

Heat Illness – A Practical Primer

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INTRODUCTION

In the United States, heat injury results in approximately 650 preventable deaths annually. From 1999–2009, over 7,000 heat-related deaths were reported with 72% directly due to heat exposure and the remaining 28% due to heat-induced exacerbations of underlying medical conditions. Also, annually, an estimated 9,000 high school athletes are treated for exertional heat illness and this number is increasing.¹

Heat illnesses encompass the spectrum of disease from heat rash, heat cramps, heat exhaustion, to heat stroke. With proper precautions, heat-related deaths are entirely preventable. While typically believed to be a problem of those in warmer environs, the highest recorded incidence of exertional heat stroke occurs at the Falmouth Road Race in Massachusetts with a reported rate of 1–2 events per 1,000 entrants. However, proper planning and vigilance have resulted in no deaths.^{2,3} This review examines the physiology, risk factors, diagnosis, treatment, and prevention of heat illness.

HOW DOES THE BODY NORMALLY KEEP COOL?

Cooling is a complex interplay of conduction, convection, radiation, and evaporation. Conduction is the direct transfer of heat across a temperature gradient through physical contact, for example, placing ice packs in the axilla and groin. Convection is similar except heat is lost through the movement of liquids or gas, such as warm core blood carried to the skin's surface or fanning the body. Radiation is a form of heat loss through infrared rays without physical contact, as occurs when the sun transfers heat to the earth. Conduction, convection and radiation rely on a heat gradient so in warmer climates, the degree of heat transfer is not significant. Evaporative heat loss occurs when heat is lost through water vapor pressure, such as evaporating sweat. Evaporation is responsible for 80-90% of the body's heat dissipation during exercise in the heat and is the most effective involuntary heat loss mechanism.⁴

An increase in body temperature, sensed by the hypothalamus, results in cutaneous vasodilation and a reduction in the basal metabolic rate, accomplished by alterations in subcutaneous blood flow, sweat production, skeletal muscle tone, and overall metabolic activity. If this response is inadequate to manage the heat stress, cholinergic sympathetic fibers stimulate sweat production. Sweat needs to evaporate (not be wiped off) in order to contribute to cooling. The

conversion of 1.7 ml of sweat to the gaseous phase consumes 1 kcal of heat.

Athletes typically lose about 1 liter of water per hour (via sweat) but during high-intensity exercise or in well-trained, acclimated athletes, this can increase to up to 2–2.5 liters of water per hour.⁵ The rate of sweat production is dependent on environmental conditions, intensity of exercise, fitness level, and acclimation of the individual, while the rate of sweat evaporation relies on ambient temperature, humidity, and the area of skin exposed to air.

RISK FACTORS

The elderly (>65), children (<18), outdoor workers such as firefighters and construction workers, athletes, and those taking certain medications, are among those at risk for developing heat illness. During July and August, the risk of heat illness rises dramatically. This is due to increased temperature and humidity and a slowing of the evaporative process, increased ambient humidity, overall dehydration and in athletes lack of heat acclimation, deconditioning, and wearing athletic equipment.^{6,7}

MEASURING HEAT STRESS

Heat stress conditions are measured by the wet-bulb globe temperature (WBGT), which takes into account the dry-bulb (ambient) temperature, wet-bulb temperature (humidity), and globe temperature (radiant heat). Environmental conditions are quantified using the equation $WBGT = 0.7T_{wb} + 0.2T_g + 0.1T_a$ (T_{wb} is the natural wet-bulb temperature, T_g is the globe thermometer temperature, and T_a is the dry-bulb temperature).

Ideally, the WBGT should inform activity modification; however, measurement requires specialized equipment that is not frequently available. Heat index is used as a substitute, and while this takes into consideration temperature and humidity, it excludes the contribution of radiation and so reflects heat stress in shady areas.

HEAT CRAMPS

Heat cramps are painful involuntary muscle spasms seen in fatigued muscles under heat stress. They are caused by electrolyte imbalances and dehydration, and are often seen in people who have been profusely sweating and in athletes

who exercise vigorously while rehydrating with hypotonic liquids such as water. Prevention algorithms include rehydrating with sports drinks when exercising in the heat and ensuring adequate dietary sodium intake during meals. Heat cramps are treated by withdrawing from the activity, resting in a cool environment, and hydrating with sports beverages or salty foods. Many people may have re-occurring episodes of heat cramps, highlighting the importance of identifying any modifiable, pre-disposing factors such as a low-salt diet, salty sweaters (those with a very high sodium concentration in their sweat)⁸, and those unaccustomed to the duration/intensity of exercise. Exertional complications of sickle cell trait (SCT) includes muscle cramping and diligence should be taken to differentiate between the two.⁹ In general, SCT causes cramping that is painless and the muscles look and feel normal.

HEAT SYNCOPE

Heat syncope manifests as lightheadness or a temporary loss in consciousness. This typically occurs while standing for long periods of time in the heat or immediately after cessation of exertion. When an individual is exposed to a hot, humid environment, cutaneous blood vessels dilate to cool the body. Venous pooling results in inadequate venous return and decreased cardiac output compromising cerebral perfusion and leading to syncope. Recovery is quick once supine. The elderly are at an increased risk of heat syncope as a result of underlying medical conditions and the medications used to treat them. It is critical that this is distinguished from collapse due to a more serious pathology; for example, arrhythmogenic or SCT collapse does not resolve when supine, as opposed to the transient nature of heat syncope.⁹

HEAT EXHAUSTION

Heat exhaustion is the inability to continue work or exercise in the heat and occurs when an individual has not maintained their fluid or electrolyte intake. This can develop over repeated exposure to elevated temperatures, especially in the non-acclimated population. In addition to physical exhaustion, heat exhaustion includes profuse sweating, weakness, chills, malaise, nausea, vomiting, and irritability. Risk factors include inadequate fluid intake, a BMI greater than 27 kg/m², and an inappropriate work-to-rest ratio. Objectively, the individual will have an elevated core temperature but, in contrast to exertional heat stroke, the temperature will remain below 40°C (104°F). Treatment includes immediate removal from the environment to a cool area. Clothing should be loosened and athletic equipment (helmets, shoulder pads, etc.) removed. Patients with heat exhaustion should also rehydrate with cool fluids and avoid heat stress for 24 to 48 hours. Replacement should be done with cool fluids as this is better absorbed.

HEAT STROKE

Heat stroke occurs when the body's cooling mechanisms become overwhelmed and are unable to keep up with the heat gained/produced by the body. There are two classifications of heat stroke, classical heat stroke (CHS) and exertional heat stroke (EHS). Both are fatal if untreated. CHS typically occurs in the elderly with predisposing conditions or taking medications that impair heat loss and in young children who are left in unventilated cars on hot days. This is a non-exertional form of heat stroke. In contrast, exertional heat stroke (EHS) typically occurs in healthy, young athletes who are exercising in hot or humid conditions and is among the top 3 leading causes of death in the athlete.

An elevated rectal temperature (usually >40°C (104°F)) with CNS dysfunction is the pathognomonic feature of heat stroke.^{10,11} Sweating is not a diagnostic criterion to differentiate between heat exhaustion and heat stroke.¹⁰ CNS dysfunction includes confusion, disorientation, irritability and in severe cases, coma, seizures, and delirium. Many EHS victims will have an initial rectal temperature between 106-109°F. A rectal temperature is the only valid temperature measurement in EHS cases as all other devices have been demonstrated to be invalid within a working/exercising population.¹²

COOLING METHODS FOR EHS

The treatment goal is to reduce body temperature to 102°F or less within 30 minutes of collapse as mortality increases significantly when cooling is delayed.¹³⁻¹⁶ To achieve this, cooling to 102°F prior to hospital transfer is optimal. Discontinuing cooling to transport the patient arrests the cooling process, subjecting the cells to continued denaturing, and placing the individual at risk for death. Optimal cooling equates to a mean cooling rate of >0.15°C per minute or >1°C every 6 minutes.

Ice water immersion offers a cooling rate of ~1°F every 3-4 min and is the most rapid and ideal method to lower core temperature.¹⁷⁻¹⁹ Ice water immersion should be started once heat stroke is discovered and discontinued when the body temperature is 102°F. Cooling should not be discontinued to transport to the hospital.

If ice water immersion is not possible, alternative methods such as cold water immersion,¹⁸ rotating wet ice towels or cold water dousing should be used even though they are not as efficient. Isolated fanning has been found to cool at a rate of 0.02° C/min. but has been found to potentiate the effect of other cooling methods. Ice packs applied to areas with major arteries and capillary beds including the axilla, groin, and neck has a cooling rate of 0.028°C/min. and even though commonly employed is inefficient. Even though these are not optimal methods of cooling, when used, can be continued en route to the hospital.

Common misconceptions limiting immersion is that ice or cold-water immersion would lead to peripheral

vasoconstriction retarding heat loss, and would result in shivering, increasing core body temperature. These hypotheses were originally observed in normothermic individuals but when investigated in those with EHS, have not been substantiated.¹⁷ If shivering becomes a concern, benzodiazepines can be administered when the patient is in a monitored setting.

PREVENTION

The elderly are at increased risk for heat illness. The ability to sense and dissipate heat are both compromised, underlying medical conditions, such as hypertension and diabetes, reduce the ability to dissipate heat and medications exacerbate this problem. While fans can help cool a person, this is not very efficient when temperatures approach 90°F. When available, air conditioning is optimal. During the warmest hours of the day the elderly should be encouraged to take a cool shower or go to the mall, library, a senior center, or the movies. Neighbors or relatives should check on the elderly twice a day to evaluate for heat illness. Eating small meals frequently is preferred in the heat and since the elderly and children don't sense thirst at the same serum osmolality as young adults, they should not wait until they are thirsty to drink fluids.

In athletes, the majority of heat illnesses occur in July and August as summer practices begin. Athletes are typically deconditioned and have not acclimated to the environmental conditions. Acclimatization results in decreasing basal body temperature, heart rate and results in an earlier onset of sweating. The NCAA and the Interassociation Task Force for secondary schools recommends a gradual increase in exposure to hot and humid environmental conditions over a 10- to 14-day period.²⁰ Many states have adopted heat acclimatization policies for secondary school athletics. The policy for Rhode Island can be found on the Rhode Island Interscholastic League website.

Proper hydration contributes to preventing heat illness as it can help keep body temperature and heart rate lower and dehydration may reduce the gains made during acclimatization. Hydration should occur before, during, and after exercise as outlined in the position statement by the American College of Sports Medicine (http://journals.lww.com/acsm-msse/Fulltext/2007/02000/Exercise_and_Fluid_Replacement.22.aspx). Athletes should be encouraged to avoid water losses of more than 2% of their body weight and to monitor their urine color as an indicator of hydration status.

Lastly, organized sports should ensure best practices including sideline personnel prepared to provide prompt care and an emergency action plan to optimize emergency care.

SUMMARY

Heat stroke is one of the top three causes of death for athletes. Vigilance is required to prevent these illnesses and when faced with an individual who is suffering an exertional

heat stroke, the goal is to aggressively cool the patient to 102°F within 30 minutes to optimize survival. The elderly are also at risk for heat illness and physicians caring for these patients should discuss prevention and treatment plans.

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