Heat-related illness

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Heat-related illness represents a continuum of disorders from minor syndromes such as heat cramps, heat syncope, and heat exhaustion to the severely life-threatening disorder known as heat stroke. It represents an important cause of wilderness-related morbidity and mortality, accounting for at least 7% of wilderness-related deaths [1].

While heat-related illness is seen most commonly during summer heat waves and high environmental temperatures [2], heat emergencies can occur in more temperate environments, especially in patients who have chronic medical conditions, mental illness [3], occupational exposure to high temperatures [4], or insufficient acclimatization [5].

Between 1979 and 1999 more than 8000 deaths in the United States were attributed to excessive heat exposure. Heat-related illness is most prevalent in the elderly and is more frequent in children under 4 years of age [6].

Physiology

Body temperature is regulated through a balance between heat creation (and absorption) and heat dissipation [7]. Heat is created as a byproduct of cellular metabolism and the mechanical work of skeletal muscle and is also gained by way of radiation from the sun, direct contact with hot objects, and absorbed when the ambient temperature rises above body temperature. Body heat is dissipated through all four physical mechanisms of heat transfer: (1) conduction of heat through the body from core to periphery, (2)
Convection of heat from the core to periphery by way of blood circulation and from skin surface to ambient air, (3) radiation of heat to the environment, and (4) evaporation of sweat into ambient air. Convection and evaporation are far more important than other methods of heat transfer because they are regulated primarily by the body to control temperature. In humans there is a negligible contribution of heat loss from the pulmonary capillary bed to ambient air by way of respiration; however, this mechanism is responsible for substantial heat dissipation in other mammals through panting.

The body exhibits a normal diurnal temperature rhythm, ranging from approximately 36°C in the early morning to 37.5°C in the late afternoon [7]. Body temperature is controlled within this narrow range by the preoptic nucleus of the anterior hypothalamus in the brain, which receives afferent information on cutaneous (surface) and internal (core) temperatures and relays efferent impulses to the skin by way of sympathetic nerves to stimulate cutaneous vasodilatation and sweating. Cutaneous vasodilatation increases convective transfer of heat from the core to the periphery by way of the systemic circulation [8]. Sweating results in increased heat dissipation by cooling the skin through evaporation. When the hypothalamic set point is normal but physiologic conditions are such that heat is being produced abnormally or cannot be dissipated, the condition is known as hyperthermia. In contrast, when the hypothalamic set point is elevated the condition is known as fever [7].

Exposure to high temperatures and an elevation of body temperature induces a number of changes in the cutaneous circulation. Skin blood flow increases from a baseline of approximately 250 mL/minute to approximately 6 to 8 L/minute through increased cardiac output and concomitant vasoconstriction of the renal and splanchnic circulation. This change results in an increase in heat dissipation well above the resting state of 80 to 90 kcal/hour. Evaporation of sweat cools the skin, further enhancing heat transfer. Cutaneous dilation and sweating increase as body temperature rises until a steady state of heat production and dissipation is again reached and body temperature stops rising. Endurance athletes and conditioned workers might reach and maintain body temperatures of 40°C (104°F) without substantial morbidity [9].

Acclimation

The human body’s response to heat stress is quite resilient if given several weeks for adaptation to occur. This process, known as acclimation or acclimatization, involves a number of physiologic and biochemical adjustments that allow an individual to undergo heat stress that would otherwise result in substantial morbidity or even death [10]. First, physical conditioning improves cardiac performance, making substantial increases in skin blood flow readily possible without compromising oxygen delivery to
critical tissues or leading to syncope or cardiovascular collapse. Second, acclimation results in expansion of plasma volume, increased renal blood flow, and improvement in the body’s ability to shunt blood away from noncritical circulatory beds (including the splanchnic circulation), thus improving the kidneys’ ability to withstand mild to moderate degrees of exertional rhabdomyolysis. Acclimation also results in enhanced activation of the renin-angiotension-aldosterone system, enhancing the ability of the kidneys and sweat glands to retain sodium and prevent volume depletion. Despite this enhanced ability to retain sodium, acclimated sweat glands can actually secrete a greater volume of sweat by secreting sweat with significantly less tonicity, making volume depletion less likely and putting the heat-stressed individual at less risk for volume depletion or dilutional hyponatremia if fluid replacement is maintained solely with free water or other nonelectrolyte-containing solution. Acclimation might also reduce the physiological set point or internal temperature thresholds for initiation of cutaneous vasodilatation and sweating and increase the degree of vasodilatation and sweating for a given temperature rise [8].

Pathophysiology

Mechanisms that disrupt convective and evaporative heat loss, including decreased cardiac reserve (impaired cardiac performance, drug therapy), volume depletion (as a result of exertion or diuretic therapy), systemic vasoconstriction (intercurrent illness), and conditions resulting in anhidrosis (anticholinergics, phenothiazines), significantly impair the body’s ability to dissipate heat and put patients at substantial risk for heat illness.

The pathophysiology of severe heat exhaustion and heat stroke has been the subject of recent clinical research [10–13]. Heat stress causes damage to the organism by way of at least three mechanisms. First, heat is directly toxic to cells. An increase in cellular temperature results in protein denaturation and interrupts critical cellular processes, resulting in apoptosis and cell death. Temperatures above 41.6°C to 42°C are considered to be above the critical thermal maximum for humans and can be expected to produce injury over even a few hours. Extreme temperatures, above 49°C, result in near immediate cell death and tissue necrosis. Second, heat stress results in release of inflammatory cytokines including tumor necrosis factor-α, interleukin-1 (β) and interferon γ, and the anti-inflammatory cytokines IL-6, IL-10, and TNF receptors p55 and p75. Finally, elevated temperatures seem to result in injury to vascular endothelium, resulting in enhanced vascular permeability, activation of the coagulation cascade, and disseminated intravascular coagulation (DIC).

In this respect, severe heat illness can be seen as a combination of direct cytotoxicity and a severe systemic inflammatory response in which encephalopathy predominates early in the course of the disease [10]. If left
unchecked, renal failure, coagulopathy, hepatic dysfunction, and ultimately multiple organ dysfunction system will result.

**Prevention**

Fluid replacement is of prime importance in preventing heat-related illnesses. Fluid replacement should be sufficient to replace all losses, and 1 to 2 L/hour might be necessary in extreme situations. Overhydration should be avoided; severe cases of hyponatremia have been reported in marathon runners, particularly in women and slow runners, secondary to excessive fluid consumption [14]. Fluid replacement is best accomplished with carbohydrate–electrolyte solutions, although free water is acceptable if sufficient salt intake through other mechanisms is ensured [9]. The consumption of salt tablets alone is dangerous and should be avoided. White or light-colored clothing reflects significantly more radiant energy and are of particular importance even on an overcast day when the ambient temperature is high.

Environmental temperature is only one facet of heat stress to organisms. Direct sunlight, relative humidity, and wind speed contribute significantly to heat stress. In conditions of high ambient temperature, the decreased gradient between the skin surface and the periphery makes convective heat transfer less efficient. During states of high humidity or low wind, evaporation of sweat is less efficient, thus the ability to dissipate heat is impaired further.

The best and most widely used single measurement of environmental heat stress is the wet-bulb globe temperature (WBGT) index, which is a function of ambient heat, humidity, and radiant stress from direct sunlight. It is given by the formula:

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\text{WBGT Index} = (0.7)T_{WB} + (0.2)T_{BG} + (0.1)T_{DB}
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Where \(T_{WB}\) is equal to the wet-bulb temperature measured with a water-saturated cloth wick over a dry bulb thermometer in direct sunlight, \(T_{BG}\) is equal to the black globe temperature, measured with a thermometer placed inside a standard black globe in direct sunlight and \(T_{DB}\) is equal to the shaded dry bulb temperature. While determination of the WBGT index requires a special instrument, analysis of the formula reveals that the index is predominantly driven by wet-bulb temperature. Hence, humidity plays the most critical role in heat stress. The American College of Sports Medicine recommends canceling sporting events when the WBGT index is above 28°C [9]. This index should serve as a general guide for planned wilderness activities and reduced for patients who have less than outstanding cardiopulmonary conditioning or insufficient acclimatization.
Minor heat-related syndromes

Heat edema

Heat edema results when cutaneous vasodilatation and pooling of increased interstitial fluid in dependent extremities lead to swelling of the hands and feet. It is self-limited and rarely lasts more than a few weeks. Treatment consists of elevation of the extremities, and, in severe cases, application of compressive stockings. Administration of diuretics might exacerbate volume depletion and should be avoided.

Heat syncope

Heat syncope results from volume depletion, peripheral vasodilatation, and decreased vasomotor tone and occurs most commonly in elderly and poorly acclimatized individuals. Postural vital signs might or might not be demonstrable on presentation to the emergency department. Patients should be thoroughly evaluated for injury resulting from a fall, and all cardiac, neurologic, or other potentially serious causes for syncope should be considered. Treatment consists of rest and oral or intravenous rehydration.

Heat cramps

Heat cramps are characterized by painful muscle spasms, especially in the voluntary muscles of the calves, thighs, and shoulders, which most often occur several hours after vigorous exertion and begin during rest or showering. Especially common during athletic events, they are thought to result from dilutional hyponatremia as individuals replace evaporative losses with free water but not salt [15–17]. Core body temperature might be normal or elevated. Treatment consists of rest, administration of oral electrolyte solution or intravenous normal saline, and admonishments to the patient to replace future fluid losses with a balanced electrolyte solution. Heat cramps generally occur in the absence of other symptoms, but they can also be accompanied by symptoms and signs of heat exhaustion.

Heat exhaustion

Heat exhaustion is the most common heat-related illness [18]. It is characterized by volume depletion that develops in conditions of heat stress [19]. Classically, heat exhaustion is classified as water depletion and salt depletion heat exhaustion. Water depletion heat exhaustion occurs in individuals working in a hot environment with inadequate water replacement. Salt depletion heat exhaustion takes longer to develop and occurs when individuals working in hot environments endure heavy sweating with concomitant replacement of fluid losses with hypotonic solutions. Most patients present with a combined picture of water and salt depletion.
Individuals who have heat exhaustion will present with systemic complaints including fatigue, weakness, dizziness, headache, nausea, vomiting, and muscle cramps. Patients might also experience orthostatic syncope. On physical examination these patients usually have core temperatures of less than 40°C and will not have signs of severe central nervous system damage. Depending on the predominant mechanism, patients can present with tachycardia, orthostatic hypotension, and clinical evidence of dehydration. Patients who have heat exhaustion will usually have profuse sweating.

Mild heat exhaustion and heat stroke are at opposite ends of the heat illness disease spectrum. Cases that fall in between might be difficult to differentiate [19]. If there is uncertainty differentiating between heat exhaustion and heat stroke, the patient should be treated for heat stroke promptly. If heat exhaustion is not treated appropriately it can lead to heat stroke.

Laboratory abnormalities, including hyponatremia or hypernatremia, can also be present depending on the predominant mechanism. In heat exhaustion, hyponatremia is suggested in persons presenting with altered mental status or seizures in the absence of severe hyperpyrexia, hypoglycemia, trauma, or other apparent cause, especially if symptoms progress after exercise has ceased [20].

**Classic heat stroke**

Classic heat stroke is typically seen in debilitated patients during high environmental temperatures and humidity. This condition develops over several days, most commonly during heat waves. Persons especially vulnerable to this condition are the poor, the elderly, alcoholics, persons who do not have access to air conditioning, and persons who are socially isolated. Patients taking psychiatric medications are at particular risk [21]. The use of diuretics and the presence of chronic and debilitating diseases such as cardiac conditions, dementia, and chronic obstructive pulmonary disease have also been associated with the development of heat stroke [22]. Infants and ill, febrile children are also affected more commonly [23].

The two major diagnostic findings of heat stroke are the presence of a core temperature of 40°C or more and central nervous system dysfunction [10]. Characteristically, patients presenting with classic heat stroke present with anhidrosis, but absence of sweat is not considered to be a diagnostic criterion. The adherence to strict diagnostic criteria might lead to a delay in the diagnosis and treatment of heat stroke with serious consequences for the patient [24].

Central nervous system dysfunction is usually manifested by delirium, seizures, or coma. Other manifestations of neuropsychiatric dysfunction such as hallucinations, cerebellar dysfunction, or bizarre behavior can also
be present. Patients can present with systemic symptoms such as nausea and vomiting similar to heat exhaustion but with altered mental status. Most patients will present with tachycardia and hypotension [2,22]. Hyperventilation, resulting in a respiratory alkalosis with concomitant metabolic acidosis, is commonly seen [7,25].

Laboratory abnormalities might include evidence of hemoconcentration, acute renal failure (ARF), elevated liver function tests, hypernatremia or hyponatremia, and hypokalemia [22,24].

**Exertional heat stroke**

Exertional heat stroke is seen more commonly in poorly acclimatized young persons involved in strenuous physical activity in a hot environment. Exertional heat stroke cases typically occur in healthy military recruits, miners, and athletes [24]. In retrospective analyses, most of the cases of exertional heat stroke are preventable [26].

Patients who have exertional heat stroke typically present with a core temperature of 40°C or more and central nervous system dysfunction manifesting as seizures, delirium, or coma. The majority of these patients present with profuse sweating [18,27], but this is not necessary to establish the diagnosis [28]. Patients usually present with tachycardia, hypotension, and tachypnea. Vomiting and diarrhea are common, occurring in up to two thirds of patients [29]. Twenty-five percent of patients who have exertional heat stroke will develop ARF [18]. Patients can also develop hemorrhagic diathesis as a result of DIC.

ARF, rhabdomyolysis, and disseminated intravascular coagulation are more common in victims of exertional heat stroke than in victims of classic heat stroke. Laboratory abnormalities might include elevated white blood cell count, elevated liver function tests, and hypokalemia in the early stages of exertional heat stroke, with hyperkalemia manifesting later. Sodium levels can be normal or slightly elevated depending on the hydration status of the patient. Patients can also have elevated creatinine phosphokinase (CPK) secondary to rhabdomyolysis. Patients who have peak CPK levels above 10,000 IU/L are at significant risk for the development of ARF [30]; however, low levels of CPK on initial evaluation do not rule out the development of ARF in patients suffering heat stroke [31]. Marked lactic acidosis, hypocalcemia, and hypoglycemia can also be seen.

**Field treatment**

All victims of even minor heat illness should be assessed for the possibility of—and continuously monitored for the development of—heat stroke. Patients should be removed immediately from the hot environment to a cool, shaded area. All clothing should be removed and initial cooling
begun while assessment of the patient is performed. If possible, core temperature should be determined. All victims who have hyperpyrexia and altered mental status in an appropriate wilderness scenario should be treated for heat stroke [15].

Patients who have possible heat stroke should be cooled immediately, even as determination and stabilization of airway, breathing, and circulation is performed. Morbidity and mortality is directly associated with the duration of elevated core temperature [29,32–34]. In particularly austere environments, prompt cooling might be difficult. While there is extensive debate in the literature regarding the best method for rapid cooling of heat stroke victims, in no way should cooling of the heat stroke victim be delayed if any method is available. Cold water or ice water immersion should be instituted if readily available. Alternatively, evaporative cooling by dousing the patient with cool water or spraying water over the patient’s skin and fanning the patient while transporting him or her to the receiving facility should be undertaken [35]. Evaporation can be achieved in a moving vehicle with open ventilation or by placing the patient within helicopter downdraft. Ice packs are frequently applied over large vessel areas in the neck, axillae, and groin; however, several studies have not shown a significant reduction of cooling time with their use [36,37].

If possible, a core temperature should be checked as frequently as every 5 to 10 minutes during the cooling process to ensure adequate cooling and to prevent iatrogenic hypothermia. Patients should be placed on a cardiac monitor and administered supplemental oxygen if available.

Hypotension should be treated with intravenous normal saline. Most victims will not require large amount of intravenous fluids. Seizures and excessive shivering can be treated with a benzodiazepine. Victims of heat stroke need to be transported rapidly to an appropriate medical facility.

Healthy patients suffering less severe signs of heat illness need not be immediately evacuated but provided with sufficient volume repletion and rest before transfer out of field; however, patients at any risk for the development of heat stroke should undergo constant supervision and monitoring and removal from the environment.

Hospital treatment

Heat cramps

Patients experiencing heat cramps should be treated according to their presentation. Most cases are relieved rapidly by administration of balanced salt solutions [15,19]. Mild cases without evidence of dehydration can be treated using oral salt solutions 0.1% to 0.2% (0.25–0.5 teaspoon of table salt in 1 quart of water). Severe cases should be treated using intravenous saline solution 0.9%, which usually produces rapid relief.
Heat exhaustion

Treatment for patients presenting with heat exhaustion should be tailored to the patient’s presenting symptoms. Failure to treat heat exhaustion might allow progression to heat stroke. If the patient is hyperpyrexic and there is any question about the patient’s mental status, the patient should be treated aggressively for heat stroke.

Clothing should be removed and the patient should remain in a cool environment. Vital signs, including a rectal temperature, should be obtained. Core temperatures in these patients are more commonly below 40°C, and rapid body cooling techniques are seldom needed [23]. Mild cases, in which the patient is alert, has normal vital signs, and there is no clinical evidence of dehydration or vomiting, should be treated with rest in a cool environment and oral fluid replacement and salt replacement if needed. These patients are expected to have a full recovery in a short period of time.

Patients who present with more serious symptoms, abnormal vital signs, vomiting, and those who fail to improve with conservative treatment should be treated with intravenous fluids. The amount of normal saline solution needed depends on the degree of hypovolemia. The most common abnormal vital sign is tachycardia; orthostatic hypotension can also be present. Serum sodium, potassium, glucose, magnesium, calcium, and phosphorus should be measured as well as hematocrit, blood urea nitrogen, and creatinine. Electrolyte abnormalities should be corrected appropriately. Free water deficits should be replaced slowly over 48 hours to avoid complications. Rapid correction of hypernatremia can cause cerebral edema [16,19].

Young, healthy patients who do not have significant electrolyte or other laboratory abnormalities and who have adequate response to treatment can be discharged home after a period of observation. Patients should be advised to rest and continue adequate fluid intake over the next 24 to 48 hours [15,23]. Admission should be considered for elderly patients or patients who have chronic medical problems, significant electrolyte abnormalities, or risk of recurrence [19]. Patients who are adequately treated but who do not have complete resolution of symptoms over several hours should also be admitted.

Heat stroke

The most important aspect of treatment for heat stroke patients is rapid core temperature reduction. Morbidity and mortality are directly related to how rapidly the core temperature is reduced [29,32,33]. Controversy exists in the heat stroke literature regarding which is the most effective body cooling method for heat stroke victims.

Patients should be assessed and stabilized for airway, breathing, and circulation. Airway stabilization is extremely important because patients who have heat stroke can develop seizures, increasing the risk of aspiration. All clothing should be removed, intravenous access should be established, and the
patient should be placed on a cardiac monitor and administered supplemental oxygen. A urinary catheter should be placed to monitor urine output. While stabilized, the patient should be cooled rapidly. Constant core temperature monitoring is recommended using an esophageal or rectal probe.

The two most widely accepted methods of rapid body cooling are evaporative cooling and cold or ice water immersion. Controversy remains in the literature on heat stroke regarding which method is the more effective. Ice water immersion can reduce core temperature rapidly. In a study of 252 exertional heat stroke cases, ice water immersion lowered core temperature to less than 39°C in 10 to 40 minutes. There were no fatalities in this group [32]. In another study ice water immersion cooled twice as fast as evaporative cooling [38]. Opponents to rapid cooling using the ice water immersion technique state several theoretical and practical reasons to avoid this technique: inducing peripheral vasoconstriction and shivering can lead to a paradoxical increase in core temperature, discomfort to patients and to medical attendants, difficulty performing cardiopulmonary resuscitation, difficulty monitoring vital signs, and unsanitary conditions in cases in which vomiting and diarrhea occur [39]. In several studies in which ice water immersion was used, there was no report of any of the above-mentioned complications [28,33,38,40].

A body cooling unit was designed by Weiner and Khogali [41] using the evaporative cooling technique. When this device was tested in six experimental cases of exertional heat stroke it was shown to be faster than cold water immersion and did not induce shivering. The body cooling unit was tested in 18 cases of classic heat stroke taking from 26 to 300 minutes to reduce core temperature to 38°C. There were two fatalities in this group [42]. A randomized, controlled trial comparing ice water immersion and evaporative cooling in classic or exertional heat stroke has not been done.

It would seem that ice water immersion is the fastest way to lower core temperature. Evaporative cooling is an alternative primary rapid body cooling technique. Other techniques such as the use of cooling blankets, ice packs to the neck, groin, and axillae, and gastric and peritoneal lavage can be considered as extra treatment but not as primary treatment.

Heat stroke patients should be cooled rapidly to a core temperature of 38.5°C to 39°C, after which point active cooling should be stopped to avoid hypothermia. Continuous core temperature monitoring is needed to keep temperatures between 37°C and 38°C and to identify any rebound of hyperthermia requiring further treatment. Patients will show significant clinical improvement as the core temperature drops, including increase of blood pressure and mental status improvement.

Hematocrit, electrolytes, blood urea nitrogen, creatinine, liver function tests, CPK, coagulation studies, and urinalysis should be measured. Electrolyte and metabolic abnormalities should be corrected appropriately. Seizures and excessive shivering can be treated with benzodiazepines. Hypotension almost always responds to administration of intravenous
fluids, but in select cases patients might require vasopressors when volume resuscitation is complete. In at least one study, isoproterenol, a β-adrenergic agonist, has been suggested [40]. While isoproterenol has the theoretical advantage of increasing peripheral blood flow and cutaneous circulation, it is rarely used today and is unlikely to result in significant blood pressure rise. Alpha adrenergic agents such as dopamine have the theoretical disadvantage of causing peripheral vasoconstriction at higher doses and they might impair cutaneous perfusion; however, they can be expected to raise blood pressure more reliably.

Patients can develop renal failure, requiring dialysis. A history of environmental exposure to heat is frequently encountered in patients who have sepsis or another infectious cause of fever. If the patient’s history is unclear or an infectious etiology for fever is possible, workup and presumptive treatment for sepsis, meningitis, or other serious bacterial infection should be started. Heat stroke patients should be admitted to an intensive care unit for appropriate monitoring.

Even with the most sophisticated life support and rapid cooling techniques, prevention remains the most important intervention for heat injuries. Awareness of risk factors and weather conditions, adequate clothing, hydration, and access to shaded, cooler areas during heat waves can prevent most heat injuries. Elderly patients at risk for classic heat stroke should be checked on frequently during times of extreme hot weather. It is also important to avoid strenuous physical activity during extreme hot weather and to have frequent rest periods and adequate fluid and salt intake during physical activity in hot, humid weather [9,43].

Outcomes

Most patients who have heat injuries have good outcomes if they are treated promptly. In patients who have heat stroke, mortality should be less than 10% with adequate treatment and supportive care. Adequate treatment of heat stroke includes rapid reduction of core temperature, prompt attention to the ABCs (Airway, Breathing, Circulation), cardiovascular support, control of seizures, and prompt transport to an emergency medical facility. A mortality rate of 14% was found in a series of 28 cases of classic heat stroke [22]. Poor prognostic factors include hypotension, the need for endotracheal intubation in the emergency department, altered coagulation profile in the emergency department, and advanced age [44].

The vast majority of patients who have exertional heat stroke will recover without sequelae [27]. Long-term effects for heat stroke survivors are rare with adequate treatment. In a case-control study of 21 young patients suffering exertional heat stroke followed up for 6 months and tested for heat tolerance and psychological sequelae, none were found to have any abnormal findings [45].
Moderate to severe residual neurologic defects in classic heat stroke survivors have been reported in up to 33% of cases [2]. Neurologic deficits that have been reported are paraplegia, paresis, dysarthria, memory loss, concentration difficulty, and ataxia [27]. With appropriate rehabilitation, significant motor and cognitive function can be recovered over time [46].

References