Curr Rev Musculoskelet Med (2014) 7:355–365
DOI 10.1007/s12178-014-9240-0

Heat-related illness in sports and exercise

Andrew W. Nichols

Published online: 21 September 2014

Abstract Exertional heat-related illness (EHRI) is comprised of several states that afflict physically active persons when exercising during conditions of high environmental heat stress. Certain forms of EHRI may become life threatening if not treated. Exertional heat stroke (EHS), characterized by a core body temperature of >40 ° C and mental status changes, is the most severe form of EHRI. EHS must be treated immediately with rapid body cooling to reduce morbidity and mortality. Many EHRI cases are preventable by following heat acclimatization guidelines, modifying sports and exercise sessions during conditions of high environmental heat stress, maintaining adequate hydration, avoiding exertion in the heat when ill, and by educating sports medicine personnel, coaches, parents, and athletes on the early recognition and prevention of EHRI. Heat exhaustion, exercise-associated collapse, exercise-associated muscle cramps, exercise-associated hyponatremia, and exertional rhabdomyolysis are also described.

Keywords Heat illness · Heat injury · Athletic heat injury · Exertional heat illness · Exertional heat stroke · Heat stroke · Heat exhaustion · Muscle cramps · Exertional hyponatremia · Exertional rhabdomyolysis

Introduction

Exertional heat-related illness (EHRI) refers to a spectrum of heat illnesses that occur during sports and exercise participation in conditions of environmental heat stress often characterized by high ambient temperatures and humidity levels. The most serious form of EHRI, exertional heat stroke (EHS), is among the leading causes of on-the-field sudden death in athletes. The number of sports-related EHS deaths in the United States has doubled since 1975, and more deaths were reported between 2005 and 2009 than during any 5-year period of the preceding 30 years [1•, 2, 3•, 4]. American football is the sport associated with the greatest number of EHRI fatalities in the United States [2]. The National Center for Catastrophic Sports Injury Research database identified heat illness as the third most common cause of sports-related fatalities in US high school and college football players between the years of 1990 2010, accounting for 15.6 % (n=38) of reported deaths. College football players were 3.8-times more likely to perish from EHRI as compared with high school football players [5•]. The National High School Sports-Related Injury Surveillance Study found that high school football athletes experienced an 11.4-times higher rate of EHRI time-loss events compared with athletes participating in 8 other high school sports [3•, 6•, 7•]. The month of August, which is typically associated with pre-season football camps, accounted for 66.3 % of EHRI time-loss events [3•, 7•]. An average of 5946 persons were treated annually in US hospital emergency departments for EHRI (2.0 ED visits/100,000 population) with a hospitalization rate of 7.1 %. ED visits were most common among males (72.5 %) and persons in the 15- to 19-year age group (35.6 %) [8, 9].

Thermoregulation

Body heat may be transferred by 4 mechanisms: (1) conduction, which refers to heat that flows from a cooler to a warmer object by direct contact; (2) convection, which involves heat transfer via air circulation at the body surface; (3) radiation, which arises through the transmission of electromagnetic

A. W. Nichols (✉)
Family Medicine and Community Health, John A. Burns School of Medicine, University of Hawaii at Mānoa, University Health Services Mānoa, 1710 East-West Road, Honolulu, HI 96822, USA
e-mail: nicholsa@hawaii.edu

waves; and (4) evaporation, which occurs via sweat at the skin surface. When the body is at rest or exercising moderately in cooler temperatures, the majority of heat loss occurs through conduction, convection, and radiation. As environmental heat stress and exercise intensity increase, evaporation of sweat at the skin surface becomes the predominant mechanism for body heat dissipation. The hypothalamus triggers sweating and increased cutaneous blood flow upon detection of an elevation in core body temperature.

The wet-bulb globe temperature (WBGT), which determines the saturated immobile air temperature that would impose an identical thermal load as the actual environment, is the best measurement of external thermal load. Ambient dry air temperature (standard thermometer), the true capacity of air to evaporate water according to its relative humidity and air velocity (wet-bulb thermometer), and solar radiation (globe thermometer) readings each contribute to the calculation of the WBGT [10••]. Hot temperatures reduce conductive heat exchange between the air and skin because of the small temperature gradient. High humidity reduces the efficacy of sweating in releasing body heat because of high air water vapor pressure. Lack of air movement and wind minimizes convective heat dissipation. Infrared radiation adds to heat absorption during exercise in the sun. Thus, conditions of high temperature and humidity, still (windless) air, and extreme solar radiation contribute to elevated environmental heat stress.

Heat acclimatization refers to the gradual advancement of physiological adaptations to exercise in conditions of high environmental heat stress. The process occurs optimally over 10–14 days of heat stress exposure exercise sessions that last 60–90 minutes per day. Physiological changes include increases in plasma volume, sweat rates, cutaneous vasodilation, and aldosterone production; and decreases in urinary sodium excretion, sweating threshold, sweat electrolyte content, and heart rate at a given workload.

Risk factors for EHRI

Risk factors for EHRI may be internal or external. General internal risk factors include a history of previous EHRI, poor physical fitness level, lack of heat acclimatization, excessive or inappropriate clothing, protective equipment, young or old age, inadequate sleep, excessive motivation, and poor education about EHRI. Medical conditions that may predispose to EHRI are obesity, recent febrile illness, gastroenteritis, sickle cell trait, sweat gland dysfunction, sunburn, poorly controlled diabetes mellitus and hypertension, cardiovascular disease, diabetes insipidus, and cystic fibrosis [11]. Recent concussion with residual CNS dysfunction and prior history of malignant hyperthermia are also linked to increased EHRI risk [12, 13]. Experimentally induced muscle-damaging exercise appears to be a risk factor for both subsequent acute kidney injury and EHRI during exertional heat stress conditions [14, 15•]. Total heat loss measurements using direct calorimetry confirmed that body heat dissipation is significantly impaired during exertion in the heat in 40–70 year old men compared with younger men [16].

Among the medications and supplements associated with EHRI are stimulants (eg, amphetamines, methylphenidate, ephedra, alpha agonists, and thyroid agonists), anticholinergics, antihistamines, benzodiazepines, various cardiovascular drugs (eg, beta blockers, calcium channel blockers, and diuretics), alcohol, laxatives, tricyclic antidepressants, antipsychotics, and illegal drugs (eg, cocaine, heroin, lysergic acid diethylamine, and phencyclidine) [11]. Attention deficit hyperactivity disorder stimulant medicines (eg, methylphenidate) have been shown to increase body temperatures during activity in warm environments and perhaps enhance exercise performance. These agents are hypothesized to increase EHRI risk because of the inhibition of CNS signals intended to decrease or discontinue exercise as the body temperature rises [17•]. Aspirin and clopidogrel ingestion were also both shown to increase core body temperatures and possibly the risk of EHRI in middle-aged subjects engaged in exercise under heat stress [18]. Ephedra has been linked to EHRI risk through the activation of dopaminergic receptors with impairment of heat dissipation via vasoconstriction [19]. Caffeine may increase the risk of EHRI through diuresis, sympathetic nervous system stimulation, and metabolic rate enhancement; although studies are inconclusive [19]. Creatine and branched chain amino acids (BCAA) have not yet been associated with a heightened EHRI risk [20, 21].

Environmental conditions of high heat stress (especially WBGT >28 ° C), is a critical external risk factor for EHRI. Additional hazards include exercise intensity (>75 % VO2 max) and duration (>1 hour), poor awareness of the risks of EHRI by the coach, parent, and athlete, lack of an EHRI emergency action plan, delayed recognition and management of early EHRI, and inadequate access to fluids and shade [11].

Types of EHRI

The various EHRI types with their associated clinical signs, symptoms, and criteria for diagnosis are summarized in Table 1 [22]. Among the milder forms of EHRI is the self-limited heat rash (also known as miliaria rubra and "prickly heat") that presents with a pruritic papulovesicular eruption over the clothed areas, and heat edema that produces mild edema in the dependent areas of the extremities. Both conditions are associated with a normal core body temperature.

Table 1 EHRI types and criteria for diagnosis (adapted with permission from Howe [22])

Condition	Core temperature	Associated symptoms	Associated signs
Heat edema	Normal	None	Mild edema in dependent areas
Heat rash	Normal	Pruritic rash	Papulovesicular skin eruption on clothed areas
EAC	Normal	Dizziness, generalized weakness	Loss of postural control, with rapid MS recovery once supine
EAMC	Normal to <40 °C	Painful muscle contractions	Affected muscles may feel firm to palpation
HE	Normal to 40 °C	Dizziness, malaise, fatigue, nausea, vomiting, headache	Flushed, profuse sweating, cold clammy skin, normal mental status
EHS	>40 °C	Possible history of HE symptoms before onset of MS changes	Hot skin ± sweating, CNS disturbance
ER	Normal to >40 °C (if with EHS)	Severe muscle pain, ± EHS, often associated with "novel over-exertion"	↑↑CK levels, myoglobinuria, renal injury, ↑K, cardiac arrhythmias
EAH	Normal to 40 °C	Peripheral edema, possible history of HE symptoms before onset of MS changes	Na <135 mg/dL, CNS disturbance

CNS central nervous system, *EAC* exercise-associated collapse, *EAH* exercise-associated hyponatremia, *EAMC* exercise-associated muscle cramps, *EHS* exertional heatstroke, *ER* exertional rhabdomyolysis, *HE* heat exhaustion, *MS* mental status.

Exercise-associated collapse

Exercise-associated collapse (EAC) may be defined as an event in which the conscious athlete is unable to stand or walk without assistance because of lightheadedness, faintness, dizziness or syncope. EAC characteristically presents at the completion of a bout of strenuous physical activity, and accounts for 59 %–85 % of medical visits to marathon, ultramarathon, and distance triathlon medical tents [23, 24]. The incidence of EAC increases with higher ambient temperatures, humidity levels, and levels of dehydration [23, 25]. Exercise-associated postural hypotension (EAPH) is diagnosed when an athlete with postexercise symptoms demonstrates a decline in systolic blood pressure of at least 20 mm Hg upon rising from supine position to an upright posture.

The etiology of EAC is multifactorial. Endurance training in the athlete induces an increase in cardiac output and a reduced resting heart rate. Concurrently, the actively exercising lower extremity muscles require greatly increased blood flow that necessitates reduced peripheral vascular resistance. The working skeletal muscles are forced to act as a "second heart", which promotes venous blood return to the heart. Upon the discontinuation of endurance exercise, the second heart no longer assists in the return of blood to the heart, leading to venous pooling in the lower extremities and resultant orthostatic intolerance. Heat stress contributes further to EAC and orthostatic intolerance by directly reducing cardiac baroreflex control, increasing cardiovascular strain with impairment of aerobic exercise performance, and raising cerebral vascular resistance, which reduces the threshold for neurogenic collapse [26].

Before confirming the diagnosis EAC, EHS and EAH must be ruled out by determining that the core body temperature is <40 ° C, an absence of mental status changes, and normal serum sodium levels. The endurance sport athlete with EAC should be treated initially with supine positioning in which the legs are elevated 12–24 inches above the heart, and oral rehydration as tolerated. The patient's vital signs, rectal temperature, and mental status must be continually reassessed. Intravenous fluids may be administered if the patient is not able to tolerate PO fluids. Criteria for discharge include being able to ambulate on one's own power and retaining normal mental status. Hospital emergency department transfer should be considered if the patient fails to meet these criteria within 1 hour [27•]. Runners with prior episodes of EAC who are prone to orthostatic intolerance, may consider wearing lower extremity compression stockings to help prevent recurrence [28].

Exercise-associated muscle cramps

Exercise-associated muscle cramps (EAMC) typically affect the larger skeletal muscles during or after exercise in the heat. Although EAMC is usually associated with excessive sweat losses, the individual is not necessarily overheated and if so, the core temperature does not exceed 40 ° C. Muscle twitches occur initially with progression to severe and widespread muscle spasms. Muscle fatigue, dehydration, high sweat rates, and high sodium sweat concentrations predispose to EAMC [29–31]. Muscle cramps may result when excessive sweat sodium losses are replaced with hypotonic fluids, causing extracellular fluid volume contraction and mechanical deformation of nerve terminals during muscle shortening that initiates muscle hyperexcitability [32, 33]. Treatment options include rest, prolonged passive muscle stretching, salt supplementation through oral or IV fluids and salt-containing foods, massage, and ice massage. Refractory muscle cramps may require intravenous benzodiazepines. Prevention focuses on

maintaining fluid and salt balance. Athletes at risk may be "high volume" and/or "high sodium" sweaters. Case studies have shown the efficacy of increasing dietary salt in reducing muscle cramps in tennis players [33, 34]. Supplementation with calcium salts, sodium bicarbonate, quinine, and dextrose have not been shown to be effective in preventing EAMC.

Heat exhaustion

Heat exhaustion (HE) is the most common form of EHRI and is characterized by hypotension and cardiovascular insufficiency in persons who become dehydrated while exercising in conditions of environmental heat stress. Symptoms include nausea and vomiting, tachycardia, dizziness, muscle cramps, energy depletion, central fatigue, and syncope. Core body temperature may be elevated but to no higher than 40 ° C, and the mental status remains intact. HE is often difficult to differentiate clinically from the early stages of exertional heat stroke. The rectal temperature measurement provides essential information to guide the appropriate management of the athlete who collapses while exercising in the heat.

The initial treatment of HE involves discontinuation of the activity, removal of clothing and sports equipment that restrict heat dissipation, relocation to a cool shady area, supine positioning with the lower extremities elevated, oral rehydration, monitoring of rectal temperature, vital signs, and mental status, and consideration of ice towel application and/or ice water immersion cooling therapy. Intravenous rehydration may be administered. If core temperature rises to greater than 40 ° C and/or if mental status changes develop, progression to EHS must be assumed prompting the implementation of aggressive cooling measures and activation of the EMS. Although there are no clear and validated guidelines for return to play following an episode of EH, common sense suggests that individuals should abstain from exercise for at least 24 hours following the resolution of all symptoms and laboratory abnormalities.

Exertional heatstroke

Exertional heatstroke (EHS) is a life-threatening medical condition exemplified by a core body temperature that exceeds 40 ° C, CNS dysfunction, skeletal muscle injury, and multiple organ damage. The markedly elevated core body temperature may become irreversible without aggressive body cooling measures because of thermoregulatory mechanism failure. Early signs and symptoms of EHS are often similar to those of HE and include clumsiness, stumbling, headache, nausea, and dizziness. These may progress to apathy, confusion, and impaired consciousness. Rectal temperature measurement is essential to accurately assess core body temperature and confirm the diagnosis of EHS. Aural, oral, skin, temporal, and axillary temperature determinations do not sufficiently differentiate between HE and early EHS [35–38]. Although the risk for EHS is greatest when vigorous sustained exercise is performed during environmental conditions in which the WBGT is greater than 28 ° C, EHS may also strike exercisers in cool (8°–18 ° C) to moderate (18°–28 ° C) environments. This suggests that physical conditioning inadequacies, poor heat acclimatization, viral illnesses, and medications may produce individual variations in EHS susceptibility [39, 40••].

Severe EHS ensues when the core body temperature reaches a critically elevated threshold temperature of 41.6°–42 ° C for a prolonged duration of time, leading to cellular protein denaturation, damage, and interruption of normal mechanisms [11, 41]. Untreated EHS may progress on to multisystem organ damage, rhabdomyolysis, renal failure, acute respiratory distress syndrome, liver damage, hyperkalemia, hypercalcemia, cardiac arrhythmias, hypoglycemia, disseminated intravascular coagulation (DIC), and death.

The "multiple hit" hypothesis proposes that a rapid onset of core body temperature elevation compromises molecular tissues protective mechanisms via the vagus nerve, in conjunction with endotoxin release and the production of pyrogenic cytokines that further interfere with thermoregulation [42–44]. A systemic inflammatory response syndrome (SIRS) may develop leading to vascular endothelial damage and a consumptive coagulopathy via both the fibrinolysis and coagulation pathways [44, 45•]. The cardiovascular system becomes challenged because of blood flow diversion from the viscera to the skin and vital organs, producing intestinal ischemia, increased epithelial membrane permeability, and endotoxin leakage. Additionally, cerebral blood flow may be impaired, causing cerebral edema, cerebellar atrophy, Purkinje layer damage, and a breakdown of the "blood-brain" and "blood-cerebrospinal fluid" barriers.[40••, 45•]

The early recognition and treatment of EHS with whole body cooling is paramount to survival. Body cooling should be instituted as early as possible and before transporting the victim. The gold standard for rapid body cooling is cold water and ice water immersion therapy (water temperature 2°–15 ° C), preferably initiated within 10 minutes, in which case the survival rate is near 100 % [46–48]. Cold water immersion produces a 0.5 ° C lowering of body temperature in the first 3 minutes, after which the rate of body temperature decline typically accelerates. Total immersion cooling typically takes about 20 minutes and should cease when the core body temperature reaches 38.3°–38.9 ° C [11, 46]. Other effective though less rapid cooling methods include the placement of frequently rotating ice water soaked towels on the victim's head, trunk, and extremities, and ice packs on the neck, axillae, and groin; and cool air mist exposure with fanning, a technique that is most valuable when the ambient relative humidity is low [49]. The immediate therapeutic goal is to

reduce core body temperature to less than 38.9 ° C as quickly as possible. The primary difference between mild EHS and severe EHS is determined by the duration of core body temperature elevation. When body cooling is rapid and body core temperature returns to normal range within 1 hour of the onset of symptoms, most EHS victims recover fully [50]. Those who are not cooled down quickly and are exposed to more than a cumulative 60 ° C/minute above the 40.5° body core temperature (eg, 60 minutes at 41.5 ° C, or 30 minutes at 42.5 ° C) are susceptible to severe EHS complications [11, 50, 51]. In addition to rapid body cooling, intravenous normal saline fluid should be administered to improve organ tissue perfusion and facilitate heat exchange, oxygenation, and the removal of waste products. Seizures may be treated with benzodiazepines. Rhabdomyolysis should be managed with IV fluids and diuretics such a mannitol to limit renal cellular destruction. EHS liver damage results in hepatitis and coagulopathy, while heart muscle injury may provoke arrhythmias that often convert to normal once cooling occurs.

There are no known comprehensive and validated guidelines for determining when it is safe for an athlete to return to play after EHS [47, 50]. Experts recommend that return-to-play decisions be based upon the severity of the EHS episode [52]. Minimum recommendations are to abstain from all exercise for at least 7 days, at which time the EHS patient should undergo a follow-up physical examination and recheck of abnormal laboratory and imaging studies [11]. Once cleared for return to physical activity, initial exercise sessions should be conducted in a cool environment, and if well tolerated, exercise intensity and duration can be gradually increased along with progressive heat exposure over 2 weeks to achieve reconditioning and heat acclimatization. The athlete may be considered for full activity clearance once thermal tolerance is achieved and after 2–4 weeks of such training. If return to vigorous activity is delayed because of heat intolerance or a recurrence of symptoms, laboratory exercise thermal testing may be obtained. The Israeli Defense Forces use a standard heat tolerance test (HTT) to assess readiness for return to military activities [53–55, 56•], although experts question the validity and applicability of this test to determine safe sports clearance for athletes [40••]. A new HTT protocol was designed and utilized for an elite triathlete with prior episodes of EHS [57].

Exertional rhabdomyolysis

Exertional rhabdomyolysis (ER) is an acute syndrome of major muscle breakdown provoked by physical trauma, muscle hypoxia, genetic defects, infections, elevations in body temperature, metabolic and electrolyte disturbances, and excessive unaccustomed physical exercise. Muscle breakdown products, electrolytes, myoglobin, and other proteins leak into the blood stream. Serum creatine kinase levels typically rise dramatically. ER presents clinically with muscle pain, soreness, stiffness, and swelling, with progression to the loss of mobility and weakness. Delayed onset muscle soreness (DOMS) because of normal physical exercise training also produces elevations of serum creatine kinase levels and may be considered a non-life threatening mild form of ER. Features that help differentiate life threatening severe ER from DOMS include: muscle pain that is more severe and sustained than expected; swelling of muscles and adjacent tissues; muscle weakness; limitations of active and passive joint range of motion; and the presence of myoglobinuria. “Novel overexertion,” EHS, and exertional collapse in athletes with sickle cell trait are important risk factors for ER. Prior history of EHS (7–11 X increased risk), male gender, African-American ethnicity, younger age, less education, and shorter lengths of military service are specific ER risk factors in soldiers [58].

The treatment of severe ER necessitates body-cooling measures if indicated, aggressive IV rehydration, activation of the EMS, and renal dialysis when required. Coaches and athletes should be educated on the prevention and early recognition of ER secondary to novel overexertion with a particular attention to new strength and conditioning drills, avoidance of high intensity conditioning immediately following breaks from conditioning, gradual progression in the intensity of a training regimen, avoidance of punishment exercise, providing readily available fluids during activity, promoting awareness of urine color to help monitor an athlete’s hydration status, and in the development of an emergency action plan for EHS and ER. Case clusters of ER have been reported in groups of college football players who engaged in intensive conditioning back-squat drills immediately following the winter break [59], high school football players who performed high numbers of repetitions of chair dips alternated with pushups on the first day of summer football camp [60], college swimmers required to perform maximal numbers of squats and pushups repeatedly on the first day of training after summer break [61], college lacrosse players executing maximal sets of biceps curls on the first day following a 3-month training break, and a women’s college lacrosse team following strenuous punishment exercise provoked by the first team loss of a competitive season [62, 63].

Exercise-associated hyponatremia

Exercise-associated hyponatremia (EAH) is a potentially life-threatening condition characterized by a reduced serum sodium level (<135 mg/dL) and mental status changes. Most cases affect endurance athletes who have ingested excessive quantities of hypotonic fluid during prolonged (generally >3 hours) physical activity, causing a dilutional hyponatremia. Consumption of more oral fluids than are lost in the sweat may

actually cause a weight gain during exercise. EAH most commonly affects women, those with short stature, and athletes with longer race times. Early symptoms include vomiting, swelling of the hands and feet, restlessness, confusion, wheezing, and fatigue. Any failure to reverse the low plasma sodium concentration and osmolarity may cause progression to seizures, pulmonary edema, cerebral edema, brainstem herniation, coma, respiratory arrest, and death [64]. Circumstances that create the highest risks for severe EAH complications are low absolute serum sodium value, rapid rates of sodium level declines, and increased duration of hyponatremia.

Sports medicine personnel should be aware of the signs of EAH. These include overdrinking, nausea and vomiting, dizziness, muscle twitching, peripheral tingling or swelling, headache, disorientation, acute mental status changes, physical exhaustion, pulmonary embolus, seizures, and cerebral edema. EAH must be strongly considered in any endurance athlete who develops mental status changes. Portable, rapid serum sodium measuring devices are readily available for use in the field. Upon confirmation, EAH is best managed with intravenous hypertonic saline (3 %–5 %) fluid and immediate hospital emergency department transport. Mild symptoms may be corrected using fluid restriction, salty food consumption, or a small volume of hypertonic intravenous solution.

Decisions on appropriate return to play and prevention of EAH should emphasize the establishment of individualized hydration protocols based on personal sweat rates, sport dynamics, environmental factors, heat acclimatization status, exercise duration, and exercise intensity. Athletes who exercise in hot environments and sweat copiously should be counseled to consume adequate dietary sodium with meals, strive to correct fluid losses accumulated during exercise, and pay attention to body weight changes, urine color, and thirst cues.

Prevention of EHRI

Several professional medical and sports organizations have issued guidelines on the prevention of EHRI during sports participation [10••, 65••, 66••, 67••, 68, 69••, 70, 71••]. The Inter-Association Task Force (IATF) issued guidelines to reduce the risk of EHRI in secondary school athletes, including an emphasis on pre-season heat acclimatization, recommendations on the appropriate duration and intensity of sports practices and gradual appliance of protective equipment that restricts heat dissipation, sideline fluid and ice bags availability, and the importance of a written emergency action plan [65••, 71••]. A recent survey of high school football trainers assessed compliance with these guidelines and found that 20 % of respondents were involved in the management of at least 1 episode of EHS during the past football season (average 0.5±1.37 EHS event/program), and that programs conformed well with recommendations to remove an affected athlete's equipment/clothing, relocate to a shady area, have ice bags/coolers available, and develop a written policy for initiating the Emergency Medical System (EMS). On the other hand, compliance was low with such recommendations as: (1) early activation of EMS for possible EHS (29 %); (2) taking rectal temperatures (<1 %); (3) prompt usage of ice bath immersion rapid body cooling therapy for suspected EHS cases; (4) limits on practice durations; and (5) the gradual introduction of football equipment. States with legislatively mandated pre-season heat acclimatization and EHRI prevention guidelines demonstrated higher rates of accordance [7•, 65••, 72].

The IATF also developed best practice recommendations for reducing sudden death during collegiate and secondary school sports conditioning sessions. These suggestions emphasize gradual acclimatization, slow introduction of new conditioning activities, avoidance of using exercise as punishment, proper education and credentialing of strength and conditioning staff, provision of appropriate medical coverage, development of emergency action plans, and raising awareness of medical conditions that may predispose to EHRI [67••, 71••]. American Academy of Pediatrics guidelines to reduce EHRI risk in children and adolescents additionally encourage strict activity modification if indicated by the measured WBGT, avoidance of exercise if ill or recovering from a febrile illness or gastroenteritis, training of coaches, athletic trainers, and physical education teachers to monitor children and adolescents closely for signs and symptoms of EHRI, provision of 2 hours rest or longer between same day events, and a universal commitment to the safety of the child and adolescent [66••].

Table 2 summarizes EHRI preventative strategies. Heat acclimatization is universally recognized as a critical strategy for the prevention of HRI. Although optimal heat acclimatization requires 60–90 minutes of daily gradually progressive exercise during exposure to environmental heat stress over 10–14 days, even a few days of progressive heat exposure will benefit an athlete who intends to compete in a hot and humid environment [73]. Heat acclimatization results in measurable physiological changes that include increases in plasma volume, sweat rate, and cutaneous vasodilation; and decreases in the sweating threshold, electrolyte content of sweat, heart rate at a given physical workload, and urinary sodium excretion. Successful heat acclimatization increases work capacity in a hot and humid environment, reduces rectal temperature during exercise in the heat, and diminishes the risk for EHRI [74–76]. The NCAA implemented heat acclimatization guidelines in 2003, with the subsequent adoption of similar standards by the National Football League and 13 state high school athletic associations to date [77].

Table 2 Strategies for the prevention of EHRI

- Heat acclimatization
  - Gradual increase in practice duration
  - Gradual increase in practice intensity
  - Gradual increase in environmental heat exposure
  - Gradual introduction of equipment that interferes with heat dissipation
- Maintain hydration status
  - Fluid availability
  - Self-monitoring of hydration status
- Sideline preparation
  - Medical availability
  - Monitoring of WBGT
  - Sideline fluid and ice availability
  - Shade/air conditioning availability
  - Rapid body cooling availability
  - Rectal temperature measurement availability
  - Written emergency action plan
- Education of staff on the early recognition and prevention of EHRI
- Preparticipation physical evaluation and ongoing medical evaluation
  - EHRI risk factor identification
  - Develop preventive strategies for athletes with risk factors
- Practice/competition scheduling
  - Early or late in the day
  - Adequate rest breaks between same-day events
  - Modify or cancel practices/competitions based upon WBGT recommendations
- Maintain good physical conditioning
- Avoid exercise as punishment
- Avoid "novel" overexertion
- Pre-exercise cooling

EHRI exertional heat-related illness, *WBGT* wet-bulb globe temperature.

Maximum sweat rates vary by age, level of conditioning, and the strenuousness of physical activity. Maximal sweat rates range from 300–700 mL/hour in 9–12 year old children, 2.5 liters/hour in elite adolescent tennis players, and as much as 3 liters/hours in a trained 70 kg adult athlete [34, 78]. Investigations of elite male tennis players identified significantly higher rectal temperatures (39.4° vs 38.7°), heart rates, thermal comfort, thermal sensation, and perceived exertion during 2-hour matches played in cool (WBGT = 19°) compared with hot (WBGT = 34°) conditions [79•]. Additionally, time between points averaged 10 seconds longer during hot playing conditions, suggesting a behavioral adaptation to environmental heat stress [79•].

The preparticipation physical evaluation and ongoing medical assessments can help identify individuals who are most susceptible to EHRI. Awareness of known risk factors for EHRI, such as history of prior episodes, sickle cell trait, use of certain medications, and recent febrile illnesses, can assist in determining participation readiness and to provide education and establish preventive strategies to help reduce EHRI risk.

Maintenance of an athlete's hydration status is essential to preserve exercise capacity and reduce the risk of EHRI as each 1 % reduction in body weight because of fluid loss is associated with a core body temperature increase of 0.25 ° C and a heart rate elevation of 6–10 beats per minute [80, 81]. Fluids must be readily available pre-exercise, during exercise, and postexercise. The athlete should self-monitor his/her hydration status with regular assessments of thirst, pre- and postexercise body weights, and observation of urine color. The presence of more than 1 of these self-assessment dehydration markers indicates that dehydration is likely.

Thirst is a valuable indicator of dehydration but does not develop until fluid losses reach 1 %–2 % of body weight. Pre- and postexercise body weight measurements can help guide rehydration needs with each 1 kg of body mass loss during exercise requiring 1 liter of fluid replacement preferably within 2 hours after the completion of exercise. Urine color can be assessed using a urine color chart in which darker urine indicates a greater level of dehydration. Additionally, urine specific gravity determination is may be helpful with levels of $\leq$1.020 indicating a euhydrated state.

Adequate sodium intake is particularly essential during periods of exercise in conditions of high environmental heat stress as sodium sweat losses must be replaced and salt ingestion promotes the rehydration process. The consumption of plain water causes a rapid fall in plasma sodium concentration and osmolarity leading to decreased vasopressin and aldosterone activity with concurrent increases in urinary output [82–84]. As ingested sodium enters the cells, restoration of extracellular fluid volume occurs via osmosis. Salt consumption additionally increases thirst and oral rehydration fluid palatability, which encourages the voluntarily consumption of more fluids [82]. Intake of carbohydrate containing beverages (3 %–12 % concentration) and carbohydrate-protein containing beverages after exertion induced dehydration offer additional advantages in promoting fluid retention and thermoregulation capacities compared with water alone [85–87]. A meta-analysis suggests that glycerol consumption can induce a state of hyperhydration by increasing fluid retention and potentially reduce EHRI risk [17•, 88]. Cool or cold beverages for ingestion are preferred to room temperature fluids, and result in a 50 % greater consumption of liquids during exercise [89].

Pre-game voluntary intravenous hyperhydration is a common practice in asymptomatic professional and collegiate football players, although existing evidence does not support a role in EHRI [90, 91]. A survey of NCAA Division I college football athletic trainers disclosed that 30 % of programs used pre-game intravenous fluids, administered to an average of 2–3 players per game, with the intended purpose of preventing

muscle cramps, heat illness, and dehydration. The primary care team physician most commonly administered intravenous fluids, 47 % were given at player request, and 24 % reported the occurrence of intravenous infusion-related complications [90].

The WBGT should be strictly monitored before and during scheduled sports activities to assess environmental heat stress and determine EHRI risk levels [11, 65••]. Thresholds and guidelines used to restrict or cancel sports practices and events vary by organizations and sports governing bodies [70]. Tennis officials at the January 2014 Australian Open in Melbourne cited the "Extreme Heat Policy" when deciding to suspend play for several hours because of center court temperatures, which soared to 43 ° C [92]. A Brazilian judge ruled that mandatory water breaks should be implemented in the 2014 FIFA World Cup soccer tournament if the WBGT exceeds 32 ° C, a ruling that first affected the United States vs Portugal match in Manaus on June 22, 2014. The risks of EHRI can be reduced by scheduling exercise sessions/competitions during the early or late hours of the day to minimize the effects of mid-day higher ambient temperatures and levels of solar radiation. Clothing and protective equipment should be appropriate for the activity and designed to facilitate the dissipation of body heat through sweat evaporation.

Among body pre-exercise cooling strategies considered to possibly counteract the adverse effects of exercise-related elevated core body temperatures are cold-water immersion and the use of cooling vest garments. Both have been shown to enhance exercise performance by some but not others. A recent investigation found that external cooling vests worn by football players during practice breaks in warm and humid environmental conditions did not produce significantly lower core body temperatures [93–95]. A recent meta-analysis of exercise pre-cooling and cooling during exercise detected a combined 6.7 %±0.9 % positive effect of using these techniques on exercise performance. Additionally, pre-cooling led to a statistically significant reduction in postexercise core body temperature compared with controls, although cooling during exercise did not show a similar effect [96]. Evidence suggests a role for pre-cooling using cold-water immersion to reduce the risk of EHRI in susceptible individuals. Limits should be placed on the long pre-exercise warm-up as an additional strategy to reduce EHRI risk as this can lead to an elevation in the core body temperature, undesirable level of body heat storage, reduced performance, and excessive heat strain. Timing of the warm-up should be conducted so as to allow ample time to restore normal body temperature and to achieve full rehydration. Finally, the Heat Illness Symptom Index has been validated in college football players as a tool to detect early EHRI based upon self-reporting of subjective symptoms [97].

Evidence-based future research is necessary to provide further guidance in the prevention of EHRI. Ideas for research include the characterization of true thermal cardiovascular strain profiles, determination of sports specific safety thresholds for restricting or canceling sports practices and competitions, and optimization fluid pre-exercise, intra-exercise, and postexercise hydration. Additional efforts should help to clarify understanding of ideal pre-cooling and cooling during competition tactics based upon the influences of genetic differences to heat stress physical performance and acclimatization responses [10••].

Conclusions

EHRI includes several medical conditions that affect physically active persons who engage in exercise during circumstances of high environmental heat stress. The various forms of EHRI may be differentiated based upon clinical presentations, physical findings, and laboratory determinations. EAC, the most common medical condition presenting to marathon medical tents, may only be diagnosed after ruling out EHS and EAH, and typically responds readily to supine positioning with the leg elevated. EAMC are associated with normal to moderately elevated core body temperatures and frequently involve exertion in the heat with mild to moderate dehydration and excessive sweat salt loss.

Rectal temperature measurement is essential to differentiate between HE (<40 ° C) and EHS (>40 ° C) as the initial clinical presentations are often similar and the management strategies differ substantially. EHS is the most severe form of EHRI and is characterized by a marked elevation in core body temperature of (>40 ° C) and mental status changes. Rapid body cooling must be implemented immediately to reduce EHS morbidity and mortality. EAH manifests with mental status changes and serum sodium levels of less than 135 mg/dL. Serum sodium abnormalities must be rapidly corrected in the presence of mental status changes.

Several professional medical organizations and sports governing bodies have issued guidelines on the prevention of EHRI. Heat acclimatization is widely recognized as a critical strategy to help prevent EHRI. Additional useful preventive practices include fluid availability and maintenance of adequate hydration, having an emergency action plan for cases of EHS, modifying or cancelling exercise sessions under circumstances of high environmental heat stress, awareness of known EHRI risk factors such as sickle cell trait and current of recent febrile illnesses, and education of sports medicine personnel, coaches, and athletes on the early recognition, management, and prevention of EHRI. Future research should provide evidence-based recommendations to further reduce the risks of EHRI in athletes.

Compliance with ethics guidelines

Conflict of interest Andrew Nichols declares that he has no conflict of interest.

Human and animal rights and informed consent No human or animal studies performed by the authors. This article does not contain any studies with human or animal subjects performed by any of the authors.

References

Papers of particular interest, published recently, have been highlighted as:
- • Of importance
- •• Of major importance

1.• Maron BJ, Doerer JJ, Haas TS, Tierney DM, Mueller FO. Sudden deaths in young competitive athletes: analysis of 1866 deaths in the United States, 1980-2006. Circulation. 2009;119:1085. *This paper contains an analysis of data from the largest registry of sudden death cases in United States competitive athletes over the past few decades. Causes, numbers, and epidemiology of deaths are detailed.*

2. Mueller FO, Cantu RC. Catastrophic sports injury research: 29th Annual Report Fall 1982-Spring 2011. University of North Carolina, Chapel Hill 2008. Available at: http://www.unc.edu/depts/nccsi/AllSport.pdf. Accessed 8 May 2014.

3.• CDC. Heat illness among high school athletes—United States, 2005-2009. MMWR. 2010;59:1009–13. *This report provides specific epidemiology data from the National High School Sports-Related Injury Surveillance Study on EHRI conditions among United States high school athletes during a recent 5-year period.*

4. Mueller FO, Cantu RC. Catastrophic sports injury research: 26th Annual Report Fall 1982-Spring 2008. University of North Carolina, Chapel Hill 2008. Available at: http://www.unc.edu/depts/nccsi/AllSport.pdf. Accessed 8 May 2014.

5.• Boden BP, Breit I, Beachler JA, Williams A, Mueller FO. Fatalities in high school and college football players. Am J Sports Med. 2013;41:1108–16. *Authors analyzed data from the National Center for Catastrophic Sports Injury research to describe the causes and incidences of fatalities in United States football high school and college football players between 1990 and 2010.*

6.• Kerr ZY, Marshall SW, Comstock RD, Casa DJ. Exertional heat stroke management strategies in United States high school football. Am J Sports Med. 2014;42:70–7. *Results of a survey of United States high school athletic trainers to estimate the incidence of EHS cases and the utilization of EHS management strategies during preseason high school football activities.*

7.• Kerr ZY, Casa MA, Marshall SW, Comstock D. Epidemiology of Exertional Heat Illness Among U.S. High School Athletes. Am J Prev Med. 2013:8–14. *This article analyzes National High School Sports-Related Injury Surveillance System data from 2005-2011 to describe the epidemiology of EHRI in high school athletes.*

8. US Consumer Product Safety Commission. NEISS All Injury Program: sample design and implementation. Washington, DC: US Consumer Product Safety Commission; 2001.

9. CDC. Nonfatal sports and recreation heat illness treated in hospital emergency departments—United States, 2001-2009. MMWR. 2011;60:977–80.

10.•• Bergeron MF, Bahr R, Bärtsch P, Bourdon L, Calbet JA, Carlsen KH, et al. International Olympic Committee consensus statement on thermoregulatory and altitude challenges for high-level athletes. Br J Sports Med. 2012;46:770–9. *Provides a consensus review of thermoregulatory (and other environmental) challenges faced by elite athletes, with recommendations for the management, prevention, and future needed evidence-based research of EHRI.*

11. Armstrong LE, Casa DJ, Millard-Stafford M, Moran DS, Pyne SW, Roberts WO. American College of Sports Medicine position stand. Exertional heat illness during training and competition. Med Sci Sports Exerc. 2007;39:556–72.

12. Alosco ML, Knecht K, Glickman E, Bergeron MF, Gunstad J. History of concussion and exertional heat illness symptoms among college athletes. Int J Athletic Ther Training. 2012;17:22–7.

13. Muldoon S, Deuster P, Voelkel M, Capacchione J, Bunger R. Exertional heat illness, exertional rhabdomyolysis, and malignant hyperthermia: is there a link? Curr Sports Med Rep. 2008;8:74–80.

14. Junglee NA, Di Felice U, Dolci A, Fortes MB, Jibani MM, Lemmey AB, et al. Exercising in a hot environment with muscle damage: effects on acute kidney injury biomarkers and kidney function. Am J Physiol Renal Physiol. 2013;305:F813–20.

15.• Fortes MB, Di Felice U, Dolci U, Dolci A, Junglee NA, Crockford MJ, et al. Muscle-damaging exercise increases heat strain during subsequent exercise heat stress. Med Sci Sports Exerc. 2013;45:1915–24. *This article provides evidence that exercise-induced muscle damage may independently predispose athletes to EHRI.*

16. Larose J, Boulay P, Sigal RJ, Wright HE, Kenny GP. Age-related decrements in heat dissipation during physical activity occur as early as the age of 40. PLoS One. 2013;8:e83148.

17.• Lopez RM, Casa DJ. The influence of nutritional ergogenic aids on exercise heat tolerance and hydration status. Curr Sports Med Rep. 2009;8:192–9. *This article provides a comprehensive review of the effects of various nutritional ergogenic aids on heat tolerance and susceptibility to dehydration.*

18. Bruning RS, Dahmus JD, Kenney WL, Alexander LM. Aspirin and clopidogrel alter core temperature and skin blood flow during heat stress. Med Sci Sports Exerc. 2013;45:674–82.

19. Morton RH. Effects of caffeine, ephedrine and their combination on time to exhaustion during high-intensity exercise. Eur J Appl Physiol Occup Physiol. 1999;80:610–2.

20. Lopez RM, Casa DJ, McDermott BP, Ganio MS, Armstrong LE, Maresh CM. Does creatine supplementation hinder exercise heat tolerance or hydration status? A systematic review with meta analyses. J Athl Train. 2009;44:215–23.

21. Cheuvront SN, Carter R, Kolka MA, Lieberman HR, Kellog MD. Branched-chain amino acid supplementation and human performance when hypohydrated in the heat. J Appl Physiol. 2004;97:1275–82.

22. Howe AS, Boden BP. Heat-related illness in athletes. Am J Sports Med. 2007;35:1384–92.

23. Roberts WO. A 12-yr profile of medical injury and illness for the Twin Cities Marathon. Med Sci Sports Exerc. 2000;32:1549–55.

24. Holtzhausen LM, Noakes TD. The prevalence and significance of postexercise (postural) hypotension in ultramarathon runners. Med Sci Sports Exerc. 1995;27:1595–601.

25. Roberts WO. Determining a "do not start" temperature for a marathon on the basis of adverse outcomes. Med Sci Sports Exerc. 2010;42:226–32.

26. Crandall CG. Heat stress and baroreflex regulation of blood pressure. Med Sci Sports Exerc. 2008;40:2063–70.

27.• Aspslund CA, O'Connor FG, Noakes TD. Exercise-associated collapse: an evidence-based review and primer for clinicians. Br J Sports Med. 2011;45:1157–62. *The authors provide a thorough review of current evidence-based evidence concerning the evaluation, management, and prevention of exercise-associated collapse in the endurance athlete.*

28. Privett SE, George KP, Whyte GP, Cable NT. The effectiveness of compression garments and lower limb exercise on post-exercise

blood pressure regulation in orthostatically intolerant athletes. Clin J Sport Med. 2010;20:362–7.
29. Casa DJ, Clarkson PM, Roberts WO. American College of Sports Medicine roundtable on hydration and physical activity: consensus statements. Curr Sports Med Rep. 2005;4:115–27.
30. Bartok C, Schoeller DA, Clark RR, Sullivan JC, Clark RR, Landry GL. The effect of dehydration on wrestling minimal weight assessment. Med Sci Sports Exerc. 2004;36:160–7.
31. Stofan JR, Zachwieja JJ, Horswill CA, Murray R, Anderson SA, Eichner ER. Sweat and sodium losses in NCAA Division I football players with a history of whole-body muscle cramping. Med Sci Sports Exerc. 2003;35:S48.
32. Layzer RB. The origin of muscle fasciculations and cramps. Muscle Nerve. 1994;17:1243–9.
33. Bergeron MF. Heat cramps during tennis: a case report. Int J Sport Nutr. 1996;6:62–8.
34. Bergeron MF. Heat cramps: fluid and electrolyte challenges during tennis in the heat. J Sci Med Sport. 2003;6:19–27.
35. Armstrong LE, Maresh CM, Crago AE, Adams R, Roberts WO. Interpretation of aural temperatures during exercise, hyperthermia, and cooling therapy. Med Exerc Nut Health. 1994;3:9–16.
36. Deschamps A, Levy RD, Cosio MG, Marliss EB, Magder S. Tympanic temperature should not be used to assess exercise induced hyperthermia. Clin J Sports Med. 1992;2:27–32.
37. Ronneberg K, Roberts WO, McBean AD, Center BA. Temporal artery and rectal temperature measurements in collapsed marathon runners. Med Sci Sports Exerc. 2008;40:1373–5.
38. Huggins R, Glaviano N, Negishi N, Casa DJ, Hertel J. Comparison of rectal and aural core body temperature thermometry in hyperthermic, exercising individuals: a meta-analysis. J Athl Train. 2012;47:329–38.
39. Rae DE, Knobel GJ, Mann T, Swart J, Tucker R, Noakes TD. Heatstroke during endurance exercise: is there evidence for excessive endothermy? Med Sci Sports Exerc. 2008;40:1193–204.
40.•• O'Connor FG, Casa DJ, Bergeron MF, Carter R, Deuster P, Heled Y, et al. American College of Sports Medicine roundtable on exertional heat stroke—return to duty/return to play: conference proceedings. Curr Sports Med Rep. 2010;9:314–21. *This article summarizes ACSM conference proceedings that review the literature on return to play/return to duty in individuals who have experienced EHRI events. Experts provide consensus recommendations on guidelines for return and directions for future research.*
41. Seto C, Way D, O'Connor N. Environmental illness in athletes. Clin Sports Med. 2005;24:695–718.
42. Sonna LA, Sawka MN, Lilly CM. Exertional heat illness and human gene expression. Prog Brain Res. 2007;162:321–46.
43. Sonna LA, Wenger CB, Flinn S, Sheldon HK, Sawka MN, Lilly CM. Exertional heat injury and gene expression changes: a DNA microarray analysis study. J Appl Physiol. 2004;96:1943–53.
44. Bouchama A, Knochel JP. Heat stroke. N Engl J Med. 2002;346:1978–88.
45.• Leon LR, Helwig BG. Heat stroke: role of the systemic inflammatory response. J Appl Physiol. 2010;109:1980–8. *This article provides a thorough discussion of the mechanisms and processes that contribute to the pathophysiology of EHS.*
46. Casa DJ, McDermott BP, Lee EC, Yeargin SW, Armstrong LE, Maresh CM. Cold water immersion: the gold standard for exertional heatstroke treatment. Exer Sport Sci Rev. 2007;35:141–9.
47. Bouchama A, Dehbi M, Chaves-Carballo E. Cooling and hemodynamic management in heatstroke: practical recommendations. Crit Care. 2007;11:R54.
48. Taylor NA, Caldwell JN, Van den Heuvel AM, Patterson MJ. To cool, but not too cool: that is the question—immersion cooling for hyperthermia. Med Sci Sports Exerc. 2008;40:1962–9.
49. McDermott BP, Casa DJ, Yeargin SW, Ganio MS, Armstrong LE, Maresh CM. Recovery and return to activity following exertional heat stroke: considerations for the sports medicine staff. J Sport Rehabil. 2007;16:163–81.
50. Costrini A. Emergency treatment of exertional heatstroke and comparison of whole body cooling techniques. Med Sci Sports Exerc. 1990;22:15–8.
51. Armstrong LE, Deluca JP, Hubbard RW. Time course of recovery and heat acclimation ability of prior exertional heatstroke patients. Med Sci Sports Exerc. 1990;22:36–48.
52. O'Connor FG, Williams AD, Blivin S, Heled Y, Deuster P, Flinn SD. Guidelines for return to duty (play) after heat illness: a military perspective. J Sport Rehabil. 2007;16:227–37.
53. Moran DS, Heled Y, Still L, Laor A, Shapiro Y. Assessment of heat tolerance for post exertional heat-stroke individuals. Med Sci Mon. 2004;10:CR252–7.
54. Robinson S, Wiley SL, Boudarant LG, Mamlin S. Temperature regulation of men following heatstroke. Isr J Med Sci. 1976;12:786–95.
55. Moran DS, Erlich T, Epstein Y. The heat tolerance test: an efficient screening tool for evaluating susceptibility to heat. J Sport Rehabil. 2007;6:215–21.
56.• Kazman JB, Heled Y, Lisman PJ, Druyan A, Deuster PA, O'Connor FG. Exertional heat illness: the role of heat tolerance testing. Curr Sports Med Rep. 2013;12:101–5. *This paper provides a review of heat tolerance testing that is applicable to the athlete who has sustained an episode of EHRI.*
57. Johnson EC, Kolkhorst FW, Richburg A, Schmitz A, Martinez J, Armstrong LE. Specific exercise heat stress protocol for a triathlete's return from exertional heat stroke. Curr Sports Med Rep. 2013;12:106–9.
58. Hill OT, Wahi MM, Carter R, Kay AB, McKinnon CJ, Wallace RF. Rhabdomyolysis in the US active duty Army, 2004-2006. Med Sci Sports Exerc. 2012;44:442–9.
59. Smoot MK, Amendola A, Cramer E, Doyle C, Kregel KC, Chiang HY, et al. A cluster of exertional rhabdomyolysis affecting a Division I football team. Clin J Sports Med. 2013;23:365–72.
60. Oh JY, Laidler M, Fiala SC, Hedberg K. Acute rhabdomyolysis and triceps compartment syndrome during high school football camp. Sports Health. 2012;4:57–62.
61. Galvez G, Stacy J, Howley A. Exertional rhabdomyolysis in seven Division-I swimming athletes. Clin J Sports Med. 2008;18:366–8.
62. Eichner ER. An outbreak of muscle breakdown: a mortality play in four acts. Curr Sports Med Rep. 2010;9:325–6.
63. Cleary MA, Sadowski KA, Lee SY, Miller GL, Nichols AW. Exertional rhabdomyolysis in an adolescent athlete during preseason conditioning: a perfect storm. J Strength Cond Res. 2011;25:3506–13.
64. Murray B, Eichner R. Hyponatremia of exercise. Curr Sports Med Rep. 2004;3:117–8.
65.•• Casa DJ, Csillan D. Preseason heat-acclimatization guidelines for secondary school athletics. J Athl Training. 2009;44:332–3. *The Inter-Association Task Force for Preseason Secondary School Athletics offers specific definitions and recommendations for the 14-day pre-season heat acclimatization period.*
66.•• American Academy of Pediatrics Policy Statement—Climatic heat stress and exercising children and adolescents. Pediatrics. 2011;128:e741–7. *The AAP Council on Sports Medicine and Fitness Council on School Health issued a policy and recommendations to improve safety and minimize the risk of EHRI for children and adolescents during exercise and sports participation.*
67.•• Casa DJ, Anderson SA, Baker L, Bennett S, Bergeron MF, Connolly D, et al. The Inter-Association Task Force for preventing sudden death in collegiate conditioning sessions: best practices recommendations. J Athl Training. 2012;47:477–80. *This article is a consensus statement of the Inter-Association Task Force that offers 10 specific conditioning recommendations intended to*

reduce conditioning-related morbidity and mortality in college athletes.

68. Bahr R, Reeser JC. New guidelines are needed to manage heat stress in elite sports—The Federation Internationale de Volleyball (FIVB) Heat Stress Monitoring Programme. Br J Sports Med. 2012;46:805–9.
69.•• Casa DJ, Guskiewicz KM, Anderson SA, Courson RW, Heck JF, Jiminez CC, et al. National Athletic Trainers' Association Position Statement: preventing sudden death in sports. J Athl Training. 2012;47:96–118. *This position statement of the National Athletic Trainers' Association presents specific guidelines for athletic trainers for the prevention, screening, recognition, and management of the most common conditions that cause sudden death in sports participation. Specific recommendations are given for EHS and exertional hyponatremia*.
70. Mountjoy M, Alonso JM, Bergeron MF, Dvorak J, Miller S, Migliorini S, et al. Hyperthermic-related challenges in aquatics, athletics, football, tennis, and triathlon. Br J Sports Med. 2012;46:800–4.
71.•• Casa DJ, Almquist J, Anderson SA, Baker L, Bergeron MF, Biagioli B, et al. The inter-association task force for preventing sudden death in secondary school athletics programs: best-practices recommendations. J Athl Train. 2013;48:546–53. *This consensus statement of the Inter-Association Task Force presents specific best practice guidelines for the prevention, screening, recognition, and management of the most common conditions that cause sudden death in secondary school athletes. Specific recommendations are given for EHS and exertional hyponatremia*.
72. Kerr ZY, Marshall SW, Comstock RD, Casa DJ. Implementing exertional heat illness prevention strategies in US high school football. Med Sci Sports Exerc. 2014;46:124–30.
73. Grantham J, Cheung SS, Connes P, Febbraio MA, Gaoua N, Gonzalez-Alonso J, et al. Current knowledge on playing football in hot environments. Scand J Med Sci Sports. 2010;20 Suppl 3:161–7.
74. Lorenzo S, Halliwill JR, Sawka MN, Minson CT. Heat acclimation improve exercise performance. J Appl Physiol. 2010;109:1140–7.
75. Buchheit M, Voss SC, Nybo L, Mohr M, Racinais S. Physiological and performance adaptations to an in-season soccer camp in the heat: associations with heart rate and heart rate variability. Scand J Med Sci Sports. 2011;21:e477–8.
76. Daanen HA, Jonkman AG, Layden JD, Linnane DM, Weller AS. Optimising the acquisition and retention of heat acclimation. Int J Sports Med. 2011;32:822–8.
77. Available at: http://ksi.uconn.edu/prevention-strategies/high-school-state-policies/heat-acclimatization-state-policies/. Accessed 12 Aug 2014.
78. Bergeron MF, Waller JL, Marinik EL. Voluntary fluid intake and core temperature responses to adolescent tennis players: sports beverages vs water. Br J Sports Med. 2006;40:406–10.
79.• Périard JD, Racinais S, Knez WL, Herrera CP, Christian RJ, Girard O. Thermal, physiological and perceptual strain mediate alterations in match-play tennis under heat stress. Br J Sports Med. 2014;48:i32–8. *Compared physiological responses, thermal observations, and exertional perceptions of elite male tennis players under cool and hot environmental conditions*.
80. Casa DJ, Armstrong LE, Hillman SK, Montain SJ, Reiff RV, Rich BS, et al. National Athletic Trainers' Association position statement: fluid replacement for athletes. J Athl Train. 2000;35:212–24.
81. Sawka MN, Burke LM, Eichner ER, Maughan RJ, Montain SJ, Stachenfeld NS. American College of Sports Medicine position stand: exercise and fluid replacement. Med Sci Sports Exerc. 2007;39:377–90.
82. Maughan RJ, Leiper JB. Sodium intake and post-exercise rehydration in man. Eur J Appl Physiol Occup Physiol. 1995;71:311–9.
83. Shirreffs SM, Maughan RJ. Volume repletion after exercise induced volume depletion in humans: replacement of water and sodium losses. Am J Physiol Renal Physiol. 1998;274:868–75.
84. Rehrer N. Fluid and electrolyte balance in ultra-endurance sport. Sports Med. 2001;31:701–15.
85. Clapp A, Bishop P, Smith J, Mansfield E. Effects of carbohydrate-electrolyte content of beverages on voluntary hydration in a simulated industrial environment. Am Ind Hyg Assoc J. 2000;61:692–9.
86. Osterberg KL, Pallardy SE, Johnson RJ, Horswill CA. Carbohydrate exerts a mild influence on fluid retention following exercise-induced dehydration. J Appl Physiol. 2010;108:245–50.
87. Okazaki K, Goto M, Nose H. Protein and carbohydrate supplementation increases aerobic and thermoregulatory capacities. J Physiol. 2009;587:5585–90.
88. Goulet ED, Gauthier P, Labrecque S, Royer D. Glycerol hyperhydration, endurance performance, and cardiovascular and thermoregulatory responses: a case study of a highly trained triathlete. JEP Online. 2002;5:19–28.
89. Burdon CA, Johnson NA, Chapman PG, O'Connor HT. Influence of beverage temperature on palatability and fluid ingestion during endurance exercise: a systematic review. Int J Sport Nutr Exerc Metab. 2012;22:199–211.
90. Gesik NY, Tan SK, Prentiss GT, Fitzsimmons S, Nichols AW. The use of pregame hyperhydration with intravenous fluids in national collegiate athletic association football bowl subdivision teams. Clin J Sport Med. 2013;23:488–90.
91. Fitzsimmons S, Tucker A, Martins D. Seventy-five percent of National Football League teams use pregame hyperhydration with intravenous fluid. Clin J Sports Med. 2011;21:192–9.
92. Périard JD, Bergeron MF. Competitive match-play tennis under heat stress: a challenge for all players. Br J Sports Med. 2014;48:i1–3.
93. Ranalli GF, Demartini JK, Casa DJ, McDermott BP, Armstrong LE, Maresh CM. Effect of body cooling on subsequent aerobic and anaerobic exercise performance: a systematic review. J Strength Cond Res. 2010;24:3488–96.
94. Cleary MA, Toy MG, Lopez RM. Thermoregulatory, cardiovascular, and perceptual responses to intermittent cooling during exercise in a hot, humid outdoor environment. J Strength Cond Res. 2014;28:792–806.
95. Lopez RM, Eberman LE, Cleary MA. Superficial cooling does not decrease core body temperature before, during, or after exercise in an American football uniform. J Strength Cond Res. 2012;26:3432–40.
96. Bongers CC, Thijssen DH, Veltmeijer MT, Hopman MT, Eijsvogels TM. Precooling and precooling (cooling during exercise) both improve performance in the heat: a meta-analytical review. Br J Sports Med. 2014.
97. Coris EE, Walz SM, Duncanson R, Ramirez AM, Roetzheim RG. Heat illness symptom index (HISI): a novel instrument for the assessment of heat illness in athletes. South Med J. 2006;99:340–5.