



Heat-Related Illness

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Heat-related illnesses encompass disorders ranging from minor syndromes to life-threatening emergencies. The number of children suffering from heat-related illness is increasing. Because of physiologic differences and unique behavioral characteristics, children are at high risk for suffering heat-related illnesses. This article reviews physiologic responses to heat stress and highlights particular differences and behavioral considerations unique to children. It will address the diagnosis of heat-related illness in the emergency department, including the need for accurate temperature assessment and laboratory diagnostic tests. Management strategies follow a description of each illness. The science surrounding possible treatments for moderate to severe heat-related illness is reviewed.

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Heat-related illnesses encompass a spectrum of disorders from minor syndromes including heat edema and heat cramps to heat stroke, a life-threatening emergency. These illnesses arise when there is a disruption in the regulation of the body's temperature because heat input from the environment and body metabolism is increased compared with heat output from the skin via radiation, evaporation, and convection. Although heat illness is most often associated with tropical and wilderness medicine [1], summer heat waves also pose a serious health risk. Even in temperate climates, susceptible patients including the elderly, infants, children with cystic fibrosis, and patients with chronic medical conditions are at a significant risk for such illnesses [2,3]. During 1999 to 2003, more than 3400 deaths attributed to heat were reported in the United States, most occurring in the elderly, but 228 (7%) of these deaths occurred in children younger than

15 years [4]. Previously, over a 20-year span (1979-1999), only 4% of 8000 heat-related deaths were reported in patients younger than 15 years [5]. This increase in mortality demonstrates the need for adequate awareness, knowledge, and education regarding children and risks for heat injury and illness.

Physiology

Body temperature is the result of an interplay among heat production, absorption, and dissipation. Physiologic mechanisms regulated primarily by the hypothalamus maintain core body temperature between 36°C and 37.5°C despite wide variations in ambient temperature [6]. Fever is an elevation of core temperature resulting from increased metabolic activity and shivering [7]. In contrast, the hyperthermia of heat illnesses represents an elevation of core temperature attributable to an imbalance between absorption of heat from the environment and/or the failure to dissipate it.

Heat transfer occurs in the following 4 ways: Conduction describes energy transferred from one solid object to another as a result of direct contact and a difference in temperature. Convection occurs when a gas or liquid absorbs heat and moves it away from the body. Radiation of electromagnetic (infrared) waves, primarily

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from the sun itself, accounts for most of the heat absorbed from the environment. Evaporation is the change from a liquid to a gaseous (higher energy) phase. Of these 4 mechanisms, radiation and evaporation account for most of the heat transfer in humans [6,8]. Because the ability to dissipate heat via radiation decreases as ambient temperature increases, convection gains greater importance in preventing heat-related illness. Convection and evaporation are directly controlled by innate physiologic responses, namely, circulatory dynamics and sweating.

Heat Loss via Circulatory Changes

The skin has a large vascular supply that provides an effective means of dissipating heat. In adults, blood flow may vary tremendously given environmental and host conditions. In severe hypovolemic states, blood flow may be near zero; however, blood flow to the skin has the potential to consume as much as 30% of total cardiac output, rivaling the flow of blood to the brain [6]. Vasomotor tone modulates blood flow and, thus, heat transfer. These changes in tone may produce as much as a 6-fold increase in heat conductance to the skin [9]. These marked variations in flow can alter perfusion to other organs and contribute substantially to the pathology seen in all forms of heat illness.

Heat Loss via Sweating

Evaporative cooling is the most important physiologic mechanism for dissipation of heat for humans. Eccrine sweat glands, found throughout the body, can produce up to 1 to 2 L of sweat per hour [6]. If sweat does not evaporate either because of physical barriers (eg, clothing or athletic protective equipment) or high humidity, sweating results in fluid losses without a cooling effect.

Sweat production also relies heavily on secretion of sodium and chloride into the sweat duct lumen. Initially, the concentration of these ions parallels that of plasma. However, the distal sweat duct reabsorbs most of the sodium and chloride with the final concentration ranging between 5 and 60 mEq/L depending on the degree of acclimatization [6]. The unacclimated are at a greater risk for electrolyte imbalances related to heat illness.

Behavioral Responses

Usually only mentioned briefly in discussions of the pathophysiology of heat-related illness, behavioral adjustments to ambient temperature account for the greatest degree of control over the balance of heat absorption and dissipation. These adjustments include activities such as seeking shade or avoiding strenuous exercise in high ambient temperatures. This makes infants and young children vulnerable to heat illness because they cannot make these decisions on their own. Two studies in preterm and term infants report decreased motor activity and increased extensor positioning in response to higher isotherm temperatures [10,11].

Pediatric Considerations

Young children mount different physiologic responses to heat stress compared with adults and thus display greater risk of suffering heat-related illness. Differences include greater surface area-to-mass ratio, higher metabolic rate, inability to increase cardiac output, greater ability to alter peripheral blood flow, lower blood volume, and lower amount of sweat produced per gland [8,12].

Most of the heat absorbed during environmental stress comes from radiation. The greater relative surface area-to-mass ratio of a child allows for absorption of more incident radiation being distributed to less tissue. An equivalent dose of radiation would be expected to raise core temperature more in a child than an adult.

Children younger than 5 years fail to increase their cardiac output in the face of significant heat stress when compared with older children, adolescents, and adults [12]. Although their heart rate increases, their stroke volume declines. Despite this, children demonstrate a greater ability to increase blood flow to the skin compared with adults [12]. At first glance, this may seem advantageous; however, in combination with a lower blood volume, this may contribute to greater susceptibility to syncope, exhaustion, and cardiovascular collapse.

Sweat production appears to be a product of both the size of sweat glands (smaller in children) and the sensitivity of the gland to heat stress (less sensitive in children). In general, prepubertal children produce less sweat than postpubertal children and adults [12]. This limits the heat-stressed child's ability to use evaporative heat loss in the face of heat stress.

Their developmental status also places children at greater risk of heat-related illness because of their inability to move independently from one environment to another. The nonambulatory and very young child depends on adult caregivers to remove him from direct sunlight or a closed automobile. They cannot regulate air-conditioning units, fans, and thermostats. Adolescent athletes often succumb to the pressures of coaches to exercise in extreme temperatures with limited water breaks and in heavy protective equipment. Their perceived invincibility and lack of experience compound these factors and place them at greater risk of heat-related illness.

Pathophysiologic Changes Resulting From Severe Heat Stress

Unlike febrile illnesses where the thermoregulatory mechanisms remain intact, core temperatures in severe heat illness can rise to levels that injure cells directly. As noted previously, core temperatures are typically maintained in a narrow range, in which enzymatic processes

and the related cellular proteins function optimally. Heat denatures cellular proteins leading to an interruption of metabolic processes. Disseminated intravascular coagulation (DIC) may result from direct injury to vascular endothelium, and vascular permeability may also be altered [13]. Hyperventilation produces an initial respiratory alkalosis that eventually gives way to metabolic (lactic) acidosis as direct injury to cells progresses. Most of the manifestations of shock (shock liver, oliguria and acute tubular necrosis, altered mental status, gastrointestinal mucosal injury) and the subsequent consequences of cellular injury in those tissues can be found in heat stroke.

Specific Heat-Related Illnesses

Miliaria Rubra (Prickly Heat)

This heat rash is common in infants and young children, especially during the hot and humid months of summer. Obstruction of epidermal and dermal sweat ducts, usually due to tight fitting clothes or emollients, produces an erythematous papular rash that may include pustules. Typically concentrated on the face, upper trunk, and the neck, it may also be seen on extremities. Treatment consists of wearing loose fitting clothes and removing oil-based topical lubricants.

Heat Edema

Heat edema is swelling of the extremities due to vasodilation and venous stasis. Predominantly involving the elderly, edema develops after prolonged periods of sitting or standing. During these periods, cutaneous vasodilation occurs and when combined with venous stasis results in accumulation of interstitial fluid in the dependent extremities. The edema is not complicated by symptoms of lymphatic disease or congenital heart disease. It is self-limiting and rarely lasts longer than a few weeks. Treatment consists of limb elevation and compressive stockings in more severe cases. Diuretics are not indicated and may potentially exacerbate fluid depletion.

Heat Syncope

Heat syncope is fainting secondary to insufficient cerebral perfusion during and after exertion in the heat. Volume depletion, peripheral vasodilation, and decreased vasomotor tone increase blood flow to the periphery of the body while decreasing central venous return, which leads to decreased blood flow to the central nervous system. Heat syncope most commonly occurs in elderly and poorly acclimatized individuals. Immediately after a syncopal episode, the patient's skin may be cool and moist; transient hypotension with a weak pulse is often present. However, abnormal vital signs may not be present upon presentation to a medical facility. Core temperature is normal or slightly elevated.

Treatment consists of placing the patient in a recumbent position, rest, cooling, and oral or intravenous rehydration. Patients should be thoroughly evaluated for any injuries resulting from falls. Cardiac and neurologic disease as well as other potentially serious causes for syncope should be considered in the differential diagnosis depending on the findings of the history and physical examination.

Heat Cramps

Heat cramps are sporadic, often severe muscle spasms, which most commonly occur in the voluntary muscles of the extremities and abdomen. They arise abruptly after vigorous exertional stress. Cramps tend to occur during rest, after work is complete, or while showering and are believed to result from electrolyte depletion [14]. They are a common occurrence during and after sporting events, or physical labor, when individuals profusely sweat and replace salt and water losses with free water without adequate salt replacement.

Muscle spasms typically occur for only a few seconds at a time but may last for several minutes or longer. During spasm, cramping muscle is often palpated as a firm mass. Exposure to cold air or water and voluntary contraction or passive movement of muscles may produce a cramp. Core body temperature is usually normal, although it may be slightly elevated, and laboratory findings include hyponatremia, hypochloremia, and occasional hypokalemia with low urine sodium. An individual with heat cramps will rarely have systemic complaints, although he or she may present with signs and symptoms of heat exhaustion.

Treatment of heat cramps consists of replacing fluid losses and correcting electrolyte abnormalities. Rest and increased salt intake with adequate fluid intake is usually sufficient. An oral electrolyte solution can be used. If cramps are prolonged or do not resolve with conservative measures, treatment with an intravenous infusion of 5 to 10 mL/kg of normal saline over 20 minutes is recommended [14]. Appropriate management of salt balance to prevent future cramping should be discussed with the patient.

Heat Exhaustion

Presentation and Evaluation

Heat exhaustion is an illness with relatively vague and nonspecific symptoms. On the spectrum of heat-related illness, it represents a moderate degree of compromise but may be a warning of impending heat stroke, a medical emergency. General irritability, fatigue, weakness, light-headedness, headache, increased thirst, nausea, vomiting, and muscle cramps are common symptoms. Systemic symptoms differentiate heat exhaustion from cramps. Temperature may be normal, though mild hyperthermia (<40°C) is not uncommon. Core

temperatures must be followed with rectal measurements because peripheral measurements may not be accurate. Mild tachycardia, orthostasis, and tachypnea may be noted. Sweating is typically preserved. Other physical findings include dry mucous membranes, flushed skin, and muscle tenderness.

Two categories of heat exhaustion exist and differ in their etiology and time course. Water depletion (hypernatremic) heat exhaustion arises more rapidly from inadequate fluid replenishment. Salt depletion (hyponatremic) heat exhaustion results from more prolonged free water replacement with insufficient sodium intake [8]. Salt depletion is more likely to occur in a child with cystic fibrosis because salt losses in their sweat do not allow them to acclimatize [14]. A basic metabolic panel may identify hypernatremic or hyponatremic states. However, purely isolated forms of either are rare [15,16]. Other general signs of dehydration may be noted in the laboratory evaluation, including elevated hematocrit and serum urea nitrogen. Elevations of transaminases, creatinine, and creatine kinase as well as acidosis should alert the physician to the possibility of heat stroke.

Management

The critical step in managing any heat-related illness is removal of the patient to a cool shaded environment with cessation of activity. If the scenario allows, remove clothing to improve convective and evaporative losses. These measures, in addition to the provision of oral fluids, constitute the mainstay of heat exhaustion treatment. Antipyretics play no role in the treatment of heat illness and could exacerbate injury.

Significant vital sign abnormalities, vomiting, and severe or recalcitrant muscle cramps, and significant dehydration accompanying heat exhaustion warrant intravenous fluid resuscitation. Laboratory studies (basic metabolic panel) should be obtained before initiating volume expansion. Active cooling methods are typically not necessary because the mild temperature elevation is not likely to be directly toxic to cells.

Once the symptoms have subsided, temperature and other vital signs have normalized, and, if present, laboratory abnormalities resolved, the patient suffering from heat exhaustion may be safely discharged. Discharge instructions should emphasize avoidance of heat stress in the following days as well as instructions on fluid and electrolyte replacement. Persistent signs and symptoms should prompt the physician to consider alternative diagnoses and possible admission.

Heat Stroke

Heat stroke is a medical emergency. The differentiation between heat exhaustion and heat stroke relies on a core temperature greater than 40.6°C and the presence of

central nervous system dysfunction. Although sweating is often absent, do not rely on this sign as an indication of heat stroke.

Most episodes of heat stroke occur during summer heat waves where ambient temperatures and humidity remain high for several days [17,18]. Most of the mortality occurs in the elderly population, although children with febrile illnesses or those trapped in automobiles constitute other at-risk populations [18,19]. Reports indicate that even with temperate daytime temperatures (23°C or 73°F), intravehicle temperatures can exceed 50°C (122°F) [20].

Types of Heat Stroke

Traditionally, heat stroke has been categorized as either classic or exertional. Classic heat stroke more commonly afflicts children and the elderly and is more insidious in onset. Infants and small children are at greater risk because they cannot independently secure fluids, alter their environment, or seek medical attention with milder forms of heat illness. Fever, with its increased endogenous heat production, compounds environmental exposure.

A healthy male between adolescence and middle age who is working or exercising in a hot environment typifies the victim of exertional heat stroke. Unlike the classic presentation, these individuals are often still sweating at presentation and healthy at baseline. Indeed, the ability to generate significant heat from muscle use is inextricably tied to the development of heat stroke in this population.

Presentation and Evaluation

Despite the mentioned distinctions, heat stroke may affect persons of all ages and health status. Systemic manifestations are present because heat stroke is a multisystem disease. The central nervous system and cardiovascular changes are the most profound and easily recognizable in the patient suffering from heat stroke. Central nervous system abnormalities include seizures, delirium, and coma in the most severe forms but more commonly include severe headache, hallucinations, irritability, and cerebellar dysfunction including ataxia. Tachycardia is typically present as physiologic mechanisms attempt to maximize heat dissipation through convection. Maximal cutaneous vasodilation steals a significant amount of blood from the vital organs and lowers peripheral vascular resistance, placing the patient at risk for distributive shock. As in other forms of shock, hypotension is a relatively late finding in the heat stroke victim and should warn the physician of impending circulatory collapse. Hyperventilation with respiratory alkalosis, pulmonary edema, diarrhea and vomiting, oliguria, muscle spasm, and either dry hot skin or profuse sweating are other signs of heat stroke (Table 1).

Laboratory evaluation may reveal numerous abnormalities. Hyper- or hyponatremia, hypokalemia, hypo-

Table 1 Signs and symptoms of heat illness.

Heat Exhaustion	Heat Stroke
Irritability	Seizures
Fatigue/weakness	Delirium/hallucinations
Light-headedness	Ataxia
Sweating	+/- sweating, but hot skin
Headache	Headache
Nausea/vomiting	Nausea/vomiting/diarrhea
Tachycardia	Tachycardia
Increased thirst	Syncope
Muscle cramps	Hyperventilation
Temperature, <40°C	Temperature, ≥40°C

glycemia, and azotemia may be noted on the basic metabolic panel. Elevated prothrombin time and partial thromboplastin time and decreased platelet count suggest DIC, which is more typical of exertional heatstroke [8,13]. Because of the heavy muscle activity accompanying exertional heat stroke, elevated serum creatine kinase and myoglobinuria may be seen. Elevated transaminases indicate liver injury. Elevated white blood cell counts are possible.

Management

As with any emergency, airway, breathing, and circulation are addressed first. Endotracheal intubation for coma, pulmonary edema, or shock is warranted. Succinylcholine should be avoided given the risk of malignant hyperthermia and myoglobinuria. Normal saline and Ringer's lactate are appropriate resuscitative fluids; concerns for sodium imbalance can be addressed after the patient's condition stabilizes. In severe cases of heat stroke, one may consider cooled intravenous fluids; however, no studies have evaluated their utility. Prudence should be exercised in providing parenteral fluids so as to avoid exacerbating pulmonary edema. In addition, as active cooling methods take effect, peripheral vasoconstriction will return significant blood volume to the central circulation [21]. Adjuncts include esophageal or rectal temperature probes, cardiorespiratory monitors, bladder catheterization, and an orogastric tube.

After ensuring that appropriate resuscitation is underway, attention focuses on rapidly cooling the patient to reverse the hemodynamic effects and limit further injury. This begins in the field by evacuating the potential heat stroke victim to a cool and shaded area, removing clothing and assisting evaporative and convective heat dissipation with misted water and fanning if available. Ice packs may be placed where major vessels travel near the skin surface (neck, axillae, and groin), although the utility of this practice has not been firmly established.

Once in a health care facility, more sophisticated modalities are used. Cool or ice water immersion and evaporative cooling are 2 widely accepted methods;

however, no trials have compared these methods. Obviously, water mist with fanning is likely to be safer, better tolerated, and more comfortable for small children. In addition, submersion adds to the difficulty of managing medical instrumentation (eg, monitors, venous access devices, and endotracheal tube). If cool water immersion is implemented, continuous direct observation of the patient is mandatory. Cool water may be less uncomfortable than ice water with similar rates of cooling [16]. Seizures should be managed aggressively in both prehospital and hospital settings to avoid the endogenous heat production associated with increased muscle activity.

The goal of active body cooling is not normothermia but to lower body temperature enough that physiologic mechanisms may work more effectively. Once core temperature has fallen below 39°C, active cooling should cease to avoid hypothermia [15]. The patient should improve clinically as the temperature returns to normal. Admission to a pediatric intensive care unit should be strongly considered given the potential sequelae of heat stroke such as renal failure, neurologic injury, pulmonary edema, and DIC. In patients that survive the acute illness, most recover completely [22].

Summary

Although most patients appear to recover, heat-related illness is preventable. Children are at increased risk of heat-related illness. Developmental limitations and chronic diseases compound this risk. Treating heat illness requires the physician to be cognizant of its nonspecific presenting symptoms, the populations most at risk and the potential for life-threatening complications. Prompt diagnosis and treatment are crucial.

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