Prevention of Heat-Related Illness

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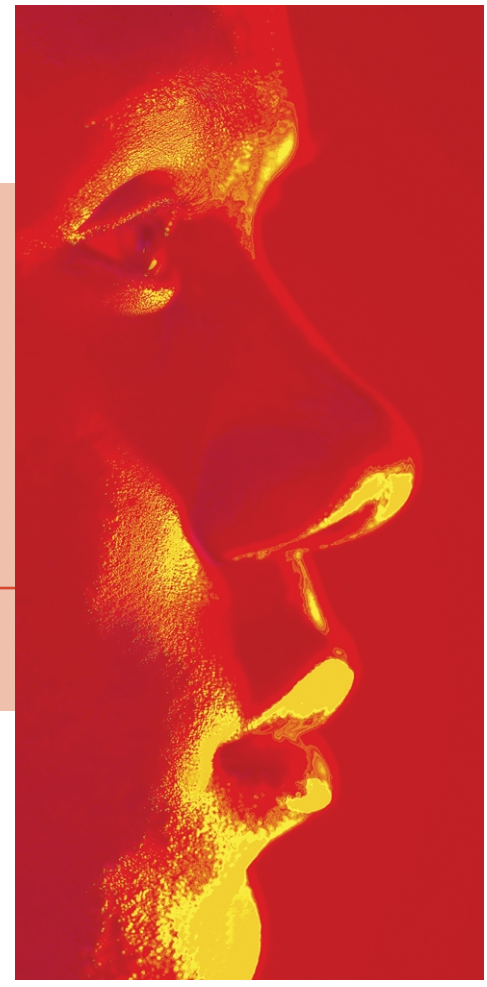

ABSTRACT

Heat-related illnesses, especially heat stroke, are a significant cause of morbidity and mortality in vulnerable persons during prolonged heat spells in temperate climates. Heat stroke produces a classic triad of symptoms: anhidrosis, temperature above 40°C (105°F), and mental changes. The very young and very old are especially vulnerable. Timely cooling measures must be initiated to prevent progression to multiple complications and death.

Keywords: Anhidrosis, heat stroke, thermoregulation, vulnerable populations

Heat stroke is a life-threatening disorder that occurs when a person is exposed to prolonged, high ambient temperatures. The effects of heat stroke and possible death have been known since ancient times. However, the pathogenesis of heat stroke was not established until the middle of the 19th century when it became evident that heat stroke resulted in neurologic abnormalities, multiorgan failure, and death when not diagnosed early and appropriately treated. Although anyone can experience heat-related illnesses, the very young, the elderly, those with chronic diseases, persons employed in outdoor situations, and athletes are more vulnerable.[1,2]

Every year in the United States and elsewhere, heat-related deaths occur in temperate climates during prolonged and unrelenting heat waves. In the heat wave of 1995, Chicago officials reported approximately 600 heat-related deaths. During the heat wave in August 2003, thousands of elderly, living in predominately urban areas in France, Italy, and other European countries, died of heat stroke. Similarly, in the summer of 2006, a significant

number of deaths were attributed to heat stroke during heat waves.[3-5]

PHYSIOLOGY OF THERMOREGULATION

Thermoregulation is controlled by the thermostatic center in the preoptic area of the hypothalamus. A rise in the core body temperature by less than 1°C stimulates peripheral and hypothalamic heat sensors and a thermoregulatory response, resulting in sympathetic cutaneous vasodilatation and sweating.[6,7]

The internal (core) body temperature is a sum total of heat produced by the body and environmental heat. Normally, it is maintained almost constantly at about 37°C (98.6°F) varying about 1 degree during a 24-hour period except during a febrile illness during which time the hypothalamus temporarily resets the core temperature upward. Heat production is determined by the basal metabolism rate. In turn, the basal metabolism rate is affected by thyroxin and certain cholinergics, muscle activity during strenuous exercise and shivering, and the action of the autonomic nervous system.[6,7]

The external (skin) temperature rises and falls with the surrounding ambient temperature; its ability to rise and fall is important in its ability to allow the body to lose heat to its surroundings. Heat transfer and heat dissipation from the organs are facilitated through the vascular system through vasodilatation and activation of the sweat glands. At this point, heat loss occurs through radiation, conduction, convection, and evaporation.[6,7]

Of these several mechanisms, radiation and evaporation are the most important. Conduction allows for approximately 2% of heat loss by transferring heat to a cooler surface or object. About 10% of heat is lost to the air and to water by convection. However, when the ambient air temperature exceeds the body heat temperature, heat is gained instead of lost.[6,7]

Radiation accounts for approximately 65% of heat transfer as long as the surrounding air is cooler than the body temperature. Once the ambient air temperature reaches 35°C (95°F), radiation becomes ineffective and evaporation is the only means of losing body heat.[6,7]

About 30% of body heat is lost through evaporation. In a dry environment, sweating can dissipate about 600 kcal/hour; a thermal gradient is essential for heat to be transferred from the body to the environment. However, when the humidity level reaches and exceeds 75%, heat loss by evaporation begins to fall. At this point, perspiration does not result in cooling but does intensify problems with dehydration. Loss of water and sodium by sweating may be as great as 2 L or more per hour. When not replaced, dehydration and salt depletion impair thermoregulation.[6,7]

The location of heat waves is also an important factor in heat-related illnesses. The Northeast and Midwest have the greatest number of heat-related deaths because of poor adaptation to prolonged, extreme heat, and many do not have access to air conditioning except in public places. Acclimation occurs with incremental exposure over a period of weeks and allows the body to enhance cardiovascular performance, activate the rennin-angiotensin-aldosterone axis, conserve salt by the kidneys and sweat glands, expand plasma volume, and increase the body's ability to resist exertional rhabdomyolysis.[8]

Urban areas, little variability in temperature, and the heat index are also important factors in heat-related illness. Urban areas typically have higher heat indexes (a combination of temperature and humidity) than rural areas. This phenomenon, known as the "urban heat island effect," with other risk factors make a deadly combination. Experts note that infants younger than 1 year and adults 60 years and older were more at risk in these situations. High, unrelenting heat index for at least 3 days, living on higher floors of a multistoried building, living alone, not leaving home daily, lacking access to air conditioning, being confined to bed, being dependent on others for assistance, having chronic disease, taking tranquilizers, and drinking alcohol are all identified factors that increased the risk of heat-related illness and death.[8]

PATHOGENESIS

The pathogenesis of heat illness involves thermoregulation and heat regulation, an acute-phase response and the production of heat-shock proteins. Normally, the core temperature is maintained in a steady state with little variation. As the temperature in the blood increases by less than 1°C, hypothalamic and peripheral heat receptors activate the thermoregulatory center, resulting in increased cardiac output and minute volume, tachycardia, reduction of visceral perfusion, cutaneous vasodilation, and activation of cutaneous sweat glands. If water and salt lost through sweating are not replaced, dehydration and electrolyte imbalance occur, both of which impair thermoregulation.[9,10]