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FEATURE ARTICLE

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Exertional Heat Illness: A Clinical Overview

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ABSTRACT: The human body has an array of physiologic mechanisms available to withstand the effects of environmental heat stress and humidity. Despite this, athletes routinely succumb to the effects of exertional heat illness (EHI). The incidence of EHI is highest during August and directly correlates with increases in ambient temperature and humidity. Prevention of EHI begins by assessing the internal and external factors that influence thermoregulation and heat dissipation. Exertional heat stroke is the most severe form of EHI and is one of the leading causes of preventable death in athletes. Risks of EHI are decreased when EHI education is

provided, emergency action plans are developed and rehearsed, and activity modifications are based on regionally specific environmental guidelines.

KEYWORDS: Exertional heat illness, exertional heat stroke, thermoregulation, sports medicine

Exertional heat illness (EHI) is a spectrum of pathologic states initiated by a collection of intrinsic and extrinsic risk factors that result in physiologic thermal injury. The term *EHI* encompasses multiple transient diseases, and while the presence of one disease may predispose or raise concern for another within the spectrum, they do not reliably occur sequentially.[1]

Our understanding of EHI continues to expand in regard to pathophysiology, prevention, and awareness. However, the incidence of EHI continues to inflict significant burden on the athletic community as exertional heat stroke (EHS) is one of the top 3 causes of death in athletes.2 Understanding each form of EHI is pivotal for patient safety, and clinicians must learn to recognize and appropriately manage each type of EHI in a timely manner.

Multiple studies evaluating EHI incidence among youth and collegiate American football players demonstrate the necessity for refinement of and adherence to climate-adaptive guidelines for EHI prevention.[2,3] While the treatment of EHI is subtype-specific, core strategies of management include appropriate prevention, rehydration, and monitored cooling. As the medical understanding of EHI pathophysiology evolves, the current return-to-play recommendations may need to be revised, because more formal and quantitative monitoring may pave the way for safer athlete recovery.

DEFINITION

The term *exertional heat illness* as used in this article encompasses multiple disease states that require delineation from one another. The clinical presentation of these separate disease states may overlap. Definitions of each type of EHI are described in **Table 1**.

Table 1. Definitions of EHI Types

Disease State	Definition	Common Signs/Symptoms

Exercise-associated muscle cramps	Involuntary, transient, and often painful muscle contractions, localized or generalized, that occur in the setting of rigorous, prolonged physical activity.[4]	Pain, muscle cramping, muscle twitching/tingling, residual soreness[4]
Heat rash (also known as miliaria or prickly heat)	A maculopapular, erythematous, and punctate rash caused by sweat duct plugging.[5]	Erythema, pruritis[6]
Heat edema	Dependent edema due to prolonged peripheral venous dilation.[5]	Lower extremity swelling, fullness/discomfort[6]
Exercise-associated collapse (also known as heat syncope)	Abrupt and involuntary discontinuation of physical activity secondary to transient, non-sustained decrease of central blood volume causing functional orthostasis.[7,8]	Nausea, tachycardia, dizziness/lightheadedness, inability to stand, syncope/presyncope, paleness/perspiration[7,8]
Heat exhaustion	Inability to continue exercise secondary to cardiac insufficiency and energy depletion in the setting of elevated heat exposure.[4,9]	Nausea/vomiting, dizziness/imbalance, confusion/disorientation, hypotension, oliguria[3]
Exertional heat stroke	A state of over-accumulated heat stress with thermoregulatory dysfunction marked by core body temperature ≥40.5°C (≥105°F) and alterations in mental status with possibility of end organ damage, hypercoagulability, and death.[3]	Nausea/vomiting, confusion/disorientation, ataxia, lethargy, coma, disseminated intravascular coagulation, liver/kidney Injury[3]

EPIDEMIOLOGY AND RISK FACTORS

When evaluating the risks associated with EHI occurrence, clinicians must identify both the intrinsic and extrinsic risk factors that elevate the athlete's risk. Acknowledging predictable risk factors is crucial, since mitigation of these factors reduces the risk of avoidable injury. When recreational athletes were surveyed randomly regarding their risk of EHI, nearly 20% were at risk for EHI and were unaware. In addition, they did not know how to make appropriate modifications to address these risk factors.[8]

EXTRINSIC RISK FACTORS

At its extremes, local weather can predispose outdoor athletes to an imbalance of heat production and dissipation. Analysis of weather conditions provides quantifiable data that helps stratify the degree of an athlete's risk of developing EHI. Wet-bulb globe temperature (WBGT) has been established as the most reliable assessment to estimate the risk of developing heat-related illness.[10] The WBGT is a sum of weighted meteorological variables, consisting of ambient temperature, radiant heat, relative humidity, and wind speed.[11] Collectively, these contribute to an athlete's heat stress, and the WBGT is used to provide a calculated risk of EHI.[7] The heat index takes into account only temperature and relative humidity and does not consider radiant heat or wind speed, making WBGT a superior measurement for estimating risk.[10]

Although many sports are played in environmental settings that predispose participants to EHI, American football is associated with the highest incidence.[12,13] August is the month with the highest rate of EHI, which correlates with August having the highest average WBGT.[14] When observing high school football athletes specifically, heat cramps, heat exhaustion, and heat syncope are the most common EHIs, occurring at rates of approximately 70%, 23%, and 8%, respectively.[14,15] Certain protective equipment such as helmets, shoulder pads, and goalkeeper pants create an insulation effect that alters heat dissipation, which is critical when ambient conditions are hazardous.[1,16]

Metabolic heat production is a function of the intensity of physical exertion, and medical personnel should be aware that the higher the work-to-rest ratio, the higher the risk of EHI.[2] Physiologic strain on the athlete has been suggested to be higher during intermittent exercise with variable workload and recovery periods than during continuous exercise. This is seen in American football, tennis, and hockey, where the interval of play is of high intensity, and the rest time is highly irregular.[13] The work-to-rest ratio considers not only the brief intermissions during the session, but also rest between organized practice or play. Playing multiple matches per day, extending practice hours, or holding consecutive workouts add to work-to-rest imbalance and increase the risk of EHI. For instance, an increased incidence of EHI in football players has been demonstrated within the first 3 days of beginning twice-daily practices.[17]

been demonstrated within the first 3 days of beginning twice-daily practices.

An athlete's medications are another potential extrinsic risk factor, and clinicians must be familiar with the potential effects that medications may have on performance. Caution should be used with any medication that affects thermoregulation, hydration status, or the athlete's cardiovascular physiology.[9,13,18] High-risk medications include stimulants, anticholinergics, alcohol, α-adrenergics, benzodiazepines, β-blockers, calcium-channel blockers, neuroleptics, tricyclic antidepressants, and diuretics.[9,13,18] In addition, internal, social, peer, or competitive pressures may drive athletes to perform beyond their physiologic limit. For example, the known presence of a college scout may compel an athlete to ignore physiologic cues to rest or rehydrate, increasing EHI risk.[1] Poor education about EHI and lack of an emergency action plan (EAP) are risk factors, potentially causing delayed recognition and treatment of symptoms.[1]

INTRINSIC RISK FACTORS

Intrinsic risk factors should be approached as thermoregulatory disadvantages that cannot be immediately modified for play. While a vast spectrum of diseases increases an athlete's risk for EHI, the most commonly encountered are discussed here.

Obesity has been established as a risk factor for EHI. Military recruits and football players with a higher body mass index (BMI) have been shown to have higher rates of EHI.[10,13,19] The lower surface area to body mass ratio is the most contributing factor.[1] Interestingly, the risk associated with increased BMI includes both muscular and adipose tissue components, since increased muscular tissue causes higher metabolic heat production, and insulating adipose tissue dampens heat dissipation.[1,16] In addition, larger athletes typically exhibit higher sweat rates and exhibit greater water loss as a result.[12] The perfect storm of these risk factors is commonly seen in American football, given that 86% of the heat-related deaths between 1980 and 2009 were offensive or defensive linemen, who are generally larger than other position players.[10]

Both acute and chronic comorbidities can present challenges to safe athletic play. The sports medicine team should be familiar with players who have diseases that predispose them to developing EHI. Similar to medications, any comorbid condition that disrupts hydration homeostasis, electrolyte balance, thermoregulation, cardiovascular response to heat strain, or metabolic output increases risk of developing EHI. Transient conditions include sunburn, acute gastroenteritis, alcohol use, recent febrile illness, sleep deprivation, or recent concussion.[1,7,13,16] Chronic conditions include eating disorders, diabetes mellitus, sickle cell trait, thyroid disease, cognitive dysfunction, cystic fibrosis, chronic skin disorders, and heart disease.[7,9,12,13,16,18] In addition, past occurrence of EHI is considered a major predictor of future occurrence of EHI.[8]

While EHI occurs at all levels of competition, much attention focuses on youth sports, since more than 6,500 high school athletes are treated annually for some form of EHI.[3] Children typically are less likely to recognize their own symptoms leading to EHI, have a lower surface

typically are less likely to recognize their own symptoms leading to EHI, have a lower surface area-to-mass ratio, and exhibit slower sweat rates than adult athletes.[18] However, children demonstrate similar abilities to acclimatize and thermoregulate as their adult counterparts and are able to sustain equal exercise intensities and workloads.[20] In contrast, the elderly population is considered to be at higher risk for EHI due to decreased efficacy of heat dissipation and decreased cardiovascular response.[18]

THERMOREGULATION

Humans are exposed to 4 primary methods of heat exchange. Convection is the process of a moving substrate absorbing heat from a nearby heat source and is observed with cooling fans or wind.[7-8] Factors such as air speed or temperature gradient affect the quality of convectional cooling.[18] Conduction is the direct transfer of heat energy between adjacent surfaces and is observed with the application of ice directly to the skin.[7,18] Radiation is the exposure to infrared wavelengths of heat, which can impose heat stress even in cooler ambient temperatures.[9,18] Evaporation, which humans utilize with perspiration, is the primary cooling mechanism when ambient temperatures are above 20°C (68°F).[9] Convection, conduction, and evaporation are all dependent on gradients, and if the vapor pressure or ambient temperature are unfavorable, athletes can develop a heat acquisition-dissipation imbalance.[7,12]

Heat acclimatization is the process of enhancing human physiology to tolerate elevated heat stress.[7] Heat stress is defined as the collective environmental and host conditions that elevate core body temperature (CBT).[7] Through gradual exposure to increased heat stress and increased work-to-rest ratios, humans develop improved mechanisms of thermoregulation. Such adaptations include increases in plasma volume, sweat rate, cutaneous vasodilation, and aldosterone production.[6,7,13] Molecular studies suggest an increase in heat-shock proteins, which exhibit neuroprotective and vasoprotective qualities in the setting of increased CBT.[16] These enhancements lead to decreased urinary sodium excretion, decreased sweat sodium content, and improved perfusion during athletic performance.

Acclimatization takes place over a 7- to 14-day period of increasing intensity and duration of heat stress, which can be modified by increasing workload, reducing break time between exercise intervals, or increasing exposure time to environmental heat. [1,7,8,17,18] During the first 5 to 7 days of acclimatization, athletes exhibit lower exercise heart rates, larger stroke volumes, earlier onset of sweating, higher sweat rates, and earlier increases in skin blood flow.[8] Collectively, these adaptations not only improve athletic performance, but also decrease subjective heat strain on the athlete—nonacclimatized competitors report more strain than their acclimatized counterparts.[8] These enhancements are not sustained without continued exposure to heat stress; an estimated 75% of acclimatized physiology is lost after 3 weeks of rest.[8] While the National Athletic Trainers Association (NATA) endorses specific acclimatization guidelines for football, universal recommendations applicable to other sports are lacking.[21]

PATHOPHYSIOLOGY AND PRESENTATION

Most EHI subtypes are caused by similar pathophysiologies, and occurrence of one subtype may indicate susceptibility to another.[19] Heat rash—also known as prickly heat, miliaria, or lichen planus tropicus—is a localized inflammatory reaction within sweat ducts caused by plugging.[6] As this process worsens, dermatitis and pruritis may develop.[6] Heat edema is caused by prolonged vasodilation in the setting of heat exposure.[6] A microvascular transudate pools in the extracellular space, often in the lower extremities.[6]

Exercise-associated muscle cramps (EAMCs) develop due to a combination of muscle overload, neuromuscular fatigue, and electrolyte deficiency.[11] Proposed mechanisms suggest that sodium loss and hypotonic fluid replacement create an extracellular fluid contraction, which causes nerve terminal deformation and resulting muscle "hyperexcitability."[6] Athletes exhibit involuntary and often painful muscle cramps in the overworked musculature, but generalized cramping may occur as electrolyte and hydration status worsen.[1,4] The role of dehydration and electrolyte status has been questioned, given that athletes with deficits in hydration or sodium comparable to cramping athletes may not exhibit EAMC.[1]

Exercise-associated collapse (EAC), also known as heat syncope, occurs when an exercising athlete suddenly collapses secondary to decreased cerebral perfusion.[7,8] Low central blood volume is the key dysfunction and is classically seen in the distance runner crossing the finish line who immediately discontinues motion to rest.[1] The loss of pump function from lower extremity musculature combined with decreased peripheral vascular resistance causes venous pooling and decreases venous return. Combined with decreased volume reserve, this results in EAC.[1] Preceding symptoms include lightheadedness, faintness, dizziness, and nausea with possible progression to syncope. EAC may occur within the first week of practices, during which athletes have not yet exhibited volume expansion with acclimatization.[1] Other more emergent conditions must be ruled out first, since EAC is a diagnosis of exclusion.

Heat exhaustion is described as the abrupt inability for an athlete to continue performance secondary to heat stress.[6] A threshold of dehydration is breached, resulting in cardiovascular insufficiency manifested by tachycardia, hypotension, incoordination, and oliguria.[4,9] Dizziness, syncope, nausea, vomiting, confusion, and profound fatigue are common symptoms.[1,4] Although the discontinuation is often unexpected, substantial dehydration may be present before a sensation of thirst is appreciated or the athlete is permitted to resupply.[8] Core body temperature is elevated but not greater than 40°C (104°F), and significant mental status changes should not be present.[4]

EHS is defined as a CBT obtained rectally of greater than 40°C (104°F) accompanied by alterations in mental status.[6,9,13] A severe imbalance of heat gain over heat dissipation initiates loss of automated thermoregulation.[4,18] A "multi-hit hypothesis" suggests an accumulation of

loss of automated thermoregulation.[1,6] A "multi-hit hypothesis" suggests an accumulation of metabolic and circulatory failures resulting in potentially fatal end-organ damage.[1,16,22] Thermoregulatory dysfunction is suggested to begin when peripheral blood flow is diminished due to dehydration and compensatory preservation of central blood volume, minimizing heat loss.[16] Critically elevated CBT denatures vital cell proteins, triggering losses of both cellular metabolic and barrier function.[6,22] It is additionally suggested that reduced visceral blood flow causes localized intestinal wall ischemia. Combined, these pathophysiologies result in massive oxidative stress and widespread release of endotoxins and pyogenic cytokines.[16] A state of acidosis and a systemic inflammatory response occurs, which may lead to acute respiratory distress syndrome, disseminated intravascular coagulation, and/or significant kidney, liver, and muscular damage.[1,6,10,16] Athletes may exhibit neurological symptoms such as headache, confusion, disorientation, ataxia, lethargy, irritability, and potential coma.[1] Clinicians should be aware that patients may continue to perspire despite having EHS.[8,18,23]

TREATMENT

The principles of treatment of most types of EHI involve appropriate strategies for cooling, rehydration, and maximizing circulation based on illness severity. During treatment, clinicians should monitor for overlap of EHI conditions, since EHI subtypes may exhibit overlapping signs or symptoms. It is critical to note that treatment of any form of EHI begins with rapid symptom recognition, which can be identified by clinicians, coaches, and teammates.[13,23]

Heat edema is treated with use of compression stockings during and after exercise.[6] Heat rash is typically treated symptomatically.[6] EAMCs are routinely treated with rest, passive stretching, hydration, and electrolyte replenishment.[1,6,9] Oral hydration is strongly recommended. Intravenous rehydration should only occur when oral rehydration is unavailable, typically as a result of significant nausea and vomiting, or when rapid rehydration is required. Fluid absorption, retention, and distribution are improved with administration of sodium- and carbohydrate-containing beverages, and a rate of 1 to 2 liters per hour of oral hydration has been suggested as optimal.[1,8]

Heat exhaustion and EAC have similar recommended interventions. During initial assessment, clinicians should maintain control of circulation, airway, and breathing in an athlete that abruptly decompensates during or immediately following vigorous activity.[1] Medical personnel should consider alternative causes including hyponatremia, hypoglycemia, cardiac or pulmonary etiologies, anaphylaxis, or trauma.[8] While additional possibilities are being ruled out, athletes should be transported to a shaded area with use of cooling fans if available.[18] Clothing should be removed to provide greater evaporative surface area.[1] Patients should be laid supine, and vital signs should be collected with legs elevated above heart to maximize central circulation.[1,6,18] Oral hydration, if tolerated, is recommended in the absence of vomiting or diarrhea.[1] Close monitoring of the athlete is imperative, and lack of rapid recovery or

deteriorating mental status with these conservative measures should warrant further investigation. Obtaining a CBT rectally is mandatory to evaluate for the possibility of EHS.[1] If resources or surrounding circumstances do not permit assessment via rectal temperature, a high suspicion of EHS alone should trigger use of the most effective rapid cooling available.[23]

The key to treatment of EHS is to begin reversing the heat gradient as quickly as possible. Cold-water immersion (CWI) is the most effective method of cooling and has shown reliable safety and efficacy in the treatment of EHS.[18,22] Often referred to as the "golden 30 minutes," providers should aim to lower CBT to a target of less than 38.9°C (102°F) within 30 minutes of recognition, since multiple studies report a 100% survival rate when this goal is achieved.[1,9] Monitoring of rectal temperature is recommended during the cooling process, with rechecks every 5 to 10 minutes if a rectal thermistor is not available.[8,13] CWI up to the neck is preferred as equipment permits. An alternative method known as tarp-assisted cooling has shown similar efficacy to CWI in rapid cooling.[24] Secondary cooling strategies should be implemented on nonsubmerged extremities, prioritizing submersion of the torso. Ice pack placement in the axillae, groin, and neck regions should be combined with wet-towel exchange and cool fanning on distal extremities, since these practices have shown efficacy in lowering CBT but are not considered equivalent to CWI or tarp-assisted cooling.[1]

Guided by a principle of "cool first, transport second," clinicians should aim to lower CBT below 38.9°C (102°F) before initiating transport to emergency facility.[1,13] If no medical personnel are present, coaching/supervising staff should begin the most rapid cooling technique readily available and call for emergency transport immediately, prioritizing circulation, airway, and breathing before and during transport.[1] Patients may have obstacles to cooling such as diarrhea, vomiting, or combativeness. Basic laboratory assessment at a minimum should be collected, including evaluation of electrolytes, liver and kidney function, and muscle enzymes; higher-acuity resuscitative measures and specialist consultation is recommended if clinically indicated.[25]

RETURN TO PLAY

Heat edema, heat rash, and EAMCs rarely hinder athletic performance upon resolution, and athletes may resume play as tolerated. For heat exhaustion and EAC, same-day return to play is not recommended, although most cases do not require follow-up or subsequent restriction from play.[1,8] The American College of Sports Medicine (ACSM) guidelines recommend cessation from any exercise for at least 7 to 14 days after EHS.[25] After physician assessment and normal laboratory evaluation results, gradual return to play with incremental heat exposure and intensity of exercise over 2 to 4 weeks will determine readiness for unrestricted performance.[1,16] Athletes who are unable to demonstrate heat tolerance within 4 weeks of the initial event should be referred for heat tolerance testing.[16,25]

PREVENTION

Effective prevention begins with proper education about EHI. Supervising personnel should be aware of the signs and symptoms of the EHI subtypes, and all staff should be well-versed in executing a structured EAP.[1] This includes written and well-practiced protocols for EHI treatment, designation of duties, and an accessible supply of equipment to be used for safe treatment (eg, ice tubs, rectal thermometer/thermistor, water source).[1,4] Athletes should be taught to recognize basic symptoms in themselves and teammates such as fatigue, dry mouth, increased thirst, and decreased urination or darkened urine.[7,9,18] Beginning athletic play in a state of normal hydration is important, since CBT and perceived strain during performance are increased when hydration is not optimized.[1,4,14] **Table 2** provides an abbreviated list of these key proficiencies.

Table 2. Basic EHI Prevention Strategies for At-Risk Athletes

Practice	Competency
Staff education	Recognize early signs and symptoms of EHS
	Implement emergency action plan for EHI
	Execute emergency action plan proficiently
	Permit routine breaks for hydration during practice/competition
	Permit ample time between practices/competition for rehydration
Athlete education	Modification of practice/competition given local climate conditions
	Withhold athletes from play who demonstrate safety risk
Hydration status	Recognize signs/symptoms of early EHS in self and teammates/opponents/coaches/game officials
	Practice adequate hydration before/during/after athletic play
	Demonstrate basic understanding of emergency action plan
Acclimatization	Rest/recover appropriately on breaks/off days
	Practice pre-performance hydration routinely

	Respond to physiologic cues of dehydration (thirst, dry mouth, dark urine, dizziness, lightheadedness)
	Avoid behaviors detrimental to optimum hydration
	Appropriately replenish volume losses after exercise
	Assess athlete's current acclimatization status prior to first practice
	Adhere to current National Athletic Trainers' Association guidelines for acclimatization for high school football
	Continue investigating universal acclimatization guidelines appropriate for any at-risk sport
	Continue investigating safer exercise strategies that provide equal acclimatization benefits without exposure to high heat stress

The ACSM currently endorses activity modification guidelines that dictate exercise restrictions based on local WBGT. Although these guidelines are universal, the crucial shortcoming is a lack of regional-specific considerations of temperature extremes.[2] Current ACSM guidelines use absolute WBGT to dictate activity restriction, while regional guidelines would better account for local extreme WBGT (**Table 3**). For example, a typical summer-day WBGT in South Florida is up to 31°C (87°F) compared with 25°C (77°F) in Massachusetts.[2] Using regional guidelines, this places the typical Florida temperature in the 98th percentile of temperatures in Massachusetts and would call for significant differences in play restrictions compared with the fixed WBGT used in the ACSM guidelines. Studies comparing prior EHS incidents have deduced that using region-based policies would have enforced safer restrictions compared with the universal ACSM guidelines, especially when evaluating incidents that took place in the northern regions of the United States.[2]

Table 3. Comparison of ACSM Activity Guidelines versus proposed Regional Specific Guidelines by

ACSM Activity Modification Guidelines[4]	**Regional Specific Activity Guidelines[9]**

<82.0°F (27.8°C)—Normal activities; provide separate rest breaks each hour of minimum duration of 3 minutes each during workout.	<82.0°F (27.8°C)—Normal activities; provide at least 3 separate rest breaks each hour of minimum duration of 3 minutes each during workout.
82.0-86.9°F (27.8-30.5°C)—Use discretion for intense or prolonged exercise; watch at-risk players carefully; provide separate rest breaks each hour of minimum duration of 4 minutes each.	82.0-86.9°F (27.8-30.5°C)—Use discretion for intense or prolonged exercise; watch at-risk players carefully; provide at least 3 separate rest breaks each hour of minimum duration of 4 minutes each.
87.0-89.9°F (30.6-32.2°C)—Maximum length of practice = 2 h. For football, players restricted to helmet, shoulder pads, and shorts during practice; all protective equipment must be removed for conditioning activities. For all sports, provide separate rest breaks each hour of minimum duration of 4 minutes each.	87.0-89.9°F (30.6-32.2°C)—Maximum length of practice is 2 hours. For football, players restricted to helmet, shoulder pads, and shorts during practice; all protective equipment must be removed for conditioning activities. For all sports, provide at least 4 separate rest breaks each hour of minimum duration of 4 minutes each.
90.0-92.0°F (32.2-33.3°C)—Maximum length of practice = 1 h. No protective equipment may be worn during practice, and there may be no conditioning activities. There must be 20 minutes of rest breaks provided during the hour of practice.	90.0-92.0°F (32.2-33.3°C)—Maximum length of practice is 1 hour. No protective equipment may be worn during practice, and there may be no conditioning activities. There must be 20 minutes of rest breaks provided during the hour of practice.
>92.1°F (33.4°C)—No outdoor workouts, cancel exercise, and delay practices until a cooler WBGT reading occurs.	>92.1°F (33.4°C)—No outdoor workouts, cancel exercise, and delay practice until a cooler WBGT reading occurs.

CONCLUSION

Heat safety should be a priority with any sporting event. EHI is a risk posed to any athlete, but the risk is elevated in the presence of intrinsic and extrinsic risk factors. Acclimatization guidelines that are universally applicable to every sport are still lacking, and further investigation is warranted. Early recognition of EHI is key to limit injury to the athlete and to mitigate progression of the disease into a more severe form. EHS is the most dangerous form of EHI, and the most effective treatment studied for EHS is CWI. Injury prevention from EHI stems from preparedness and planning; EAPs, activity-modification guidelines, and EHI education are

preparedness and planning. EAP, activity modification guidelines, and EAP education are

pillars of heat safety. Regional heat-safety policies may provide safer activity restriction than current models and must be further implemented.

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