Heat-Related Illness

Walter F. Atha, MD

KEYWORDS

• Heat emergency • Heat stroke • Environmental heat emergency • Hyperthermia

KEY POINTS

- Evaporation is the most effective cooling mechanism in the emergency department; cooling blankets are less effective.
- Environmental assessment of risk for heat-related illness must include wet bulb global temperature for accurate evaluation of heat stress, including humidity.
- Do not rule out heat stroke based on lack of sweating.
- Definitions of heat-related illnesses are less important than is recognition of the severity of presentation, which drives subsequent evaluation and treatment.
- Elevations in liver function tests may be seen 1 or 2 days after the initial insult.
- Elements of critical care include careful volume replacement and tolerance of mild hypotension.

A heat-related illness is defined as a physiologic insult to the body from exposure to elevated temperature, which can lead to elevation of core body temperature that surpasses the compensatory limits of thermoregulation. Presentations may be acute or delayed. Particularly severe cases can be life threatening. Heat-related illnesses are classically distinguished from "febrile" emergencies in that increased temperature is caused by environmental heat stress, rather than a change in hypothalamic function in the setting of normothermic environmental conditions.[1] Heat-related illness represents a set of syndromes that exist along a continuum from less severe illnesses, such as heat exhaustion, to multiorgan failure with heat stroke. While strict diagnostic criteria remain elusive, common descriptions are generally sufficient to provide working case definitions. For example, the term heat stroke typically implies a core temperature elevated to at least 40°C and the presence of central nervous system (CNS) dysfunction. Working terms to describe various heat-related illness syndromes are also ample, for example, prickly heat, heat edema, and heat cramps.

According to the National Oceanic and Atmospheric Administration (NOAA), 2012 was the hottest year on record for the continental United States, with an average

Disclosures: None.
Department of Emergency Medicine, Howard County General Hospital, 5755 Cedar Lane, Columbia, MD 21044, USA
E-mail address: watha@jhmi.edu

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temperature of 55.3°F (12.94°C), 3.2°F higher than the twentieth-century average.[2] If ambient temperatures continue to climb, the incidence of heat-related illnesses is likely to increase, underscoring the importance of better evaluation, diagnosis, and treatment. Epidemiologic data are less robust than they could be if definitions were better standardized; underdiagnosis remains a problem. Even so, high mortality rates have been reported in highly publicized heat waves in the United States and Europe.[3–5] The death of approximately 14,800 individuals was attributed to the heat wave in France during the summer of 2003.[5] The years 2005 to 2009 saw the highest incidence of heat-related sports deaths ever recorded in the United States.

The European heat wave of 2003 emphasized the vulnerability of certain populations to heat-related illnesses, secondary to decreased capacity to dissipate heat and increased likelihood of dehydration. These groups include the elderly, small children (because of their relatively large ratio of skin surface area to body mass), those with medical comorbidities, people with poor access to climate-controlled environments, and those with psychosocial challenges. Occupational and recreational activities requiring strenuous exercise in hot environments may confer significant risk as well (eg, athletes, firefighters, military personnel).

PHYSIOLOGY

Normal human core temperature is maintained at roughly 37°C across all populations. Normal skin temperature is nearly constant at 35°C, creating the temperature gradient necessary to dissipate heat from the core to the periphery. The body's net exothermic metabolism constantly generates heat at a basal rate ofapproximately100 kcal/h.[6]

In addition, the body absorbs heat from and dissipates heat to the environment by 4 basic mechanisms: conduction, convection, evaporation, and radiation (**Table 1**). Conduction is heat transfer via direct contact between surfaces. Convection is heat transfer from a solid or liquid to a moving liquid or ambient air. Both conduction and convection result in net heat loss when the body surface is warmer than the ambient air or surface, and in net heat gain when the ambient air or contact surface is warmer than the body. Radiation is heat transfer via electromagnetic waves. With evaporation of sweat, the water changes from liquid to vapor, with a concomitant reduction in skin temperature of 0.58 kcal per milliliter of sweat.[7] These mechanisms are augmented by therapeutic cooling maneuvers. For example, the use of fans in conjunction with continually wetting the skin increases convection and evaporation-mediated heat transfer. The use of moving water in cold-water immersion systems increases the effectiveness of convective heat transfer. As ambient temperature and relative humidity increase, 2 principal mechanisms of heat dissipation, evaporation and convection, become less effective, thereby diminishing the effectiveness of fans when used alone.[8]

Table 1
Mechanisms of body heat transfer

	Mechanism	Dissipation/Absorption
Conduction	Direct contact between surfaces of divergent temperatures	Both: minimal
Convection	Transfer to a moving liquid or ambient air	Both: moderate
Radiation	Transfer via electromagnetic waves	Both: minimal
Evaporation	Change of state from liquid to vapor or water in sweat	Dissipation only: predominant

Environmental contribution to heat stress must take into account its impact on the body's compensation mechanisms. Relative humidity and temperature must be considered independently in assessing heat stress, because evaporation of sweat is the predominant cooling mechanism in elevated temperatures, and physiologically effective evaporation of sweat ceases when humidity exceeds approximately 75%. Wet bulb global temperature (WBGT) is the best known and most widely used scale of heat stress. WBGT accounts for independent contributions of absolute temperature, humidity, and radiant heat absorption. The calculation is:

$$\text{WBGT index} = [\text{DBT} \times 0.1] + [\text{WBT} \times 0.7] + [\text{GT} \times 0.2]$$

Dry bulb temperature (DBT) represents true ambient air temperature. Wet bulb temperature (WBT) is measured by covering a thermometer with a white cloth, sometimes called a sock, kept wet by wicking action. Globe temperature (GT) is a measure of the radiant heat effect from the sun and other proximate surfaces producing radiant heat. Analysis of the equation reveals that humidity, via WBT, is the largest contributor to the WBGT index. Complex devices are used to measure the WBGT index, but it can be reasonably approximated using mathematical estimation and allows comparison across regions. For example, WBGT along the east coast of the United States during peak summer months can be very close in range to WBGT in lower-latitude tropical regions and the hottest arid deserts on other continents. At present, the highest WBGT measurements on the planet across all outdoor natural environments are roughly 31°C.[9]

Afferent and efferent apparatuses maintain body thermoregulation. The preoptic nucleus of the anterior hypothalamus (POAH) is the temperature-sensing center in the CNS. The POAH alters the mechanisms for cooling based on temperature relative to its set point. When the hypothalamic set point is elevated, a "febrile" condition exists. A "hyperthermic" condition, on the other hand, is created by an exogenous heat source. When core temperature becomes elevated, the POAH, via sympathetic pathways, signals vasodilatation of the peripheral vasculature and shunting of blood away from splanchnic beds to preferentially perfuse the skin, augmenting heat dissipation. Blood flow to the skin can increase from a baseline of 250 mL/min to 6 to 8 L/min.[10] There is a corresponding increase in cardiac output in response to increased demand.

Increased sweat production, also signaled via the POAH, is another important mechanism of heat dissipation. Eccrine sweat glands cover most of the body, although they are more densely concentrated in the palms and soles, and are cholinergically activated. Apocrine sweat glands found mostly in the axillae are adrenergically activated and play little role in cooling. Evaporation of sweat is an efficient cooling mechanism, but depends on convection of air away from moist skin. Loose-fitting clothing allows air to circulate over moist skin. A core tenet of treatment for elevated body temperature is removal of all clothing, which increases the surface area exposed to convective air currents. Without circulation of air, a thin layer of heated air forms a local insulation barrier, preventing further convective cooling of the skin. High humidity decreases the cooling efficacy of sweating; at 95% relative humidity, sweating becomes essentially ineffective, and for this reason the WBT in the WBGT is weighted more heavily (by a factor of 0.7 vs 0.2 for radiant heat and 0.1 for dry temperature) to determine the effective heat risk.

In the cardiovascular system, increased demand results from decreased peripheral resistance, and shunting of a large portion of the circulating volume to the skin reduces preload. Inotropy is increased, and stroke volume remains the same or increases slightly. The large increase in cardiac demand is mediated primarily by increased heart rate. The increased chronotropy is stimulated by either a direct heat effect on the

sinoatrial node, or parasympathetic effects from the baroreflex and sympathetic effects from the heightened adrenergic state in hyperthermia.[11] People with poor baseline cardiac conditioning or decreased cardiac function as a result of myocardial damage or medications that suppress heart rate or cardiac work, such as β-blockers, are less able to increase cardiac output in response to the demand.[12] Hence, they are more likely to have decompensated heat-management mechanisms and rapidly elevate their body temperature. Semenza and colleagues[13] suggested that type 2 diabetics are at increased risk because of decreased peripheral vascular dilatation, presumably neutrally mediated, rather than purely cardiac dysfunction.

Relative dehydration caused by diuretics and other medications leads to the same effects of decreased circulating volume. Sweat production can be decreased by prescription and illicit medications, particularly those with anticholinergic effects. Hyponatremia, caused by increased sodium loss relative to water loss, can develop from increased sweat production. In lower mammals, panting contributes to cooling via convective heat transfer from the pulmonary capillary bed as well as a closed-loop system in the skull, which directly cools the brain; this mechanism is not significant in humans.

PATHOGENESIS AND CLINICAL PRESENTATION

When the cooling mechanisms fail, core temperature rises, leading to pathologic changes in several organ systems. The observed pathologic changes are thought to occur via direct cytotoxicity and a severe systemic inflammatory response (SIRS).[14] The cellular function of any tissue is affected by elevated temperature by denaturation of proteins, release of proinflammatory cellular mediators, including cytokines, and, at very high temperatures, cell death and apoptosis. The critical thermal maximum for humans is 41.6° to 42°C, at which point these cellular changes begin to take place. SIRS is thought to be mediated by direct injury to the vascular endothelium, causing leakage into the interstitial space. Concomitant direct activation by elevated temperature of the coagulation cascade and progression to disseminated intravascular coagulation (DIC) is a common complication of heat stroke.[15]

The 2 tissues most vulnerable to damage by elevated temperatures are the brain, particularly the cerebellum, and the liver. On this basis, the common understanding of the diagnosis of heat stroke requires both CNS changes and evidence of hepatocellular damage, manifested by elevated liver function tests (LFTs). CNS dysfunction can manifest as dizziness, confusion, dysmetria, ataxia, and, eventually, coma. The particular sensitivity of the cerebellum explains the predominance of cerebellar signs and symptoms early in disease progression. Hepatic and renal insult also occurs secondarily to hypoperfusion from CNS-mediated splanchnic and renal blood shunting to the skin, in addition to direct heat damage. Elevations in LFTs might not manifest for more than 12 hours, so normal values should not be used to rule out severe insult in clinically ill patients. Most patients with elevated LFTs will experience complete recovery without hepatic damage, when treated appropriately. However, an interesting case report attributed the death of a normal subject to fulminant hepatic failure that occurred directly after prolonged temperature elevation in a sauna.[16] Abnormalities in the CNS and the hepatic system distinguish heat stroke from heat exhaustion (which is not associated with CNS or liver insult). While many patients with extreme heat illness stop sweating, many continue to sweat profusely. Anhidrosis does not exclude the diagnosis of heat stroke.

Other organ system effects can be severe. Direct cardiomyopathy without evidence of coronary artery disease was attributed to prolonged heat exposure in a case

study.[17] The patient described in that report was the first for whom concomitant coronary artery occlusion was excluded by coronary angiography at the time of ST-segment elevation in conjunction with heat stroke. Stress-induced cardiomyopathy caused by heat exposure was the presumed diagnosis. Bowel ischemia from splanchnic shunting can manifest as diarrhea and has been implicated in recent research as contributing to the SIRS-like physiologic response seen in severe heat stroke with multiorgan dysfunction. Direct endothelial damage from heat exposure may also lead to SIRS physiology via the DIC pathway (**Fig. 1**). Using SIRS as a model, novel treatment approaches to severe heat stroke using immune-modulating agents such as recombinant human activated protein C are under consideration.[10]

DIAGNOSIS AND RISK STRATIFICATION

Strict diagnostic criteria for the heat-related illnesses do not exist, except for agreement that the diagnosis of heat stroke should include a temperature of 40°C and CNS dysfunction. Therefore, recognition of the severity and extent of heat exposure as the root cause of the presenting complaint(s) takes precedence. Early recognition of elevated core temperature, knowledge of environmental exposure, careful consideration of the differential diagnosis (**Box 1**), and a search for anything in the medical history that predisposes the patient to heat-related illness are all key. Two reasonably well-defined variants of heat stroke, exertional heat stroke and classic heat stroke, should be understood; they have different causes, but their final pathways are almost identical.

Heat stroke is divided into 2 categories: exertional heat stroke (EHS) and classic heat stroke (CHS). The distinction is important because their pathologic bases are

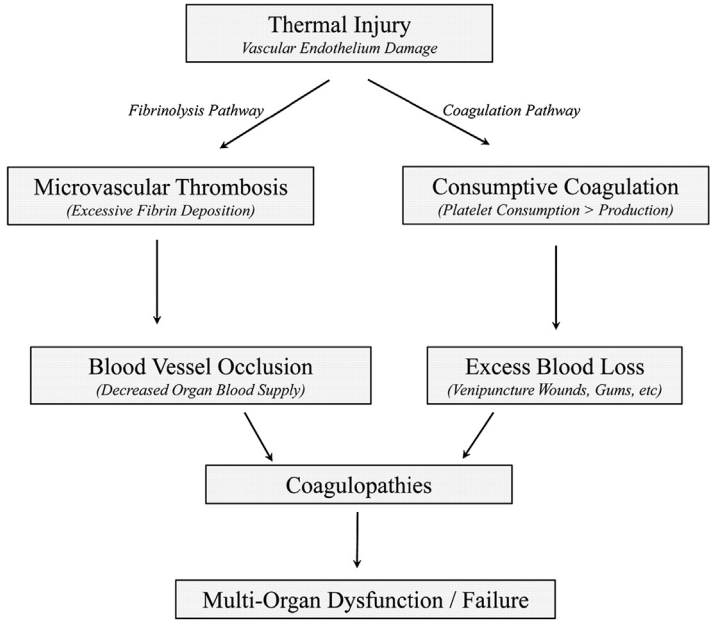

Fig. 1. Mechanisms of disseminated intravascular coagulation. (*From* Leon L, Helwig B. Heat stroke: role of the systemic inflammatory response. J Appl Physiol 2010;109(6):1983; with permission.)

Box 1
Differential diagnosis of hyperpyrexia

- Drug associated
  - Toxicity
    - Anticholinergic
    - Stimulant toxicity (phencyclidine, cocaine, amphetamine, ephedrine, MDMA)
    - Salicylate toxicity
  - Serotonin syndrome
  - Neuroleptic malignant syndrome
  - Malignant hyperthermia
  - Drug withdrawal syndrome: ethanol withdrawal
  - Drug-induced fever
- Infections
  - Generalized infections (eg, bacterial sepsis, malaria, typhoid, tetanus)
  - CNS infections: meningitis, encephalitis, brain abscess
- Endocrine derangements
  - Thyroid storm, pheochromocytoma
- Neurologic
  - Status epilepticus
  - Cerebral hemorrhage
- Environmental exposure
  - Heat exhaustion
  - Heat stroke
- Blood clots
  - Deep vein thrombosis, pulmonary embolism, deep-seated hematomas

Abbreviation: MDMA, 3,4-methylenedioxymethamphetamine (Ecstasy).
Data from Khoujah D, Hu K, Calvello EJ. The management of the hyperthermic patient. Br J Hosp Med 2011;72:571–5.

different in the populations at risk for them (they can be thought of as "active" [EHS] or "passive" [CHS]). EHS typically occurs in individuals participating in strenuous sports and those whose occupations lead to heat exposure during exertion, such as firefighters and military personnel. Even though these individuals might be in good physical condition, the environment or the use of gear elevates their temperatures beyond the regulatory range of the body's cooling mechanisms. Poor physical conditioning and lack of acclimatization (discussed later in the section on treatment) can contribute to the development of or worsen EHS. The US Army has produced a detailed technical bulletin for the recognition and treatment of heat exhaustion and EHS.[8]

CHS occurs among individuals who have impaired physiologic mechanisms for heat dissipation, stemming from comorbid metabolic or cardiac conditions, or who lack the means to escape a hot environment, for economic, psychiatric, or social reasons, including substance abuse, or because of physical challenges. CHS can be considered a passive process, although it could be exacerbated with even minimal exertion

in vulnerable populations. Decreased cardiac function limits the significantly increased cardiac output demands created by blood flow to the skin vasculature. Cardiac function can be decreased by structural heart disease and by medications used to treat heart disease. Diabetes can dampen the skin vascular vasodilatation response itself. The poor and homeless often do not have access to air conditioning, leading to group-cooling solutions that many cities adopt during hot summer months. Chronic behavioral/psychiatric challenges can prevent affected individuals from seeking appropriate environments during periods of elevated temperatures.

In the patient with undifferentiated altered mental status, heat illness may be overlooked unless core temperature is measured during the initial assessment. Patients at increased risk for CHS are also at increased risk for myriad other causes of depressed CNS status, so the diagnostic workup should not cease with the recognition of elevated core temperature. Other life-threatening causes to be considered include infection, endocrine derangements, neurologic issues, and drug-induced syndromes (**Table 2**).

The clear consensus for assessment of core temperature is rectal measurement. Oral, axillary, skin sensing, and tympanic membrane methods have all been shown to be inferior to rectal measurement.[18] Rectal temperature assessment is recommended by sports medicine organizations as the preferred methodology, although poor acceptance and impracticality are cited as impediments.[19] Although there is no absolute criterion for core temperature, most literature cites a core temperature of 40°C for the diagnosis of heat stroke.

The term heat-related illness encompasses any pathologic process caused by an acute increase in core temperature resulting from exertion, passive environmental exposure, or both. Heat exhaustion is at the less extreme end of the spectrum and can present with fatigue, rapid pulse, profuse sweating, vomiting, and weakness,

Table 2
Heat-related illness syndromes

Elevated body temperature at presentation	Heat exhaustion	Systemic symptoms: tachycardia, weakness, nausea/vomiting, profuse diaphoresis	Temperature may be normal in heat exhaustion, although presentation uniformly follows very recent exposure to high temperature
	Heat stroke	Temperature >40°C Central nervous system symptoms	Progresses to multiorgan system failure
		Exertional heat stroke	"Active": found in athletes, military personnel, firefighters
		Classic heat stroke	"Passive": found in elderly, many comorbidities, socially/environmentally limited
Normal body temperature at presentation	Prickly heat	Rash caused by chronic heat exposure	
	Heat edema	Dependent edema in poorly heat-acclimated elderly	
	Heat cramps	Severe leg cramps seen some time after cessation of vigorous exercise	

but without CNS symptoms not attributable to orthostasis. Most cases of heat exhaustion are a mixture of sodium and water depletion and are rarely pure presentations of either.

The heat-related illnesses typically seen in normothermic patients after ongoing low-grade heat exposure are prickly heat, heat edema, and heat cramps. Prickly heat, also called lichen tropicus, is a dermatologic condition presenting as a rash caused by the plugging of sweat ducts with material from the stratum corneum produced by excess sweating. As sweat production continues behind the plugged sweat duct, the resulting pressure causes the duct to rupture, leading to an inflammatory vesicular reaction. With repeated cycles of rupture and replugging of the duct with desquamated material a deeper chronic pruritic dermatitis develops, known as miliaria profunda.[20] Treatment is symptomatic for pruritus, unless secondary infection occurs (*Staphylococcus aureus* is the most common pathogen).

Heat edema is lower extremity–dependent edema seen after heat exposure, attributed to microvascular transudate of fluid with prolonged peripheral vasodilatation. It can also present in the hands. This type of edema is not associated with volume overload, and is commonly seen in elderly patients with relative hypovolemia caused by inadequate replacement of volume losses in hot environments. Heat edema is commonly found immediately following abrupt transition from a cold to a hotter climate. Elevation and compression stockings are the preferred treatment; diuretics have no role.

Heat cramps, most often experienced in the lower extremities, occur after cooling has occurred. These cramps are thought to be caused by hyponatremia, can be quite painful, and can be treated with balanced electrolyte oral solutions. The hyponatremia in heat cramps is associated with volume loss due to sweating, which should be replaced by hypotonic oral solutions rather than balanced electrolyte-containing solutions.

Heat syncope can occur in response to skin vasodilatation, resulting in functional orthostasis after exposure to heat. An elevated core temperature is not required to make the diagnosis. In a young, otherwise healthy patient who experiences only brief syncope clearly associated with orthostasis and returns to completely normal function, the diagnosis is minor heat-related illness. However, if syncope occurs in the same population at risk for CHS, a more aggressive evaluation is warranted.

Regardless of cause, the hyperthermic patient should undergo diagnostic testing proportionate to the severity of presentation. The diagnostic workup should not be guided strictly by meeting clinical definitions of discrete syndromes, but rather by clinical suspicion for morbidity based on the patient's medical history and presentation, and an understanding of the effects of severe heat stress. Laboratory evaluation might include basic chemistries to assess sodium levels and renal function, creatine kinase levels for consideration of rhabdomyolysis, and coagulation parameters for assessment of DIC/SIRS. In the obtunded patient, a chest film, electrocardiogram, computed tomography scan of the head, and, possibly, lumbar puncture are appropriate means to look for other causes of, and direct disorders arising from, elevated temperature.

TREATMENT

Because heat-related illness includes a wide spectrum of severity, the therapeutic options range from simple cooling measures and oral hydration to intensive care services. Young, healthy adults with normal examination results, other than mildly elevated core temperature, may be observed in a cool environment and provided oral hydration with cooled, slightly hypotonic solutions. Solutions with high osmolality slow gastric emptying, delaying transition of cooled fluids to the small intestine and

leading to improvement in core cooling. Most clothing should be removed to expose as much skin surface as possible to the cooler environment; wet clothing, even of light weight, significantly impairs the efficacy of evaporation of sweat in cooling.

The cornerstone of treatment in more severe heat illness is rapid reduction of core temperature and supportive care. Most elements of treatment are empiric, starting with an ABC (airway/breathing/circulation) approach. Obtunded or hemodynamically compromised patients may require endotracheal intubation. Core cooling methods should be started immediately. Almost all patients with a heat illness at any point along the spectrum will be hypovolemic, so volume replacement should occur early in the course of treatment. The preferred solution for volume replacement is normal saline, given the high likelihood of some degree of hyponatremia after sweating profusely. These measures should all be instigated by field personnel, with a focus on rapid cooling. Heled and colleagues[21] proposed that the traditional emergency medicine "golden hour" should be thought of as the "golden half-hour" in the case of heat emergency; morbidity is reduced dramatically if cooling measures, usually in the field, begin within 30 minutes after recognition.

Because studies of cooling methodologies tend to involve small numbers of patients, it is difficult to control for the many variables involved; therefore, most recommendations regarding treatment are experiential, and based on empiric understanding of the pathophysiology of heat stress and the body's response. Based on experience with young healthy athletes with EHS cited in the sports medicine literature, complete immersion of the patient below the neck in cold water seems to be the most efficient cooling method, when appropriate and available.[19] There is no such body of evidence for immersion therapy in CHS patients. The high thermal conductivity of water eliminates the local insulating effect of heated air immediately adjacent to the skin; however, circulating water is presumed to improve cooling via convection. Concerns about peripheral vasoconstriction induced by cold water, leading to decreased efficacy of cooling, as well as shivering induced by cold water, serving to continue the increase in temperature, are outweighed by the therapeutic benefit.[16] Some sports trainers advocate vigorous massage of the extremities to overcome the vasoconstriction effect and promote blood flow to the extremities. If severe shivering causes the patient significant discomfort or impedes resuscitative efforts, benzodiazepines can be used to reduce it. Phenothiazines should be avoided, as should any medication with anticholinergic properties that could reduce sweating. Immersion in ice water (2°–3°C) is more effective than immersion in cold water (10°–20°C) for rapid reduction of core temperature.[18]

Cold-water immersion is often not practical. Equipment used to resuscitate sicker patients (eg, cardiac monitoring leads, intravenous equipment, endotracheal tubes) as well as agitation, poor patient tolerance, and vomiting and diarrhea make immersion in cold water challenging. Most emergency departments do not have the facilities necessary for cold-water immersion. The most common and effective cooling method in the emergency department is a combination of spraying the skin with tepid water and running a fan to augment evaporation and convection. Although this method does not provide cooling at a rate comparable with that of immersion, it is better tolerated, practical, and associated with a low mortality rate.[19]

In theory, when immersion is not available, placing ice as close as possible to the great vessels provides high-volume exposure to the cold. Application of ice packs to the neck, groin, and axilla is another commonly used cooling method. Military units and emergency medical service organizations have used the downdraft from a stationary helicopter to cool overheated personnel and patients.[22]

Administration of cold intravenous fluids is typically not recommended. A recent case study, however, described a severely hyperthermic patient suffering EHS

(multiorgan system failure, including coma, seizures, and DIC parameters) who was placed on the therapeutic hypothermia protocol used for cardiac-arrest patients. The patient made a full recovery, pointing out the need for further study of this approach.[23] Wilson and Crandall[12] suggested consideration of this method, because (1) therapeutic hypothermia has clearly demonstrated efficacy in preserving brain function after insult resulting from cardiac arrest, (2) no research-based lower-limit temperature goal has been established, and (3) a broad body of literature has established the relative safety of hypothermia-inducing methods, including infusion of cold intravenous fluids. Other internal cooling measures, such as cold-water gastric lavage, peritoneal lavage, and rectal or bladder lavage, are poorly studied in humans and can result in water intoxication. Cooling blankets are not effective.

In general, cooling measures are often stopped before the patient becomes normothermic, so as to avoid "overshooting" and causing hypothermia. Dantrolene, an inhibitor of muscle contraction via decrease of calcium released from the sarcoplasmic reticulum, has not been shown to have benefit in hyperthermia.[24]

Hypotension should be treated as distributive shock, caused by shunting of a large proportion of the circulating volume to the periphery via vasodilatation, although hypovolemia must also be considered. Initial permissive hypotension during the cooling phase allows gradual peripheral vasoconstriction and redistribution of the circulating volume centrally. Overly aggressive volume expansion during the initial phases of management can result in pulmonary vascular congestion. Because of this concern, it is reasonable to provide isotonic intravenous fluids in 500-mL aliquots with frequent clinical assessments, including measurements of central venous pressure in critical care management. If vasopressors are needed to manage severe refractory hypotension, agents with predominant α-adrenergic effects, such as norepinephrine, should be avoided because of the theoretical concern about peripheral vasoconstriction leading to decreased core cooling. Use of vasopressor agents is associated with poor outcomes.[12]

Prevention is also particularly important. Acclimatization is an important and highly effective preventive measure, especially for people at risk for EHS, namely those who cannot completely avoid heat stress for occupational or other reasons. Although there is overlap in the physiologic effects of acclimatization and conditioning, good physical conditioning does not confer the full protective effects of acclimatization. Acclimatization requires daily exposure to high temperatures over a 1- to 2-week period. Prolonged exposure causes predictable adaptation of the body via increased plasma volume, onset of sweating at lower temperatures, increased sweat volume with lower electrolyte concentration in the sweat, lower heart rate in response to exercise, and increased stroke volume.[20]

Other preventive measures include ensuring adequate oral hydration, frequent and systematic assessment of those at risk for EHS, and avoidance of strenuous athletic activity during temperature extremes. It is critical to not leave the elderly or young children unattended in vehicles, even for a short time with partially open windows. In response to concerns about the increasing frequency of high-temperature weather emergencies, 12 European countries have set up heat-wave early warning systems.[25] Many of these systems specifically address populations at risk by arranging monitoring of socially isolated and disadvantaged individuals during a heat wave.

SUMMARY

Heat-related illnesses can be avoided or minimized by using proper preventive measures, such as correct evaluation of the environment and acclimatization or, when a

high-temperature environment is unavoidable, reducing activities that create heat stress. Recognition of a primary heat-related illness, while considering the complete differential diagnosis of hyperpyrexia, is key to appropriate treatment. Current research indicates that the most effective means of reducing core temperature is cold-water immersion, when feasible, or evaporative techniques, which are more widely accessible in the setting of an emergency department. Any CNS manifestation of heat-related illness is an ominous finding and indicates higher severity of impact, warranting more aggressive evaluation and treatment.

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