ACSM Expert Consensus Statement on Exertional Heat Illness: Recognition, Management, and Return to Activity

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Abstract

Exertional heat stroke is a true medical emergency with potential for organ injury and death. This consensus statement emphasizes that optimal exertional heat illness management is promoted by a synchronized chain of survival that promotes rapid recognition and management, as well as communication between care teams. Health care providers should be confident in the definitions, etiologies, and nuances of exertional heat exhaustion, exertional heat injury, and exertional heat stroke. Identifying the athlete with suspected exertional heat stroke early in the course, stopping activity (body heat generation), and providing rapid total body cooling are essential for survival, and like any critical life-threatening situation (cardiac arrest, brain stroke, sepsis), time is tissue. Recovery from exertional heat stroke is variable and outcomes are likely related to the duration of severe hyperthermia. Most exertional heat illnesses can be prevented with the recognition and modification of well-described risk factors ideally addressed through leadership, policy, and on-site health care.

What Is the Clinical Problem?

Athletes, elite, recreational and tactical, and occupational laborers regularly perform stressful physical activities in warm to hot environments, sometimes wearing heavy equipment (*e.g.*, football players), protective clothing (*e.g.*, firefighters) or both (*e.g.*, warfighters). Heat stress impairs exercise performance and causes physiological strain that may evolve into exertional heat illness in a wide range of temperature conditions starting as low as 15°C (1). Based on data from the National Center for Catastrophic Sport Injury Research at the University of North Carolina at Chapel Hill, deaths in athletes from exertional heat stroke (EHS) have averaged 3 per year since 1995, mainly in high school football players (2,3). Despite educational and preventive efforts to lessen EHS morbidity and mortality, recent literature reveals little to no change in the annual number of EHS deaths among athletes (4). The prevalence of exertional heat illness across all sports is not known (2,5). The difficulty assessing the data and trends surrounding the epidemiology of exertional heat illness is partly explained by the number of cases that are not treated and documented in medical care facilities, and the inconsistent terminology and case definitions (6).

The incidence rate of exertional heat illness increases as ambient temperature and relative humidity rise during the warmer months of the year (7–11); this rate is predicted to increase as average world temperature and relative humidity continue to escalate with climate change (12). The increased prevalence of obesity, physical inactivity, low physical fitness, and lack of heat acclimatization may contribute to the increase incidence rate. However, other factors such as more frequent heat waves and suboptimal prevention strategies may be responsible (8,13–15). Many medical management issues related to the recognition, treatment, and recovery of exertional heat illnesses remain controversial (16,17).

A systematic review of 62 epidemiological studies reported the highest incidence of exertional heat illness in American football, running, cycling, and adventure races (6). Marathon running and triathlons report the highest number of hospitalizations due to the extended duration of vigorous exercise (6).

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Few sports are immune from exertional heat illness and examples of rates (per 100,000 athlete exposures) during training and competition in National Collegiate Athletic Association sports are: men's American football [15.5], wrestling [2.9], cross-country [4.8], basketball [4.1], soccer [3.1]; and women's cross-country [3.5], outdoor track & field [5.9], tennis [4.3], field hockey [0.20], and soccer [3.0] (10). High school rates also vary by sport (18), some examples include girl's field hockey [3.9], lacrosse [0.6], volleyball [0.3], soccer [1.1], cross county [2.8]; and boy's baseball [0.57] and soccer [0.51] (8,19). Boy's American football consistently has the highest rate of exertional heat illness, with Kerr et al. (8,19) reporting 11 times the rate (4.42 to 5.2) of all other high school sports combined. In military and occupational settings, Army and Marine Corps personnel and laborers in occupations with heat-exposed physical activity consistently have the highest rates of exertional heat illness (14,20,21).

This American College of Sports Medicine (ACSM) consensus statement replaces the position statement published in 2007 (22) with emerging practices for recognition, prevention, and management of exertional heat illness (16,17) and focuses on exertional heat exhaustion (EHE), exertional heat injury (EHI), and EHS. Additional conditions, such as exercise-associated muscle cramps, exertional rhabdomyolysis, exercise collapse associated with sickle cell trait, and exercise-associated hyponatremia are not included in this statement, although they are important to consider in the initial evaluation of a collapsed athlete. The consensus statement will identify evidence-based strategies to reduce morbidity and mortality of exertional heat illness including a staged return to activity for athletes recovering from an EHS event.

What Is Serious Exertional Heat Illness?

Serious exertional heat illness includes EHE, EHI, and EHS; whether these entities occur independently or on a spectrum has not been determined. EHI related to strenuous exercise and elevated body temperatures often presents with athlete collapse and can range from self-limited EHE to potentially life-threatening EHS (23–26). Any athlete or laborer presenting with a clinical picture suggesting a potentially life-threating exertional heat illness should be cooled until a more thorough evaluation can be completed.

EHE is defined as the inability to sustain the required cardiac output and blood pressure to continue physical activity due to high skin blood flow requirements and/or dehydration related to heat stress. Body temperature is elevated by the metabolic heat produced during exercise but is usually <40°C (26–28). Common signs and symptoms consistent with the etiology are listed in Table 1. EHE usually has no chronic health effects and after body cooling and restoring fluid-electrolyte balance most athletes and laborers can return to activity in 1 to 2 d. However, people with repeated episodes of EHE in a 24-month span should have a thorough medical evaluation (29).

EHS is defined as a life-threatening condition characterized by central nervous system (CNS) disturbances and hyperthermia, usually >40°C rectal temperature. The term "heat stroke" reflects the presence of focal "stroke-like" symptoms associated with warm environments and hyperthermia, although the symptoms in most victims are more global (encephalopathy) than focal (stroke syndrome). CNS changes associated with EHS vary from mild personality changes to the continuum of confusion, delirium, stupor, or unconsciousness. Altered mental status with loss of orientation to person, place, or time is common. Severe agitation with florid psychosis can occur, and some victims are verbally and physically aggressive with potential to injure caregivers. In addition to CNS dysfunction, EHS is usually associated with body temperature >40°C, along with signs and symptoms of cardiovascular and other organ system distress (see Table 1). Organ and tissue damage occur with prolonged hyperthermia, sometimes because of delayed recognition and cooling, but the damage may not be clinically evident until later in the disease process. Clinical management following EHS may require critical care interventions for organ and tissue damage induced by hyperthermia and sequalae, including systemic inflammatory response syndrome (SIRS) and disseminated intravascular coagulopathy (DIC) (23,25,30).

A presenting core temperature ≥40°C alone is not sufficient to establish the diagnosis of EHS (16,23,26,30). Core temperature values ≥40°C (exertional hyperthermia) have been documented during high-intensity physical activity in both warm and hot weather with no apparent adverse effects on performance or health (31–33).

Table 1.
Signs and symptoms of exertional heat illness that often resolve with rapid cooling.

Common Signs and Symptoms of Exertional Heat Illness	Signs and Symptoms Suggesting Exertional Heat Stroke
Dizziness	Persistent mental status changes
Headache	Personality changes (frontal lobe)
Nausea	Inappropriate behavior or aggressiveness
Unsteady walk	Delirium
Generalized weakness	High rectal temperature, >40°C (104°F)
Muscle cramps	Loss of ambulatory function (ataxia)
Fatigue	Flaccid muscles or persistent rigidity
Chills	Stool incontinence
Eyes closed	Seizure
Missing assigned tasks (cognitive function)	Coma
Sweaty skin (not dry), warm or cool to touch	Recurrent vomiting
Skin color varies from pale to flushed	
Weak or rapid pulse	
Tachycardia	
Systolic hypotension	

EHI is characterized by evidence of organ (*e.g.*, gastrointestinal, kidney, liver, muscle) damage and dysfunction in the presence of hyperthermia without CNS changes seen in EHS and requires laboratory testing to establish the diagnosis (23,25,30). In an exercise setting, EHI also can be a consequence of delayed recognition and/or inadequate cooling of EHS. The exact clinical pathway to tissue or organ injury is unknown but may be a result of dehydration and reduced blood flow during a more severe EHE episode or a direct thermal injury during an EHS episode in which CNS dysfunction was minor or missed. EHI may or may not be on a continuum as an intermediate condition between EHE and EHS. EHI typically causes tissue and organ dysfunction that may persist for several weeks (29) including acute kidney injury, transient diarrhea (gut injury), and/or transaminitis associated with liver injury. More severe EHI-associated EHS can result in liver or kidney failure requiring organ transplantation for patient survival.

What Is the Pathophysiology of Exertional Heat Illness?

During exercise in the heat, the primary physiological challenge is an increase in cardiac output to support both high skin blood flow for heat dissipation and high muscle blood flow for metabolism at the expense of compensatory reductions in renal and splanchnic blood flow (26,34). When these compensatory responses are insufficient; skin, muscle, and even brain blood flow are compromised affecting tissue metabolism and heat exchange (26,30,34). In addition, as ambient temperature increases, sweating increases and sweat evaporation becomes the primary heat transfer mechanism (35). If the high rates of sweating fluid loss are not replaced, the reduced plasma volume (from dehydration) further elevates physiological strain, impairing work capabilities and increasing the risk of exertional heat illness (26,30,34).

Figure 1 diagrams progression from exercise heat stress to exertional heat illness. The greater the heat stress the greater the physiological strain as evidenced by hyperthermia, blood pressure regulation challenges, reduced tissue perfusion, ischemia, and both elevated oxidative and nitrosative stress (26,34).

If the physiological strain is not excessive, multiple heat exposures will stimulate adaptations such as heat acclimatization (37) and acquired thermal (heat) tolerance (38) which help to improve performance in the heat and protect from exertional heat illness (26). The adaptive changes will induce molecular adaptions, including heat shock protein expression, that improve tissue/organ protection or thermal tolerance. If the physiological strain is excessive, it will induce pathological events including increased gut permeability, endotoxemia, exaggerated acute phase response and systemic inflammatory response syndrome (SIRS), coagulopathy (DIC), and cell death (26,30,39). In addition, reduced cerebral blood flow, combined with abnormal local metabolism and coagulopathy, can lead to dysfunction of the CNS. These perturbations induce changes associated with EHS and EHI. There is no evidence that EHI or EHS will induce abnormalities to hypothalamic regions, but thermoregulatory feedback loops may be damaged (16). Of particular concern is intestinal barrier damage accentuating endotoxin leakage and potentiating liver damage, endotoxemia, SIRS, and sepsis (30,39). The composition of an athlete's gut microbiome may predispose an EHS or EHI victim to endotoxemia and SIRS (40).

Preliminary research indicates there may be an association with EHS/EHI and long-term health issues. For example, EHS/EHI victims were reported to have a 3.9 times higher incidence of major cardiovascular events, a 5.5 times greater incidence of ischemic stroke, and a 15 times greater incidence of atrial fibrillation during a 14-year follow-up period (41,42). Similarly, a cohort mortality study of male and female U.S. Army personnel hospitalized for exertional heat illness with

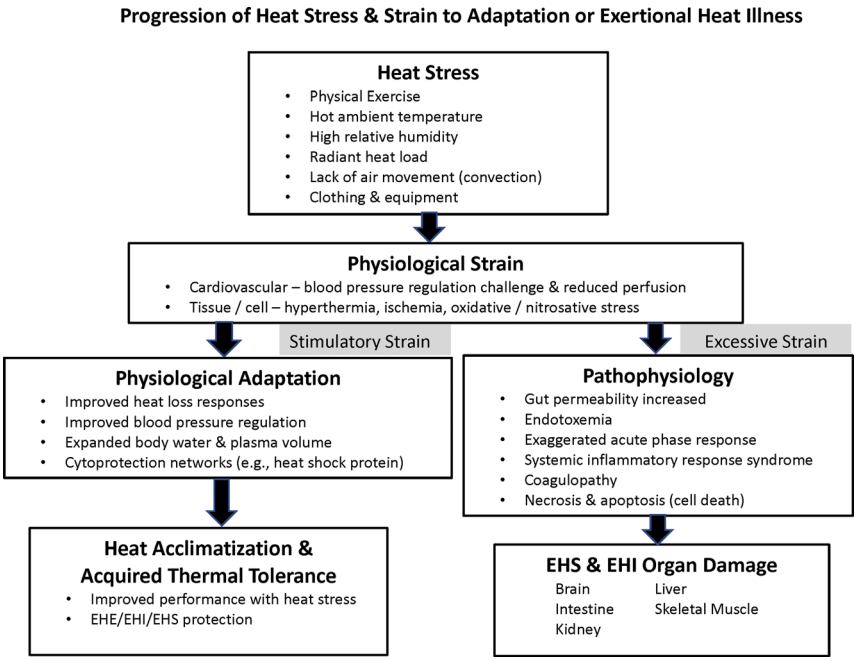

Figure 1: The impact of heat stress on physiological strain resulting in either adaptation or exertional heat illness (36).

an unknown duration of hyperthermia prior to cooling had a 40% increased long-term mortality risk when compared with hospital admissions for appendicitis as reference cases (42). Recent evidence from an animal model suggests that 30 d post-EHS epigenetic memory changes can suppress the immune system and alter heat shock protein (HSP) responses (43). In heat tolerant athletes believed to be fully recovered from a prior EHS/EHI episode (ranging from 6 wk to 10 years), after a bout of exercise-heat stress the lymphocyte HSP72 level was lower and *in vitro* lymphocyte HSP70 induction tended to be lower in post-EHI patients suggesting potential for reduced acquired cellular tolerance (44). There were no differences between control and post-EHS groups for core temperature or heart rate, emphasizing the ability to have similar physiological strain responses during a modest heat exposure and the need for more detailed molecular biomarkers (44). These findings suggest future research is needed to examine the relationship between residual tissue damage from EHS/EHI and long-term morbidity and mortality.

How Is Exertional Heat Illness Optimally Managed?

Evaluating and managing an athlete or laborer with exertional heat illness requires an effective "chain of survival" comparable to the American Heart Association's paradigm for out of hospital cardiac arrest. The "chain of survival" for EHI includes four linked steps: prehospital management; emergency medical service (EMS); advanced clinical management in a medical treatment facility; and finally, guiding the return to activity (see Fig. 2). The first three links in clinical care are detailed in this section on optimal management; the final step of facilitating a return to activity with attention to precipitating risk factors is discussed in sections V and VI.

Prehospital Management

Exertional heat illness clinical management depends on early recognition, immediate cooling, and transport to a medical facility for advanced care (see Fig. 3). The observation that the best outcomes are achieved with rapid reversal of body hyperthermia through early aggressive cooling is supported by robust literature (45–47); so, prehospital management is the most critical element of limiting the morbidity and mortality related to an EHI event. A recent consensus statement proposed four key steps in the paradigm of prehospital EHS victim care including rapid recognition, rapid assessment, and rapid cooling (45).

Rapid recognition

An evaluation for EHS is usually triggered by the collapse or near collapse of an athlete or laborer during or immediately following physical activity with heat stress. The differential diagnosis in a collapsed athlete is extensive, but most often due to sudden cardiac arrest, EHI, sports related concussion, exercise-associated hyponatremia, hypoglycemia, hypothermia, or exercise-associated postural hypotension (exercise-associated collapse). Many of these diagnoses have overlapping clinical presentations and a systematic approach incorporating vital signs and a brief cognitive assessment will expedite recognition and initial management, especially for providers in a field setting (Table 1). In all weather conditions, self-limiting postexercise collapse is usually due to sudden discontinuation of skeletal muscle pump activity causing venous pooling and postural hypotension rather than heat illness or dehydration, and the associated orthostatic instability usually resolves in less than 30 min with leg elevation and rest (48,49). A missed EHS diagnosis or delayed whole-body cooling may lead to single or multiple organ failure or death (15,50). The entire clinical picture, including the history of events leading up to the collapse, mental status changes, vital signs including rectal temperature, available point of care on site laboratory results, and regular reassessment, should be considered to optimally establish a diagnosis and manage the athlete with exertional collapse (23).

Rapid assessment

An unconscious athlete with spontaneous respirations or a conscious athlete with CNS changes should be assessed for EHS with on-site rectal temperature measurement. Rectal temperature measurement is considered the best estimate of core body temperature in the field (51–53) and provides an objective measure for assessing EHS in a collapsed athlete. Other common body temperature measurement sites such as the aural canal (tympanic membrane), oral sublingual, temporal artery or forehead, and axilla are inaccurate estimates of core temperature and should not be used for clinical decision making to assess heat related problems (51–55). If a rectal temperature cannot be measured, the on-site care team should evaluate the situation leading to the collapse, vital signs, and CNS function to determine if the victim should be managed as a heat stroke casualty and should not delay cooling until a rectal temperature can be measured.

Rapid cooling

The best outcomes for EHS and EHI require rapid on-site body cooling. On-site cooling prevents treatment delays and cooling interruptions associated with transportation to medical facilities and emergency department (ED) evaluation protocols for encephalopathy. Body cooling serves two purposes: 1) reducing organ and tissue temperatures and 2) supporting tissue perfusion by vasoconstricting blood vessels in the skin and superficial tissues, thereby moving intravascular volume from the peripheral to the central circulation.

The primary goal of prehospital cooling is to rapidly lower the body temperature into the normal range, which reduces the area under the cooling curve (degree-minutes) and protects the most heat sensitive body organs (56,57).

While a minimum cooling rate for achieving favorable clinical outcomes has not been established, cooling rates above $0.08°C \cdot min^{-1}$ appear to be acceptable, and cooling rates $>0.15°C \cdot min^{-1}$ are desirable to reduce both morbidity and

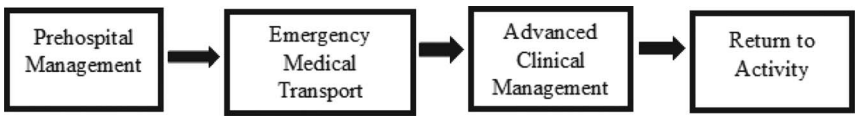

Figure 2: The exertional heat illness chain of survival promotes better outcomes and increases communication between care teams (45).

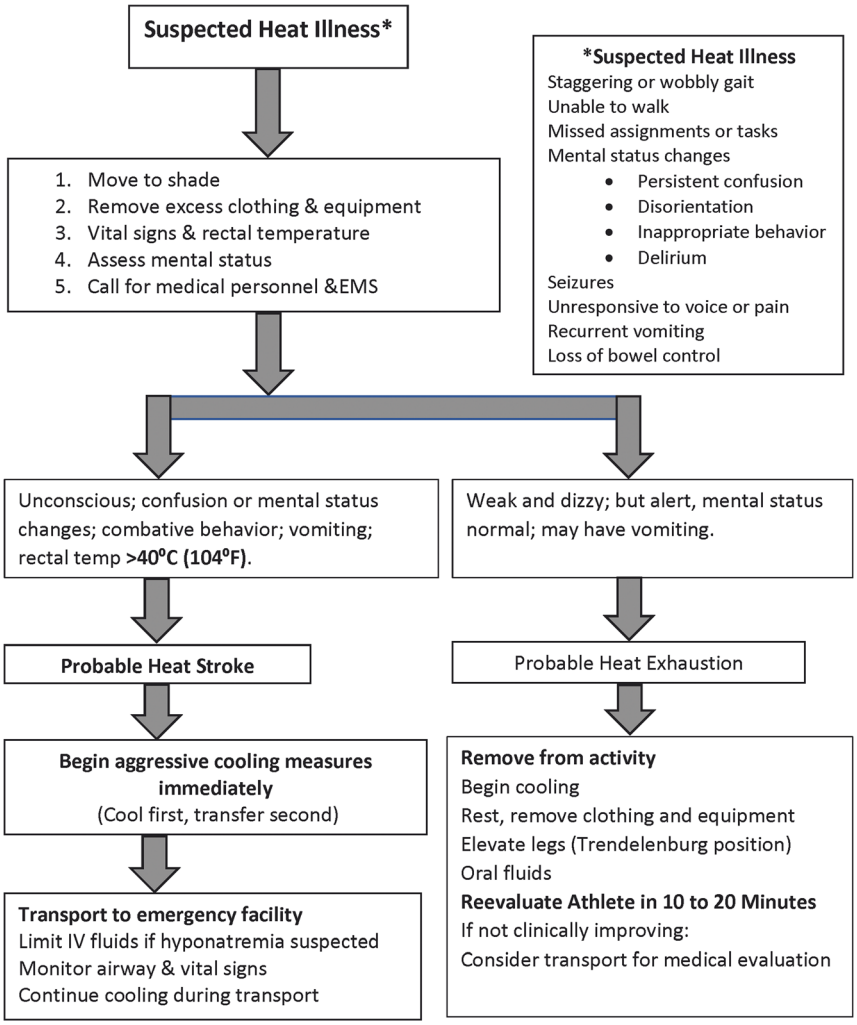

Figure 3: The evaluation and field care of an athlete with suspected EHI. Initiate immediate cooling measures based on the best and most practical cooling strategy for the site. If both rectal temperature measurement and cooling strategy are readily available, getting a rectal temperature is the best first step for clinical management decision making. Body cooling should take priority if a rectal temperature cannot be measured immediately, but a temperature measurement will eventually be needed to determine an end point for active cooling. In some settings with a heat illness care team on site, a recovered athlete may be released to family rather than transported to an emergency facility. Not all EHS casualties are unconscious, and it is important to look at the full clinical presentation. Based on field experience, the first three boxes in this cascade can take too much time and aggressive cooling should be started within minutes of collapse. Time sensitivity is obvious in cardiac arrest and acute stroke syndrome, but not necessarily engrained in those evaluating and managing heat stroke for the first time.

mortality (47,56,57). Insufficient or delayed cooling may result in clinical outcomes that include prolonged hospitalization or even death (46,47). Reducing body temperature therefore is essential to decrease the morbidity and mortality associated with EHS, and conductive heat exchange methods are the most effective in the field. All athletic venues that anticipate any chance of EHS should provide resources and personnel with expertise to facilitate whole body cooling.

The thermal conductivity of water is up to 32 times that of air and using cold water to facilitate conductive heat exchange at the skin level is the best means of rapidly reducing core body temperature (58,59). All athletic venues that anticipate any chance of EHS should provide resources and expertise to facilitate whole body cooling. Ice water tub immersion is effective for whole body cooling in hot, humid conditions (56,60) and is the preferred method of cooling in all conditions when available. At the Falmouth Road Race (11.4 km), 274 consecutive runners (ages 11 to 70 years old) have been promptly recognized and rapidly cooled on-site using ice water tub immersion with no fatalities and limited hospitalizations (61).

While cold water immersion is very effective, in some situations the use of 190-L (50-gallon) tubs with 10 to 15 kg (20 to 30 lbs) of ice may be impractical, and more portable methods may be an expedient alternative to initiate body cooling (62). Other water-based body cooling methods like rotating ice water soaked towels on the trunk, extremities, and head augmented with ice packs in the neck, axilla, and groin; repeatedly dousing the body with ice water; or continuously spraying the body with cool tap water are commonly used in the field to reduce body temperature in hyperthermic EHS casualties. Placing ice packs over major blood vessels in the groin, axilla, and neck can be combined with other cooling strategies as an adjunct but is not recommended as a lone treatment modality. However, in a "first aid" situation, ice packs over the major

vessels may be a lifesaving start to body cooling therapy. Evaporative cooling methods can be effective in air-conditioned spaces or low relative humidity environments but in high humidity conditions with restricted evaporative heat loss are not as effective as conductive cooling strategies.

The prehospital literature on cooling techniques for EHS is limited and field studies to evaluate the impact of cooling techniques are needed. When EHS is suspected or confirmed as the cause of collapse, initiating whole-body cooling is essential for favorable clinical outcomes. Table 2 lists methods that are often used in the field. The method chosen by the medical team is site-dependent and a blend of several elements, including clinical experience of the providers, available staffing, site assets and limitations, water and ice access, usual environment conditions, patient size and body type, and the incidence rate of EHS at the site (74). EHS victims with low body mass and high surface area to mass ratio (such as children or thin endurance athletes) may cool more rapidly than victims with large body mass and relatively low surface area to mass ratio (football linemen) who store more heat in the tissues (59,75). The environment also may play a role and cooling rates may be higher in cool dry conditions compared with hot humid conditions.

The recommendation to stop active cooling at ~38°C (101°F) to prevent hypothermia is not based on data showing adverse clinical outcomes. Most "overcooled" EHS casualties are in the 35°C to 36°C (95°F to 97°F) rectal temperature range, which is in the normal body temperature range and is not associated with any known adverse outcomes (23). Athletes with indwelling rectal thermistors can be monitored continuously without interrupting body cooling. When an indwelling thermistor is not available, repeatedly measuring rectal temperature interrupts body cooling and reduces the overall cooling rate. Simply continuing uninterrupted cooling until the victim clinically demonstrates a return to normal CNS function (*e.g.*, "wakes up" with eyes open, normal behavior, and conversation), heart rate, and blood pressure may be a more effective cooling strategy in this clinical scenario. Checking a rectal temperature when brain function and vital signs have returned to

Table 2.

This table identifies common onsite whole-body cooling strategies for EHS casualties, with cooling rates as reported in published studies.

Body Cooling Strategies (56, 63, 61, 64, 65, 66, 67, 68, 69, 70, 71, 72, 73)	Treatment Notes	Approximate Cooling Rate (°C·min^{-1})[a]
Ice water (~2°C) or cold water (~20°C) immersion with stirring	Immerse the full body up to the neck including upper and lower extremities (~90% body surface area) in a tank/tub, circulate the water to increase heat transfer, add ice during cooling to maintain water temperature, support head and airway above water level.	0.13 to 0.35
	Immerse as much of the torso and pelvic region (~65% body surface) following full body treatment notes, extremities not in water should be cooled using other strategies	0.04 to 0.25
Cold water dousing	Free flowing hose or bucket with cool tap water applied to the whole body—extremities, torso, hands, feet, neck, and head (with attention to the airway).	0.04 to 0.20
Tarp-assisted water ice/cold immersion	Providers hold the sides of the tarp with patient, water, and ice in the middle. Ensure as much of the torso, groin, and extremities are immersed as possible. Circulate the water as able.	0.14 to 0.17
Ice/cold water-soaked towels	Towels should be applied to the limbs (including feet and hands), trunk, and head with ice packs placed in the groin, axilla, and neck; include as much of the body as possible (~90% body surface area). Wring towels after soaking in bucket of ice water and change the towels rapidly.	0.11 to 0.16
Ice/cold water-soaked sheets	The whole body (~90% of body surface area) is wrapped in large sheets that are soaked with cold water. Sheets stay in place and are frequently rewetted. A fan directed at the body can be added.	0.05 to 0.06
Cold water immersion in portable water-impermeable bag	Immerse the full body up to the neck including upper and lower extremities (~90% body surface area) in the bag, support head and airway out of the bag.	0.04
Water spray/mister or high powered fan with water spray	Patient should be placed supine on a cot or table. As much of the body surface should be exposed (*i.e.,* remove clothes and shoes) to the fan and mist as possible.	0.03 to 0.17

Original research references provide detailed treatment implementation recommendations. See Table 8 for clinical recommendations.

[a]Cooling rate ranges are affected by body type, initial body temperature (mild exercise-induced hyperthermia or heat stroke), and environmental conditions.

normal will confirm that active cooling can be stopped. If a victim does not show signs of clinical improvement ("waking up") in 30 to 40 min, clinical reassessment is indicated.

Intravenous (IV) fluid replacement requirements vary based on the duration of physical activity and individual sweating rates. The need for IV fluid replacement is often clinically apparent following cooling after the return of peripheral blood volume to the central circulation. Peripheral IV sites complicate cooling procedures and initiating IV fluids can be postponed if the patient is clinically responding to cooling measures (61,73,76). Oral fluid ingestion is preferred to IV fluid replacement and should be started when the patient can tolerate oral intake. The risks of vigorous IV fluid administration in the field include dilutional hyponatremia and fluid overload, which can inadvertently increase morbidity and mortality (77).

Emergency Medical Transport

An EHS victim cooled on-site should be transported, as soon as possible, to a hospital ED that is equipped to evaluate and manage the complications of EHS and EHI. In road race settings that manage many exertional heat illness related problems, casualties with EHS who are promptly recognized, treated, wake up easily, and clinically stable are often discharged to home with family. In other settings not accustomed to managing EHI and EHS in the field, ED evaluation is strongly recommended following on-site cooling.

If on-site cooling was not started or completed, a suspected EHS casualty is best managed at the nearest medical facility with the capability for body cooling and medical management of EHI complications. EMS vehicles in areas with high EHI incidence rates should be equipped to begin or continue cooling therapy treatment *en route* (*e.g.*, chilled IV fluids, ice packs, cooling blankets [Bair Hugger™], fans) and use the vehicle air conditioning at high settings when EHS is suspected. Many EMS vehicles now carry refrigerated IV fluids chilled to 4°C (39°F) to augment induction of therapeutic hypothermia in cardiovascular emergencies.

Advanced Medical Treatment Facility Management

The third phase of clinical management involves advanced care in a hospital with inpatient critical care capability. When an individual with suspected exertional heat illness is transported to the hospital, the EMS dispatcher should ideally direct the patient to a facility with known experience treating heat casualties and notify the ED medical team in advance to allow preparation for immediate treatment upon arrival. The diagnosis of EHI and/or EHS can be challenging, as the patient may present with a rectal temperature <40°C due to active or passive cooling that occurred prior to or during transport. In addition, many medical care facilities are not equipped with various modalities for total body cooling, making rapid cooling difficult to achieve once the patient arrives. Sports event administrators and medical directors who expect potential EHS casualties should work with local hospital systems to facilitate appropriate treatment protocols and treatment areas.

In addition to being equipped with whole-body cooling options, the ED and hospital should be prepared for a patient that may be combative and require sedation with advanced airway management for staff safety during the evaluation. The medical team must be aware of common EHS sequelae, including acute kidney injury, rhabdomyolysis, liver failure, DIC, and SIRS. Organ and tissue damage may not be detected initially, and serial clinical testing is required to measure the extent of tissue damage (see Table 3). Intensive care and organ system support protocols will be necessary in some EHS victims. This document is not intended to outline either ED or inpatient intensive care clinical management as there are multiple resources with detailed care protocols.

Table 3.

Some of the following laboratory tests may be needed to assess clinical status and guide management.

• Complete blood count with platelet count
• Serum electrolytes (sodium, potassium, chloride, bicarb)
• Serum calcium and phosphorus
• Serum glucose
• Serum creatinine and blood urea nitrogen
• Serum uric acid
• Serum AST, ALT, LDH, alkaline phosphatase, and total bilirubin
• Creatine kinase
• Myoglobin blood and urine
• Fibrinogen, fibrinogen degradation products
• PT and activated partial thromboplastin
• Serum lactate
• Arterial blood gases

All these laboratory tests should be normal before beginning a return protocol (17,78,79).

ALT, alanine transaminase; AST, aspartate aminotransferase; LDH, lactate dehydrogenase; PT, prothrombin time.

How Can an Athlete with a Serious Exertional Heat Illness Safely Return to Activity?

Return to activity and play (RTA) decisions are challenging for medical providers managing athletes and laborers because our understanding of the pathophysiological processes involved in the evolution and recovery from EHS/EHI is incomplete (16,46,80–82). Returning an athlete safely and effectively to the full preheat illness level of performance is the primary task for the medical decision-making team. The final plan sometimes requires involving an athlete's coaches, supervisors, athletic trainers, and/or family members. Key clinical considerations include assessing organ system recovery and physical function abilities in warm or hot conditions (83).

Current research suggests that most individuals recover completely within a few weeks, especially if the heat illness incident was recognized promptly and cooled aggressively (16,82,84). However, some people with more serious cases experience long-term complications that may include multi-system organ (liver, kidney, muscle) dysfunction, neurologic damage, reduced exercise capacity, and/or heat intolerance (85–87).

A progressive increase in exercise intensity and duration in warm and hot conditions can be used to assess recovery. If an athlete can gradually regain pre heat injury exercise tolerance and expected performance for their activities in heat stress conditions, return to play is usually safe and advancing to full training and competition is acceptable. Medical eligibility for sports or occupational activity requires the individual to have

no medical ill effects (exercise related symptoms or abnormal clinical test outcomes) during the return process.

What Is Heat Tolerance?

Heat tolerance is the ability to sustain physical activity in warm to hot conditions and is partly dependent on an individual's physical conditioning and heat acclimatization status (88,89). Integrated cardiovascular, neurobiological, and systemic cellular responses determine an individual's heat tolerance (26,34). Heat intolerance may be linked to individual changes in the acclimatization process, prior illness or injury, and certain inherent or genetic characteristics (90). Some heat intolerant individuals have lost prior heat acclimatization or have inefficient heat loss responses (skin blood flow or sweating) related to a prior heat illness. Post-EHS heat intolerance may be temporary or permanent and can last from a few weeks to several years (90,91).

In the Severe Exertional Heat Illness Victim, What Clinical Criteria or Biomarkers Can Assist with Return to Exercise Activity Decisions?

Current RTA strategies are largely based on anecdotal observations and rely on provider experience for decision making to safely advance activity (22,35,81,92). Most guidelines require full symptom resolution at rest and normal laboratory findings for organs most often affected by EHI or EHS (*e.g.*, liver and kidney) before starting a cautious reintroduction of physical activity and a gradual heat acclimatization program. Assessing the basic hematologic parameters and blood chemistries for normal renal, hepatic, and coagulation normal function will give a baseline at the onset of activity, however, validated instruments to accurately predict continuing tissue or organ damage are not available and changes may only become evident with subsequent exercise related heat stress.

A follow-up evaluation about 1 wk after the incident is suggested for a physical examination and laboratory testing or diagnostic imaging (biomarkers) of the organs usually affected by EHS (22). While there is wide variability in the recovery of affected organs, recent research does outline biomarker recovery (17,93,94). A retrospective review of 2529 EHS episodes showed indicators of acute kidney injury (creatinine, blood urea nitrogen, and blood and protein in urine) peak on the day of the injury and normalize within 24 to 48 h; muscle damage and liver function-associated markers peak within 4 d after injury and can persist outside the reference ranges for 2 to 16 d and biochemical recovery from EHS is complete within 16 d for most casualties (95).

Can a Graduated Increase in Activity Be Used to Facilitate Acclimatization and a Return to Activity?

Evidence-based data to facilitate return to activity following EHI or EHS is limited and individual recovery following severe exertional heat illness is highly variable. Research is needed to evaluate the role of a graduated increase in structured physical exercise to assist in return to activity decisions (84). The U.S. Military Services have the most extensive guidelines for RTA (loss of productivity) following serious heat illness (21,29) and athletes suffering EHI or EHS can be managed following similar guidelines.

Following EHI or EHS, a medical evaluation of the patient is completed every week until all symptoms, signs, and abnormal laboratory values have resolved. Activities of daily living are the only "exercises" allowed for 2 wk. When all the symptoms and signs have resolved, low to moderate intensity physical training can be gradually increased to 60 $min \cdot d^{-1}$. When this level of exercise is well tolerated, a heat acclimatization plan (gradually increasing the duration and intensity of exercise and heat stress each day) can be started. If no symptoms of heat intolerance or abnormal blood work values are seen, the patient is released to unsupervised sport-specific physical activity.

The authors of this consensus statement recommend the following staged process to guide return to activity following a serious exertional heat illness (see Table 4). The return protocol relies on subjective measures and the athlete is advanced to the next stage if there is no evidence of exercise intolerance or fatigue. Each stage is individualized but may require up to 2 wk to complete in EHS victims. It is important that incremental elevations in both exercise intensity and heat stress be incorporated as these have additive effects on reducing gut blood flow and challenging gut permeability (96,97). If an athlete is unable to appropriately advance from stages 3 through 6, additional evaluation may be needed to determine the capacity for strenuous exercise in the heat, which may include heat tolerance testing (HTT) to assess current heat tolerance.

What Is the Role for HTT in Return to Activity and Medical Eligibility?

HTT is a potential clinical decision-making tool for athletes who are unable to advance activities in a reasonable time frame (see Table 4) or who have repeated episodes of exertional heat illness. The Israel Defense Force (IDF) Medical Corps uses an exercise HTT about 6 wk postexertional heat illness as part of the "return to duty" criteria based on changes in rectal temperature (T_{rec}) and heart rate (HR) during the test (88,98). A recent study of IDF military personnel calculated the sensitivity, specificity, and diagnostic accuracy of the HTT at 66.7%, 77.7%, and 77.2%, respectively (99). The authors concluded that the risk of EHI recurrence is measurable and that a negative HTT result is associated with a substantial reduction of EHI risk. The IDF has introduced mathematical modeling that calculates the probability of heat tolerance to improve test interpretation (99).

While case studies have demonstrated the efficacy of the IDF HTT in determining RTA for military warfighters and athletes afflicted by EHS (22,89,94,100), there are some concerns about the test's internal and external validity (101). In a recent review article, Mitchell et al. (101) question the test's external validity for tasks with high metabolic load, and the ability of the test to accurately determine true recovery and to predict people who are not likely to experience another EHS episode. Prior heat acclimatization significantly influences the outcome of an HTT, which confounds the test interpretation for an individual (102). The HTT has potential value as a tool to facilitate return to activity; however, limitations in test validity and access to the facilities required to perform the test limit the use to those with challenging cases that can be individually evaluated at special centers and to high-risk populations involved in research protocols. Further research will help establish the role of the HTT in the RTA decision making process.

Consensus strategies for return to activity following severe exertional heat illness are summarized in Table 5.

Table 4.

Staged return to activity or play after a diagnosis of exertional heat stroke.

Stage	Aim/Responsibility/Goal	Activity	Duration/Intensity	Example: HS Cross-Country Runner
1	Medical recovery Physician guided Organ system recovery	Activities of daily living for 1 to 2 wk	Gradual increase in home activities without fatigue	Home rest and return to school
2	Medical Recovery Physician Guided Sustain minimal aerobic fitness and develop confidence	Self-paced comfortable walk in low heat stress conditions (*e.g.,* an air- conditioned gymnasium)	20 to 60 min at Maximal Intensity of HR < 100 or <50% Age Adjusted maximal HR	Return to practice and walk through the warm-up and practice, if the environmental conditions are not stressful. If not, use an air conditioned area of the school
3	Early Exercise Adaptation Athletic trainer guided with physician Gradually improve aerobic exercise capability	Walk at 3.5 mph in low heat stress conditions	60 min at HR < 140 bpm or <70% of age adjusted Maximal HR	Warm up & cool down with team, 1 of 4 reps at half speed
4	Mid Exercise Adaptation Athletic Trainer Guided with Physician Gradually improve aerobic exercise capability & fitness	Walk & run in low heat stress conditions	60 min of progressively increasing run to walk ratio until constant run for 60 min	1 of 3 reps, half speed 1 of 2 reps, ¾ speed
5	Heat Acclimatization Athletic Trainer Guided Gradually improve heat Acclimatization status	Run in ambient warm or hot conditions	60 min of progressively increasing run until constant run for 60 min;	All reps, ¾ speed
6	Sports-specific acclimatization/training Athletic trainer and/or coach guided Improve sport-specific heat acclimatization & fitness	Participate in practice in ambient conditions	Initially participate in sports-specific drills with sports-specific equipment then progress to training and scrimmage.	All reps, full speed
7	Return to Sport Athletic trainer monitors during warm up and game	Normal game or competition participation in ambient conditions		Meet 1—run to finish the race Meet 2 — race to place in the race

Table 5.

ACSM return to activity considerations following a diagnosis of EHS or EHI.

- A detailed history and physical examination including unique intrinsic and extrinsic risk factors, the timing of treatment, and the rate of cooling must be considered in the RTA decision.
- The athlete should refrain from exercise for at least 7 d after release from initial medical care, at which time the clinician will address the clinical course of the heat stroke incident and carefully assess the status of end-organ function (neurocognitive, renal, hepatic, muscle, hematologic as clinically indicated);
- The clinician should carefully address any intrinsic and extrinsic risk factors associated with the EHS event.
- When medically eligible for RTA/RTP based on the return of normal end organ function, an individual can begin exercise in a cool environment and gradually increase the duration, intensity, and heat exposure over 2 to 4 wk to initiate environmental acclimatization, improve fitness, and demonstrate heat tolerance.
- If return to vigorous activity and evidence of the patient's ability to adapt to exercise-heat stress over several days is not accomplished within 4 to 6 wk, consider referral to a physician with experience in heat related disorders for further evaluation that may include HTT in a controlled setting.
- An athlete may be allowed to resume full competition between 2 and 4 wk after demonstrating sports-specific exercise acclimatization and heat tolerance with no abnormal symptoms or test results during the re-acclimatization period.

Table 6.
Predisposing factors to exertional heat illness.

Environmental Factors
• Warm-hot weather conditions
• Unusually hot for region and season
• Heat wave defined as >3 d of >32°C (90°F)
• Wearing heavy clothes, equipment, or uniforms
Individual factors
• Age (infants, older adults)
• Overweight, high body mass index
• Poor physical fitness
• Inappropriate work to rest ratios
• Inadequate heat acclimatization for current conditions
• Heat stress in the previous 1 to 3 d
• Hypohydration
Medications and drugs
• Diuretics
• Anticholinergics
• B-adrenergic blockers
• Antihistamines
• Antidepressants
• Stimulants (amphetamines, cocaine, ecstasy, ephedra)
Health conditions
• Viral or bacterial infections
• Fever
• Diarrhea or vomiting
• Skin disorders (rash, large area of burned skin)
• Diabetes mellitus
• Cystic fibrosis/trait
• Cardiovascular disease
Behavioral
• Self-imposed motivation to excel
• Leadership or organizational structure
• Peer or coach pressure to excel

What Increases Individual Risk and How Can Risk for Serious Exertional Heat Illness Be Reduced?

Predisposing Factors

Exertional heat illness can occur in both healthy and "high-risk" individuals when performing vigorous activity in warm or hot conditions. Risk factors for exertional heat illness listed in Table 6 can be unique to a particular exertional event or a given individual, and often more than one risk factor is present in an individual victim. While EHS is not completely understood and is challenging to predict, numerous risk factors associated with EHS have been identified by epidemiologic data that include environmental, physiological, drug use, and compromised health (25,30). For athletes, the most common risk factors are low physical fitness, lack of heat acclimatization, obesity, and heat waves or unexpectedly hot weather (15,30,103). Risk factors outlined in Table 6 can help identify individuals who should be more closely monitored by the sports medicine team and staff stakeholders during participation or have a "buddy" assigned to report any signs or symptoms (23,104). The most physically fit, heat acclimated, and motivated individuals tend to sustain high rates of metabolic heat production during intense physical activity and are highly motivated to continue activity even when experiencing excessive fatigue or symptoms of exertional heat illness (see Fig. 4).

In addition, the presence of a predisposing factor (*e.g.*, recent viral illness or fever) on a particular day increases the risk of heat stroke in subsequent days and sets up the "multiple-hit hypothesis" (26,30), which may account for athletes who have completed the same exercise-heat stress task many times in the past without any pathological events but on a subsequent day suffer an EHS event (16,26,30). The multiple hit hypothesis suggests that events leading up to the day may prime the system for failure by allowing an unopposed immune response.

Athletes with an EHS episode are at higher risk for a subsequent event (14). In an 11.4-km warm-hot weather road race, the relative risk of a second EHS was 3.3 in the 2 years following the initial episode and the relative risk dropped to 1.3 in the 3 to 5 years following the episode based on a total of 333 EHS patients among 174,853 finishers (106). Studies of French and U.S. military personnel show an association between hospitalization for EHS following a previous episode (107,108). Among active-duty soldiers, those with a prior serious EHI event had four-fold greater odds of experiencing a mild EHI event and those with a prior mild EHI had a 1.8-fold greater risk of having a serious EHI event at a future date (109). Malignant hyperthermia trait has recently been suggested as a predisposing factor for exertional hyperthermia, but this is controversial, and more research is needed (110).

An important risk factor for EHS is extrinsic pressure to perform beyond the level of acclimatization or fitness. EHI and EHS sometimes occurs in sport, work, or military activities when a healthy individual is pushed by a coach or supervisor to the point collapse during exercise in cool to hot environmental conditions. Leadership plays an important role of in the evolution and prevention of EHI and EHS (104).

Risk Reduction

Heat acclimatization

Heat acclimatization is an adaptive physiological and perceived exertion change that occurs with repeated exposure to exercise heat stress. These adaptations improve physical function in the heat to reduce physiological strain (37) and induce molecular adaptations that protect organs and tissues (38) by favorably influencing fluid-electrolyte balance (*e.g.*, decreased sweat and urine sodium concentration), cardiovascular function (*e.g.*, regulated heart rate, circulating blood volume, and exercising blood pressure), and body temperature (*e.g.*, increased sweat rate and skin blood flow during exercise) (37,111). A period of 1 to 2 wk of heat acclimatization is recommended to induce most physiological adaptations, which optimize performance and reduce the risk of exertional heat illness (37,112,113). When training for a specific event or task, this process is accomplished by gradually increasing the

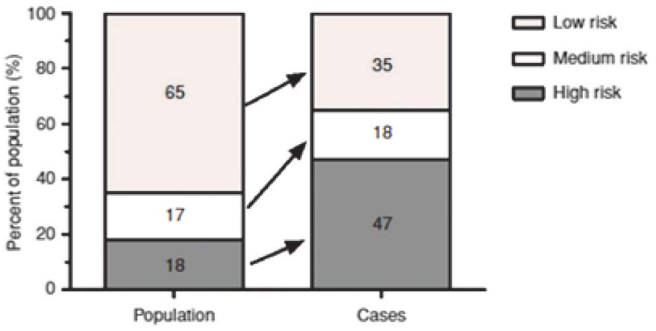

Figure 4: This graph shows the distribution of relative risk for EHS among Marine recruits; the "high risk" recruits (18% of population) accounted for 47% of EHS cases, but importantly the "low risk" recruits (35% of population) accounted for 35% of EHS cases (105). The low-risk recruits tend to be more motivated to excel and at higher risk for EHS than expected.

intensity and duration of exercise during daily heat exposure. Adaptations can be customized to the athlete and sport (111,114).

Hydration

Dehydration contributes to heat exhaustion (115) but a direct link to EHI and EHS has not been established; although it is reasonable to believe that dehydration indirectly increases physiological strain (26). Regardless, athletes often dehydrate during training and competition, and rehydration strategies are important for athletes to safely perform physical activity in warm to hot conditions (35). Hydration supports vascular volume and sweating, both essential to temperature regulation. Athletes experience wide-ranging sweat losses during vigorous exercise, especially in hot environments (116) and may incur substantial body water deficits without fluid replacement during exercise (117). Replacing mild-to-moderate fluid losses during recovery is best done by combining eating with fluid intake. For athletes with signs and symptoms of dehydration (*i.e.*, dizziness, rapid heat rate, fatigue, headache) the use of commercial oral rehydration fluids is more effective than sports drinks with lower sodium content (117,118). A variety of methods can be used to self-assess dehydration, but for athletes and active laborers the most common approaches involve monitoring body weight changes, thirst, and urine volume or concentration (22,35,117).

Administrative Strategies

Given the chance to compete, athletes often override innate behavioral strategies that reduce the risk of EHI. Decreasing the work-to-rest ratio or reducing exercise intensity as the environmental heat stress increases can minimize the risk of EHI and optimize work completed. In higher-risk conditions, site leaders should change training sessions by adding longer and more frequent rest breaks to allow heat dissipation and shorter bouts of high intensity exercise to decrease heat production. Longer rest breaks and unlimited fluid access permit better fluid replacement when athletes have high fluid losses. Other factors to consider when modifying training or competition include heat acclimatization status of participants, fitness and age of participants, intensity and duration of exercise, time of day, clothing or uniform requirements, and playing surface (*i.e.*, grass vs synthetic fields). Equipment requirements and exercise intensity are factors in football related EHS (119).

Acclimatization affects heat tolerance and different geographical areas or regions may require different environmental algorithms for modifying and canceling physical activity (120). The recommendations for curtailing activity may change through a season as athletes become fit and acclimatized to the local conditions.

Modifying, postponing, or canceling an event can be based on data specific to event outcomes (120,121). A region-specific exercise modification table (Table 7) provides a primary risk reduction strategy that works for many physical activities (103,120,122,123). Heat safety tables used by institutions should include modifications in activity duration and intensity, increased rest breaks, and removal of extra clothing or equipment.

Administrators, supervisors, and participants should understand predisposing factors of EHS (Table 6) and recognize early signs. More importantly, mandated heat safety policies and an emergency action plan should be in place at each activity site to increase exertional heat illness preparedness and reduce EHS deaths (122,124,125). Administrators should strive to develop an on-site health care delivery team with trained and licensed providers to improve triage and reduce morbidity and mortality associated with incorrect diagnosis and treatment (15,45,126,127).

Summary

EHS is a true medical emergency with potential for irreversible organ injury and death. This consensus statement emphasizes that optimal exertional heat illness management is promoted by a synchronized chain of survival with rapid recognition and management, and accurate communication between care teams (Table 8). Health care providers should understand the definitions, etiologies, and nuances of EHE, EHI, and EHS. Early identification and rapid total body cooling of an athlete with suspected EHS is essential for survival, and like any ischemic situation, time is tissue. Recovery from EHS is variable and outcomes are likely related to the duration of severe hyperthermia. Those treated before cell injury and the exaggerated acute phase response usually return to full activity within a few weeks, while those with a more complicated and prolonged course require a graduated and supervised return to prior activity levels. Finally, exertional heat illnesses can be prevented by recognizing and mitigating well described risk factors ideally addressed through leadership, policy, and on-site health care.

Research Needs and Knowledge Gaps

- Examine impact of cooling method and duration on clinical outcomes of EHI/EHS victims.
- Examine the impact of body morphology on cooling rates in EHS.
- Identify novel clinical-molecular biomarkers that predict heat intolerance, morbidity or mortality, and return-to play for EHI/EHS victims.
- Evaluate pharmacological interventions that reduce risk of morbidity and mortality in EHI/EHS patients.
- Identify HTT strategies that will accurately predict heat safety.

Table 7.

WBGT levels for modification or cancellation of workouts or sports competition for healthy adult athletes.

WBGT (°C)			Continuous Activity and Competition for Individuals Who Are Highly Fit and Fully Acclimatized to High Heat Conditions[a,b]	Training and Noncontinuous Activity	
Region category[c]					
Category 3	Category 2	Category 1		Nonacclimatized, unfit, high-risk individuals [b]	Acclimatized, fit, low-risk individuals[b,d]
≤ 10.0	< 8.7	< 6.7	Generally safe; EHS can occur associated with individual factors	Normal activity	Normal activity
10.1 to 18.3	8.8 to 17.0	6.8 to 15.0	Generally safe. EHS can occur in individuals who are not acclimatized or have risk factors	Normal activity	Normal activity
18.4 to 22.2	17.1 to 20.9	15.1 to 18.9	Generally safe. Risk of EHS and other heat illness begins to rise; high-risk individuals should be monitored	Increased risk. Increase the rest/work ratio. Monitor fluid intake.	Normal activity
22.3 to 25.6	21.0 to 24.3	19.0 to 22.3	Risk for all active individuals is increased	Moderate risk. Increase the rest/work ratio and decrease total duration of activity. American rules football restrict equipment to helmet, shoulder pads, and shorts.	Normal activity. Monitor fluid intake. Limit protective equipment.
25.7 to 27.8	24.4 to 26.5	22.4 to 24.5	Risk for unfit, nonacclimatized individuals is high	Moderate high risk. Increase the rest:work ratio; decrease intensity and total duration of activity. No protective equipment during practice and no conditioning activities.	Normal activity. Increase rest breaks. Monitor fluid intake. Consider removing protective equipment. Provide at least three rest breaks per hour with a minimum duration of 4 min each.
27.9 to 30.0	26.6 to 28.7	24.6 to 26.7	Very high risk[e] Cancel exercise or competition.	Very high risk.[e] Increase the rest:work ratio to 1:1, decrease intensity and total duration of activity. Limit intense exercise. Watch at-risk individuals carefully	Increase the rest:work ratio and decrease total duration of activity (< 2 h). Plan intense or prolonged exercise with discretion, watch at-risk individuals carefully. No protective equipment during practice and no conditioning activities. Provide at least four rest breaks with a minimum duration of 4 min each.
30.1 to 32.2	28.8 to 30.9	26.8 to 28.9		Very high risk[e] Cancel or stop practice and competition.	Limit exercise time to ≤ 1 h, no conditioning activities, 20 min of rest breaks distributed throughout exercise time, no protective equipment, watch for early signs and symptoms
> 32.3	> 31.0	> 29.0		Extremely high risk.[e] Cancel exercise.	Extremely high risk.[e] Cancel exercise

These guidelines do not account for the effects of a uniform, clothing, and protective equipment (*i.e.*, American football) which increase sweat losses and elevate body temperature; these factors should be considered when determining competition or practice limitations based on the WBGT. It is best practice to use activity modification thresholds that account for local or regional climate conditions. Use on-site WBGT devices to measure or access estimated WBGT readings for your area. WBGT forecast information is available at https://digital.mdl.nws.noaa.gov/(use drop down menu to access WBGT) to help inform next day decisions or use WBGT calculations at https://www.osha.gov/otm/section-3-health-hazards/chapter-4#wbgt that also includes a link to estimate current local WBGT conditions.

[a] High-level athletes who have been acclimatized ≥12 wk, not applicable to events with unacclimatized participants.

[b] While wearing shorts, t-shirt, socks, and sneakers.

[c] Region category 1 primarily includes the most northern regions of the contingent United States, category 2 includes the middle of the United States while also encompassing some northern and southern areas, category 3 is primarily the southeast and southwest. Use of the map within the original reference is recommended for clinicians to determine which category to use when developing the heat modification policies and procedures. Outside the United States, humid subtropical and hot desert climates best fit in category 3 and marine cooler climates in category 1 but using historical data to calculate appropriate thresholds for each region as described previously is the best risk reduction strategy. (103,120–124).

[d] At least 3 wk in the heat.

[e] Risk of EHS and EHE.

WBGT, wet bulb globe temperature.

Table 8.

Exertional heat stroke consensus summary statements and recommendations with evidence grades assessed using the strength of recommendation taxonomy (128).

Clinical Statement/Recommendation	Strength of Recommendation
Recognition	
Sports, military, and labor site administrators should be prepared for recognition, assessment, and initial management of exertional heat illness (7–10,13,129,130).	A
Subtle to profound CNS changes in conjunction with rectal temperature >40 °C are the primary identifying signs of EHS (13–15,74).	A
Rectal temperature helps differentiate EHS from other causes of athlete collapse during and following physical activity (53–55,131).	A
Management	
A planned prehospital chain of emergency treatment improves communication between care teams and exertional heat illness outcomes (45,126,127).	C
Delayed recognition and insufficient or delayed cooling can result in medical complications of EHS including death (47).	A
Early cooling is essential for optimal EHS treatment outcomes (45,47,61).	B
Mental status and vital signs (including rectal temperature) guide the cooling treatment of exertional heat illness causalities (45,56,61,132).	B
Cold water immersion has effective cooling rates with good clinical outcomes in EHS casualties and the preferred cooling strategy (47,56,63,64,70,71,74,75).	A
When cold-water immersion of the body is not possible, a patient with EHS should be cooled using the fastest cooling technique available (46, 49, 67, 69, 70, 71 74, 128).	B
Return to activity	
EHS and EHI casualties should be deemed clinically recovered by the medical team prior to resuming physical activity.	C
Recovered heat illness casualties should be progressively reintroduced to physical activity and heat exposure over several weeks to months.	C
Return to activity should be individualized for each heat stroke patient.	C
Prevention	
EHS risk rises with increasing environmental heat stress and increasing physical activity intensity (19,103,120,133–138).	A
Physical activity should be modified or canceled in environments known to be associated with high risk of EHS (120,133,134,136).	B
Heat acclimatization reduces the risk of heat illnesses and improves physical performance in the heat (124,125,139–141).	A
Administrative strategies, focused education, and responsible leadership can reduce exertional heat illness rates and deaths (15,104,124).	B

A, consistent, good-quality patient-oriented evidence; B, inconsistent or limited-quality patient-oriented evidence; C, consensus, disease-oriented evidence, usual practice, expert opinion, or case series (for information about the SORT evidence rating system, go to https:// www.aafp.org/afpsort.)

- Evaluate the effectiveness of different functional protocols for safe return to play after EHI/EHS.
- Determine if the proposed return to activity protocol be safely accelerated.

This article is being published as an official pronouncement of the American College of Sports Medicine. This pronouncement was reviewed for the American College of Sports Medicine by members-at-large and the Pronouncements Committee.

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See Supplemental Digital Content 1, [http://links.lww.com/CSMR/A140] to download a slide deck that summarizes this revised consensus statement on Exertional Heat Illness: Recognition, Management, and Return to Activity.

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