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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.

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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 isolette 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