfit, or high-risk individuals	Acclimatized, fit, low-risk individuals	
18.4 to 22.2	65.1 to 72	Increase the rest:work ratio; monitor fluid intake	Normal activity	Risk of heat stress and other heat illnesses begin to rise; high-risk individuals should be monitored or not compete
22.3 to 25.5	72.1 to 78	Increase the rest:work ratio; decrease total duration of activity	Monitor fluid intake	Risk for all competitors is increased
25.6 to 27.7	78.1 to 82	Decrease intensity and total duration of activity	Monitor fluid intake	Risk of unfit, non-acclimatized individuals is high
27.8 to 30	82.1 to 86	Increase the rest:work ratio to 1:1; limit intense exercise; watch at-risk individuals carefully	Plan intense/prolonged activity with discretion. Watch at-risk individuals carefully	Cancel activity/competition
30.1 to 32.2	86.1 to 90	Cancel or stop practice and competition	Limit intense exercise and total exposure to heat and humidity; watch for early signs/symptoms of heat stress	
>32.2	>90	Cancel exercise	Cancel exercise; heat stress exists for all athletes	

WBGT: wet bulb globe temperature.

Based on guidance from the American College of Sports Medicine. Adapted from: Wet Bulb Globe Temperature. National Weather Service. Available at: https://www.weather.gov/arx/wbgt (Accessed on August 25, 2021).

Graphic 133128 Version 1.0

Wet Bulb Globe Temperature Chart



Relative humidity (percent)	20	21	22	23	24	25	26	27	28	29	30	31	32	33	34	35	36	37	38	39	40	41	42	43	44	45	46	47	48	49	50
0	15	16	16	17	18	18	19	19	20	20	21	22	22	23	23	24	24	25	25	26	27	27	28	28	29	29	30	31	31	32	32
5	16	16	17	18	18	19	19	20	21	21	22	22	23	24	24	25	26	26	27	27	28	29	29	30	31	31	32	33	33	34	35
10	16	17	17	18	19	19	20	21	21	22	23	23	24	25	25	26	27	27	28	29	30	30	31	32	32	33	34	35	36	36	37
15	17	17	18	19	19	20	21	21	22	23	23	24	25	26	26	27	28	29	29	30	31	32	33	33	34	35	36	37	38	39	
20	17	18	18	19	20	21	21	22	23	24	24	25	26	27	27	28	29	30	31	32	32	33	34	35	36	37	38	39			
25	18	18	19	20	20	21	22	23	24	24	25	26	27	28	28	29	30	31	32	33	34	35	36	37	38	39					
30	18	19	20	20	21	22	23	23	24	25	26	27	28	29	29	30	31	32	33	34	35	36	37	39							
35	18	19	20	21	22	22	23	24	25	26	27	28	29	30	31	32	33	34	35	36	37	38	39								
40	19	20	21	21	22	23	24	25	26	27	28	29	30	31	32	33	34	35	36	37	38	39									
45	19	20	21	22	23	24	25	26	27	27	28	29	30	32	33	34	35	36	37	38											
50	20	21	22	23	23	24	25	26	27	28	29	30	31	33	34	35	36	37	39												
55	20	21	22	23	24	25	26	27	28	29	30	31	32	34	35	36	37	38													
60	21	22	23	24	25	26	27	28	29	30	31	32	33	35	36	37	38														
65	21	22	23	24	25	26	27	28	29	31	32	33	34	36	37	38															
70	22	23	24	25	26	27	28	29	30	31	33	34	35	36	38	39															
75	22	23	24	25	26	27	29	30	31	32	33	35	36	37	39																
80	23	24	25	26	27	28	29	30	32	33	34	36	37	38																	
85	23	24	25	26	28	29	30	31	32	34	35	37	38	39																	
90	24	25	26	27	28	29	31	32	33	35	36	37	39																		
95	24	25	26	27	29	30	31	33	34	35	37	38																			
100	24	26	27	28	29	31	32	33	35	36	38	39																			

Reproduced from: Thermal Comfort Observations. Australian Government Bureau of Meteorology. Available at: http://www.bom.gov.au/info/thermal_stress/ (Accessed 6 March 2012).

Graphic 82777 Version 1.0

Heat index (Celsius)

Relative humidity (%)	Temperature (°C)															
	27	28	29	30	31	32	33	34	36	37	38	39	40	41	43	47
40	27	27	28	29	31	33	34	36	38	41	43	46	48	51	54	58
45	27	28	29	31	32	34	36	38	40	43	46	48	51	50	58	
50	27	28	29	31	33	35	37	39	42	45	48	51	55	58		
55	27	29	30	32	34	36	38	41	44	47	51	54	58			
60	28	29	31	33	35	38	41	43	47	51	54	58				
65	28	29	32	34	37	39	43	46	49	53	58					
70	28	30	32	35	38	41	46	48	52	57						
75	29	31	33	36	39	43	47	51	56							
80	29	32	34	38	41	45	49	54								
85	29	32	36	39	43	47	52	57								
90	30	33	37	41	45	50	55									
95	30	34	38	42	47	53										
100	31	35	39	44	49	56										

Likelihood of heat disorders with prolonged exposure or strenuous activity

Caution	Extreme caution	Danger	Extreme danger

Data from: Jacklitsch B. Heat Index: When humidity makes it feel hotter. NIOSH Science Blog. Centers for Disease Control and Prevention. Available at: https://blogs.cdc.gov/niosh-science-blog/2017/06/05/heat-index/ (Accessed on August 25, 2021).

Graphic 133127 Version 1.0

Heat index (Fahrenheit)

Relative humidity (%)	Temperature (°F)													
	80	82	84	86	88	90	92	94	96	98	100	102	104	106
40	80	81	83	85	88	91	94	97	101	105	109	114	119	124
45	80	82	84	87	89	93	96	100	104	109	114	119	124	130
50	81	83	85	88	91	95	99	103	108	113	118	124	131	137
55	81	84	86	89	93	97	101	106	112	117	124	130	137	
60	82	84	88	91	95	100	105	110	116	123	129	137		
65	82	85	89	93	98	103	108	114	121	128	136			
70	83	86	90	95	100	105	112	119	126	134				
75	84	88	92	97	103	109	116	124	132					
80	84	89	94	100	106	113	121	129						
85	85	90	96	102	110	117	126	135						
90	86	91	98	105	113	122	131							
95	86	93	100	108	117	127								
100	87	95	103	112	121	132								

Likelihood of heat disorders with prolonged exposure or strenuous activity

	Caution		Extreme caution		Danger		Extreme dan

Data from: Heat Forecast Tools. National Weather Service. Available at: https://www.weather.gov/safety/heat-index (Accessed on August 25, 2021).

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