Heat Stroke in Physical Activity and Sport

By: Yuri Hosokawa, William M. Adams, Rebecca L. Stearns, and Douglas J. Casa

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Keywords: Risk factors | rectal thermometry | treatment | cold water immersion | prevention

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HEAT STROKE IN PHYSICAL ACTIVITY AND SPORTS

(Original version in English)[1]

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Hosokawa, Y., Adams, W. M., Steams, R. L., & Casa, D. J. (2014). Heat stroke in physical activity and sports. **PENSAR EN MOVIMIENTO: Revista de Ciencias del Ejercicio y la Salud, 12 (2),** 1-22. Exertional heat stroke (EHS) is one of the leading causes of sudden death in sport and physical activity. In American Football alone, there have been 46-documented EHS fatalities in the United States between 1995 and 2010. In 2003, National Collegiate Athletics Association mandated pre-season heat acclimatization guidelines, which successfully decreased the number of heat stroke fatalities in collegiate American football. However, despite the advancement in modern medical care and increased awareness in heat safety, lack of appropriate on-site medical care is still contributing to EHS seen especially at the youth level. It is well established in scientific literature that fatalities as a result of EHS are largely preventable with proper education on the knowledge of recognition, treatment, and prevention of EHS. This document provides a review of the current best medical practices and evidence on the epidemiology, pathophysiology, risk factors, recognition, treatment, prevention, and return to play recommendations for EHS, specifically as they relate to sport and physical activity.

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Exertional heat stroke (EHS) is one of the leading causes of sudden death in sport and physical activity (Casa, Guskiewicz, et al., 2012; Casa et al., 2014; Howe & Boden, 2007). Despite the advancement in modern medical care, lack of appropriate, on-site care continues for EHS patients (Rae et al., 2008; Rav-Acha, Hadad, Epstein, Heled, & Moran, 2004). It is well established in scientific literature that fatalities as a result of EHS in an organized sport setting are largely preventable with proper education, and the availability of medical providers knowledgeable in the recognition and treatment of EHS (Casa, Armstrong, Kenny, O'Connor, & Huggins, 2012; Casa et al., 2014; DeMartini et al., 2014). This document will provide a review of the current best medical practice and evidence on the epidemiology, pathophysiology, risk factors, recognition, treatment, prevention, and return to play recommendations for EHS, specifically as it relates to organized sport settings.

Epidemiology

Although incidence of EHS has received increased attention over the past decade, especially in areas such as American football, endurance athletic events and the military, true incidence of EHS is unknown (Epstein & Roberts, 2011), particularly in less developed countries. This is most likely due to a number of issues: the misdiagnosis of the condition, the lack of a universal reporting system (primarily in the athletics setting), and the lack of standardized guidelines to categorize these deaths (Bouchama & Knochel, 2002).

From the years 2005 to 2009, the incidence of EHS in the military was reported to be 0.22 to 0.27 per 1,000 person-years (Epstein & Roberts, 2011). In 2009 alone, the United States Armed Forces Health Surveillance Center reported 323 cases of EHS out of 2,361 cases of exertional heat illness (EHI) (Epstein & Roberts, 2011). From 2009 to 2013 the incidence of EHS in United States military service members was 0.23 per 1,000 soldiers, with the Army and Marine Corps reporting 0.40 and 0.44 EHS cases per 1,000 soldiers respectively (Armed Forces Health Surveillance Center (AFHSC), 2014). Regarding the United States military members serving in Iraq and Afghanistan, 6.4% (58/909) of the reported heat illnesses from 2009 to 2013 were the result of EHS (Armed Forces Health Surveillance Center (AFHSC), 2014).

Examining endurance athletic events, the incidence rate of EHS during an 11.3km warm weather road race was reported to be as high as 2.13 per 1,000 finishers (DeMartini, Casa, Belval et al., 2014). This particular road race has seen an average of 15.2 (13.0) cases of EHS per year over an 18 year period and the number of EHS patients has been shown to highly correlate with high ambient temperature and relative humidity (DeMartini, Casa, Stearns et al., 2015). Other work by Roberts et al. (2000) assessed the incidence of EHS during a marathon to be 1-2 cases per 10,000 race participants.

The incidence of EHS in American football has steadily increased over the past 35 years with the most deadly time period occurring in the 5 year block from 2005 to

2009 (Brady, 2011; Mueller & Cantu, 2011). Between the years of 1980 and 2009 there were 58 EHS deaths in American football, accounting for roughly two deaths per year. Examining this time frame (1980-2009) in 15-year blocks demonstrates the increasing trend in the number of EHS deaths in American football. There was an average of 1.07 deaths per year from 1980 to 1994 and 2.8 deaths per year from 1995 to 2009, with 2008 having 6 deaths alone (Grundstein et al., 2012). The increasing number of EHS deaths among American football athletes is focused primarily at the high school level versus that at the collegiate level (Mueller & Cantu, 2011). Of the 46 EHS deaths occurring in American football between 1995 and 2010, 35 deaths occurred at the high school level compared to collegiate, professional, and sandlot (leagues with no affiliation) levels (Mueller & Colgate, 2011). In addition, American football accounted for 74.4% of the total cases of EHI at the high school level and was 11.4 times more likely to occur than all other sports combined (Kerr, Casa, Marshall, & Comstock, 2013). Prior to 2003, American football at the collegiate level saw an average of 2 deaths per year from EHS. In 2003, the National Collegiate Athletics Association mandated heat acclimatization guidelines for all pre-season sanctioned practices; there have only been two EHS deaths in American football at the collegiate level since that time.

Pathophysiology

Body temperature is maintained around 37°C and is tightly controlled by the central nervous system, particularly the preoptic anterior hypothalamus (Bracker, 1991; Brewster, O'connor, & Lillegard, 1995; Haymes & Wells, 1986). More recently, evidence also supports that the dorsomedial hypothalamus is as important as the preoptic anterior hypothalamus in assisting in thermoregulation (Morrison, Nakamura, & Madden, 2008). During exercise, body temperature is increased by metabolically produced heat from the working muscles. If the environmental temperature is higher than skin temperature, the environment will also contribute to increases in body temperature as opposed to allowing heat to dissipate from the body. In response to the increased body temperature from exercise, thermoregulatory mechanisms within the body will act to balance heat gain and heat loss to maintain equilibrium (Johnson, 1993; Kenney, 1996; Rowell, 1983; Sawka & Wenger, 1988). Cutaneous vasodilation, visceral vasoconstriction, increased cardiac output (heart rate and stroke volume), evaporation of sweat from the skin and respiratory rate are physiological responses which occur to allow management of heat stress and regulation of body temperature (Bracker, 1991; Brewster et al., 1995; Casa et al., 2014; Davidson, 1985; Haymes & Wells, 1986; Kenney, 1996).

Heat gain and heat loss are achieved through conduction, convection, evaporation, and radiation; the process is represented with the equation $S=M\pm R\pm K\pm C_V-E$, where S represents the amount of heat stored, M is the metabolic heat produced, R, K, and C_V is the amount of heat lost or gained by radiation, conduction and convection respectively, and E represents evaporative heat loss (Galaski, 1985; Werner,

n.d.). During exercise, especially in the heat, the primary and most effective mechanism for heat loss is through the evaporation of sweat from the skin (Kenney, 1996). However, as relative humidity increases in the environment, the ability to dissipate heat through evaporation of sweat is greatly reduced (Bracker, 1991; Brewster et al., 1995; Cabanac & White, 1995).

When the athlete is unable to dissipate heat at the rate at which it is produced during exercise, the body experiences uncompensable heat stress that can lead to thermoregulatory failure causing EHS (Bouchama & Knochel, 2002; Epstein & Roberts, 2011; Epstein, Hadad, & Shapiro, 2004). During exercise in the heat, there is a large increase in skin blood flow with a coinciding decrease in visceral blood flow (Rowell, 1983). The shunting of blood from the core allows more blood to go to the skin to dissipate heat (Epstein & Roberts, 2011; Leon & Helwig, 2010). However, the shunting of blood from the visceral organs during heat stress subjects the organs to hypoxia which leads to hyperpermeability within the gastrointestinal tract, which in turn allows endotoxins to be released into the systemic circulation (Hall et al., 2001; Lambert, 2004, 2008; Moseley, Gapen, Wallen, Walter, & Peterson, 1994; Pals, Chang, Ryan, & Gisolfi, 1997; Rowell, 1983; Shapiro, Alkan, Epstein, Newman, & Magazanik, 1986; Smith, 2004). This release of endotoxins activates the body's inflammatory response, and together with the circulatory overload from the heat stress, it can overwhelm the thermoregulatory system (Lambert, 2004, 2008; Leon, 2007). This cascade of events contributes to the uncompensable increase in body temperature leading to EHS (Bouchama & Knochel, 2002; Epstein & Roberts, 2011; Smith, 2004).

Risk Factors

Scientific literature has identified many risk factors associated with EHS (Casa, Armstrong, Ganio, & Yeargin, 2005; Rav-Acha et al., 2004). A majority of risk factors can be avoided or mitigated with prior screening or modifications to the location, time, intensity, and type of the exercise. Consequently, it becomes crucial for the medical provider and supervisor to be aware of these factors and implement strategies to minimize the risk of EHS.

Lack of heat acclimatization, dehydration, intensity of the activity, fitness level, body composition, predisposing medical conditions, prior history of EHI, sleep deprivation, environmental conditions, type of clothing and equipment worn, and lack of knowledge, may all contribute to the risk of EHS. These factors can be further classified into intrinsic and extrinsic factors (Table 1). The intrinsic factors are characteristics that are unique to the individual, while the environment or the context in which the physical activity takes place defines the extrinsic factors (Stearns et al., 2012). It is also acknowledged in previous scientific literature (Casa et al., 2014; Rae et al., 2008; Rav-Acha et al., 2004) that risk factors present themselves in a multifaceted manner. All of these factors listed in Table 1 contribute to (1) promote further increase in the body's

heat storage and metabolic heat production, and/or (2) prohibit the exercising individual from dissipating the body heat effectively, making the individual more vulnerable to succumbing to EHS.

Table 1.

Intrinsic and Extrinsic Factors for Exertional Heat Stroke (Adapted from Stearns, O'Connor, Casa, & Kenny, 2012)

Factors
Intrinsic Factors
Poor fitness level
Body composition (high fat mass, high BMI)
Lack of heat acclimatization
Dehydration
Predisposing medical conditions (malignant hyperthermia, febrile illness)
History of exertional heat stroke with sequelae
Medications (psychiatric medications, stimulants, anticholinergic medications)
Sleep deprivation
Over motivation
Lack of knowledge on exertional heat stroke
Extrinsic Factors
Equipment
Clothing
Environmental conditions (ambient temperature, relative humidity, WBGT)
Exercise intensity
Inadequate work-to-rest ratio

Intrinsic Factors.

The amount of metabolic heat produced by the exercising individual greatly relies on the intensity of exercise or activity (Saltin & Hermansen, 1966). Furthermore, the intensity of exercise or activity which the individual could tolerate can be affected by their fitness level, body composition, and heat acclimatization status (Wallace et al., 2006). In previous reports from the Marine Corps Recruit Depot and American football (Grundstein et al., 2012; Gardner & Kark, 2003; Kerr et al., 2013), individuals with higher body mass index had higher incidence of EHI and EHS fatalities. Metabolic heat production also increases with body weight, which further explains why there is a high prevalence of EHS fatalities in American football linemen (Grundstein et al., 2012; Kerr et al., 2013).

Oftentimes, especially in the military and competitive athletic settings, individuals may choose to ignore the early signs and symptoms of EHI (dehydration, light headedness, nausea, syncope, headache, dizziness) and refuse to make compensatory changes (reduce exercise intensity or terminate exercise) to reduce the risk of EHS. This "warrior mentality" or internal motivation to continue exercise further increases the risk of EHS in individuals whose fitness level is unmatched to the physical activity. Lack of heat acclimatization is also a contributing factor in determining the exercising intensity that individuals can endure during physical activity in the heat (Armstrong & Maresh, 1991).

The adaptations observed by optimizing the physical fitness level and heat acclimatization status of the individual complement each other in enhancing and dictating performance in the heat (Armstrong & Maresh, 1991; Lorenzo, Halliwill, Sawka, & Minson, 2010; Pryor et al., 2013).

Other physiological factors that should be noted are dehydration, predisposing medical conditions, and sleep deprivation. It has been reported that for every 1% of body mass lost during exercising in the heat, there is a 0.15-0.25˚C increase in body temperature (Huggins, Martschinske, Applegate, Armstrong, & Casa, 2014). Dehydration also compromises cardiac output, sweat sensitivity, and sweat rate, which all are crucial components of the thermoregulatory response during physical activity (Adams, Ferraro, Huggins, & Casa, 2014; Armstrong et al., 2007). Medical conditions, such as malignant hyperthermia, arteriosclerotic vascular disease, scleroderma, sickle cell trait, and febrile illness are likely to disturb the thermoregulatory response by contributing in further dehydration and rise in body temperature (Bergeron et al., 2005; Bouchama & Knochel, 2002; Casa, Armstrong, et al., 2012; Cleary, 2007; Gardner & Kark, 2003; Rae et al., 2008). Some medications are reported to have potential side effects that predispose individuals to EHS. These include: psychiatric, stimulant, and anticholinergic medications, and medications that affect the cardiovascular system (e.g., antihypertensive, diuretics) (Howe & Boden, 2007). If no precautions and prevention strategies are addressed, individuals with a previous history of EHI are also at risk of EHS (Cleary, 2007). Additionally, the cumulative fatigue from heat and sleep deprivation may add stress to the body that can play a role in increasing one's body temperature (Armstrong et al., 2007; Howe & Boden, 2007; Rav-Acha et al., 2004).

Extrinsic Factors.

***Environmental Conditions*.** Extreme environmental conditions, especially high ambient temperature and relative humidity, increase the risk of EHS by reducing the capacity to dissipate heat from the body (Coris, Ramirez, & Van Durme, 2004). The heat stress from the environment can be assessed by the ambient temperature, relative humidity, and radiation from the sun. The wet bulb globe temperature (WBGT) takes into consideration these three variables, and is used by many organizations as a measure to determine the degree of threat to health when exercising in the heat (Armstrong et al., 2007; Biery, Blivin, & Pyne, 2010; Casa et al., 2014). The resulting guidelines highlight the importance of modifying exercise intensity, work to rest ratios, hydration strategy, and type of exercise to be conducted. DeMartini, Casa et al. (2014) reported an increasing trend in medical tent admittance at the Falmouth Road Race (Falmouth, MA, United States) from EHI and EHS as ambient temperature and heat index (HI) increased. Unlike WBGT, HI is calculated with only two variables, ambient temperature and relative humidity. This 11.3km road race takes place in mid-August, with environmental conditions averaging ambient temperature 23.3±2.5˚C, relative humidity 70±16%, and HI 24±3.5˚C (DeMartini, Casa et al., 2014). The environmental conditions of the race, time of year, and a relatively shorter distance enabling runners to run at a higher intensity, create EHS-prone conditions for the runners.

***Equipment*.** In sports with protective equipment, such as American football and lacrosse, the barriers created by the equipment could hinder body heat loss from evaporation, conduction, convection, and radiation (Johnson et al., 2010). These barriers could also trap the heat between the skin and the equipment and clothing, creating a hot-humid microenvironment, which hinders the ability to dissipate heat. Likewise, attention should be given to the type of clothes worn during physical activity in the heat. Furthermore, early summer practices warrant special attention, since many people are not adequately heat acclimatized. There has been an alarming number of EHS deaths reported among American football players during the first 15 days of August (Grundstein et al., 2012), which demonstrates the vulnerability of athletes to the heat stress in early summer practices.

Recognition

Early and accurate recognition of EHS is crucial in preventing long-term complications and the possibility of death. The clinical diagnosis of EHS includes (1) body temperature greater than 40-40.5˚C and (2) central nervous system (CNS) dysfunction (Armstrong et al., 2007; Casa et al., 2014; Howe & Boden, 2007; Stearns et al., 2012). When body temperature is under 40˚C but the victim is presenting with CNS dysfunction, other conditions such as hyponatremia, hypoglycemia, and concussion should be considered and ruled out before EHS is considered. The signs and symptoms of EHS are summarized in Table 2. The victim may or may not present with loss of consciousness, thus the care provider should not solely rely on the level of consciousness to determine if the victim is suffering from EHS.

The most valid and accurate method of assessing body temperature in exercising individuals is via rectal temperature (Casa, Becker, et al., 2007; Ganio et al., 2009). Other means of assessing body temperature (e.g., oral, aural, temporal, axillary) are invalid when assessing exercise-induced hyperthermia (Ganio et al., 2009). While there are other valid means of measuring body temperature such as gastrointestinal and esophageal temperature, these measures are difficult to use in the field due to the difficulty in placing the esophageal temperature probe and cost of the gastrointestinal measures. In the athletics setting, rectal thermometry is considered the “gold-standard” for assessing body temperature and is the most practical option due to its validity, portability and accuracy. When rectal temperature assessment is not available, observe for physical signs and symptoms and rule out other conditions that may share similar signs and symptoms (e.g., exertional sickling, concussion, hyponatremia, hypoglycemia, heat exhaustion). Once the other conditions are ruled out, the victim should be suspected of and treated for EHS to mitigate a further rise in body temperature and potential sequelae.

Table 2.

Signs and Symptoms of Exertional Heat Stroke. Source: the authors.

Rectal temperature greater than 40-40.5˚C *	Dehydration
Central nervous dysfunction*	Vomiting
Irritability	Diarrhea
Confusion	Hypotension
Aggressiveness	Hyperventilation
Hysteria	Tachycardia
Loss of consciousness	Dizziness
Profuse sweating	Faint
Headache	Fatigue
Nausea	Weakness
	Decreased muscle coordination

**= Necessary for diagnosis of EHS*

Treatment

The goal of treatment for EHS is to reduce the athlete's body temperature to 39°C or lower within the first 30 minutes of collapse (Casa, McDermott, et al., 2007). Failure to lower body temperature below the critical 40.5°C threshold greatly increases the risk of mortality and morbidity due to multi-organ failure (Figure 1) (Casa, Kenny, & Taylor, 2010; Casa, McDermott, et al., 2007). The amount of time spent over the critical threshold temperature dictates the degree of organ damage and eventual patient outcome, further demonstrating the importance of immediate, aggressive, whole-body cooling upon diagnosis of EHS. The mantra "cool-first, transport second" should be used when treating an athlete with EHS because transporting the athlete to a nearby medical facility prolongs the time in which he/she is above the critical threshold body temperature (Casa, Armstrong et al., 2012).

Cold-water immersion (CWI) has been identified as the "gold standard" for the treatment of EHS due to its capability of quickly lowering body temperature (Casa, McDermott, et al., 2007). Cooling rates of 0.2°C•min^{-1} have been reported when using CWI as a cooling modality for treating EHS which results in a 1°C per 5-minute drop in body temperature (Casa, McDermott, et al., 2007). While every effort should be made to have appropriate equipment on-site at any facility where EHS could occur, if CWI is unavailable, other possible means of cooling that produce adequate cooling rates are rotating ice towels over the entire body every two minutes or continuous cold water dousing with ice massage (Costrini, 1990; McDermott et al., 2009).

When EHS is confirmed, protective equipment and excess clothing should be removed from the athlete and cooling should begin. For CWI, place the athlete up to his/her neck in a tub of ice water. If the athlete is too large or the tub is not large enough, place the athlete's trunk into the water and include as much of the extremities as possible. In the latter case, cover the exposed extremities and head with ice towels that are rotated every 2-3 minutes (Casa, McDermott, et al., 2007; McDermott et al., 2009). To allow for maximal cooling, the ideal temperature of the water should be between 1.7-

15°C and the water should be stirred continuously while the athlete is being cooled (Casa, McDermott, et al., 2007) (Table 3).

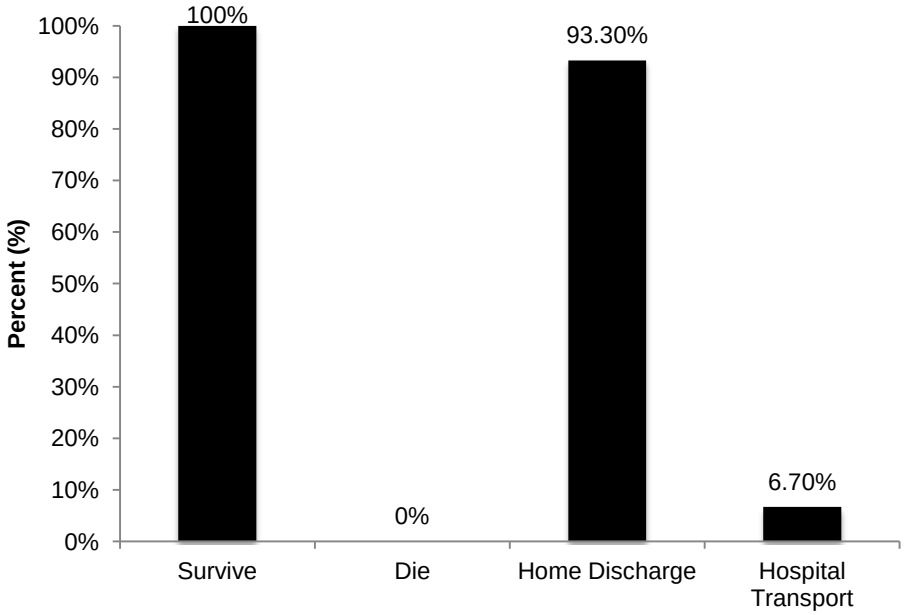

Figure 1. Outcomes of 274 EHS with immediate rectal temperature assessment, on-site treatment using cold water immersion, and on-site physician clearance at the Falmouth Road Race. In settings where physician clearance is not possible, follow-up at a hospital is recommended after treatment. Adapted from (DeMartini, Casa et al., 2014).

Rectal temperature should be monitored continuously throughout the duration of cooling and the athlete should be removed from the source of cooling once their rectal temperature reaches 39°C or less, to prevent the possibility of hypothermic afterdrop from occurring (Gagnon, Lemire, Casa, & Kenny, 2010). In the event a rectal thermometer is not available, the athlete should still be cooled before they are transported to a medical facility. When using CWI for cooling an athlete without a rectal thermometer, he/she should be cooled for 15 to 20 minutes, which with the cooling rates of CWI will be able to reduce the athlete's body temperature by 3-4°C, or until the patient starts to shiver.

Table 3.

Procedures for Cold-Water Immersion.

Procedures
Equipment - Immersion tub (e.g. stock tank) - Rectal thermometer - Ice - Water - A towel to harness the patient under the arms and hold the patient erect in the tub - Towels to cool the extremities outside of the tub with ice water
Preparation - Fill the tub with water and ice - Keep the water temperature between 1.7-15°C - Obtain rectal temperature - Remove excess clothes and equipment
Procedures (1) Immerse the patient up to the shoulders in the tub filled with water and ice (2) Keep the patient erect in the tub using a towel to maintain airway (3) Stir the water continuously in the tub (4) Monitor the vital signs and CNS status (5) Cease cooling when rectal temperature is under 39°C (6) Transport to medical facility for further evaluation

Source: the authors.

Prevention

While it may be impossible to limit all the factors that can contribute to EHS, there are a few modifiable factors that can drastically improve an athlete's ability to cope with exercise heat stress, consequently decreasing the likelihood of EHS. This includes heat acclimatization, exercise intensity, hydration status, extreme weather modifications, body cooling, and education (Armstrong et al., 2007; Casa et al., 2014). Efforts to address each of these areas have the potential to drastically reduce the risk for EHS.

Heat Acclimatization.

Heat acclimatization is a process that generally takes 10-14 days and induces many physiological changes that improve one's ability to exercise in the heat, including decreases in body temperature, increased skin blood flow, increased plasma volume, decreased heart rate, increased sweat rate, and decreased sweat sodium loss (Armstrong & Maresh, 1991). It has been recommended that heat acclimatization be implemented during the pre-season period for both high school (Casa et al., 2009) and college athletes (Casa, Anderson, et al., 2012). This has been highlighted in more recent years due to the number of athlete deaths that have occurred during the first few days when athletes return to activity (Bergeron et al., 2005; Casa et al., 2013; Grundstein et al., 2012; Kerr et al., 2013). Recent evidence has suggested that short term heat

exposure (5-7 days) is also effective in inducing heat acclimatization (Chen, Tsai, Lin, Lee, & Liang, 2013; Garrett, Rehrer, & Patterson, 2011). However, not all physiological adaptations involved with heat acclimatization occur during this shortened period (Chalmers, Esterman, Eston, Bowering, & Norton, 2014). In order to elicit all of the physiological benefits involved with heat acclimatization, 10-14 days of heat exposure are required (Armstrong & Maresh, 1991). It is recommended that all athletes follow a heat acclimatization program at the start of all pre-season or return to activity periods (Casa et al., 2013; Casa, Anderson, et al., 2012; Casa, Guskiewicz, et al., 2012).

Exercise Intensity.

Exercise intensity can increase body temperature dramatically (Rav-Acha et al., 2004) and can also be affected by fitness status. A lower fitness status means the athlete will have to work harder to keep up with their other teammates, creating greater metabolic heat. When working with a large team it is important to account for differences in individual fitness status by allowing varying times for recovery and modifying overall workout demands. Exercise intensity should also be adjusted during new exercise regimes and early in the season (Casa, Anderson, et al., 2012).

Hydration Status.

Dehydration has been well documented to negatively effect exercising body temperature (increases 0.15-0.25°C for every 1% dehydration), notably at levels greater than 2% dehydration (Casa, Clarkson, & Roberts, 2005; Casa, Stearns, et al., 2010; Cheuvront, Montain, & Sawka, 2007; Distefano et al., 2013; Huggins et al., 2014; Judelson et al., 2007; Montain & Coyle, 1992; Stearns et al., 2009). Beyond 2%, the negative effects of dehydration include: increased body temperature, increased heart rate, and decreased performance (Adams et al., 2014; Casa, Clarkson, et al., 2005; Casa, Stearns, et al., 2010; Cheuvront et al., 2007; Distefano et al., 2013; Huggins et al., 2014; Judelson et al., 2007; Montain & Coyle, 1992; Stearns et al., 2009). Avoiding dramatic levels of dehydration not only protects athletes, but also will help improve performance. One of the easiest methods to monitor and avoid dehydration is to educate athletes to monitor their urine color and utilize a body weight system where athletes are weighed in and out of practice to estimate the fluids lost within that session.

Extreme Weather Modifications.

Extreme or new weather demands on athletes also pose a great risk for heat illness. In recent years it has been noted that the majority of heat-related American football deaths occur in August in North America (Grundstein et al., 2012; Kerr et al., 2013). These coincide not only with the start of the youth and collegiate level athletics, but also the hottest time of the year in that region. Given the extra stress the environment can pose, especially when athletes are not acclimatized to it, many organizations have published work to rest (or exercise to rest) ratios which vary according to the environmental conditions (Armstrong et al., 2007; Binkley, Beckett, Casa, Kleiner, & Plummer, 2002; Casa, Anderson, et al., 2012). It is important to establish and enforce exercise modification guidelines based on extreme weather conditions. These policies should also encourage the use of the WBGT monitoring, as

this is the only measure that takes into account radiant heat in addition to the ambient air temperature and humidity.

Body Cooling.

Body cooling is another strategy that can be used as a preventative measure to minimize the risk of EHI and EHS. In general, cooling before or between bouts of exercise can attenuate the rise in body temperature while also improving performance (Arngrïmsson, Petitt, Stueck, Jorgensen, & Cureton, 2004; DeMartini et al., 2011; Siegel et al., 2010; Siegel, Maté, Watson, Nosaka, & Laursen, 2012; Yeargin et al., 2006). In most sports, athletes are able to take a multi-faceted approach to body cooling; they are able to cool prior to the start of practice or competition as well as during breaks, halftimes, and substitutions from play. For the equipment-laden athlete, such as in American football, it is not feasible to utilize most forms of aggressive cooling such as CWI during competition due to the inability to easily remove protective equipment. In cases such as this, forearm immersion and rotating ice towels over extremities has demonstrated moderate cooling effects to help attenuate the rise in body temperature during competition (DeMartini et al., 2011).

Education.

In any scenario, it is important for those individuals in charge of the health and well being of the athletes to take reasonable measures to obtain a basic understanding and knowledge of heat illnesses. By doing so, they can take the appropriate steps to prevent and provide initial treatment for heat illness if necessary. It is recommended that coaches take a basic course covering the top causes of death in athletes (above and beyond first aid and CPR courses) and what they can do to help prevent it (Casa, Anderson, et al., 2012). It is always recommended that a medical professional be present when athletes are practicing or competing in organized sport; it is important, however, that coaches are able to intervene on behalf of the athlete if necessary (Casa et al., 2013; Casa, Anderson, et al., 2012; Drezner, 2012).

Return to Activity

Return to exercise or activity following EHS will most heavily rely upon the severity of the case and how quickly and effectively that individual was provided treatment (McDermott et al., 2007; O'Connor et al., 2010). It is not uncommon for liver biomarkers to be elevated the days following the event, however, with appropriate treatment these should return to normal values rapidly, usually within a week (Roberts, 2000; Roberts, 2006). In cases where appropriate treatment was not implemented, elevated levels of these biomarkers may be an indicator of possible organ failure that could precipitate death (Leon & Helwig, 2010; O'Connor et al., 2010; Wallace, Kriebel, Punnett, Wegman, & Amoroso, 2007). Because every case of EHS is different, recovery times can vary drastically. Due to this consideration, there is no universal guideline that can be applied to every case. Nevertheless, the American College of Sports Medicine recommends a 5-stage return to training and competition progression (Armstrong et al., 2007):

a. "Refrain from exercise for at least 7 d following release from medical care; b. Follow up about 1 week post incident for physical examination, and lab testing or diagnostic imaging of affected organs based upon the clinical course of the heat stroke incident;

c. When cleared for return-to-activity, begin exercise in a cool environment and gradually increase the duration, intensity, and heat exposure over 2 weeks to demonstrate heat tolerance and to initiate acclimatization; d. If return to vigorous activity is not accomplished over 4 weeks, consider a laboratory exercise-heat tolerance test; e. Clear the athlete for full competition if heat tolerant after 2 to 4 weeks of full training.'' (Armstrong et al., 2007, p. 562).

While the applicability of heat tolerance testing has come under some scrutiny (Kazman et al., 2013), it can provide an initial indication of an athlete's ability to exercise at a low intensity in the heat. It is not currently possible to quantify or determine with diagnostic tests if an athlete has fully recovered from EHS. Current US Army recommendations utilize a recovery approach based on two classifications: EHS with sequelae and EHS without sequelae, with the latter following a much more aggressive return to activity timeline (O'Connor et al., 2007, 2010). Cases that present with sequelae may require months to years for full recovery.

No matter the severity of the EHS case, it is important to determine the predisposing factors present in order to educate the athlete and limit the potential for future heat illness. It is also imperative that any return to activity following an EHS incident is under the guidance or supervision of a physician, preferably one familiar with heat stroke. Not all cases will be the same and it is important to closely monitor an athlete's progression and return to participation. If there is any indication that the athlete is not able to tolerate the exercise progression, the process should be stalled and progressed back to the previous stage until the athlete is able to move forward without any complications.

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