
SHUTTERSTOCK/BLACKWHITEPAILYN

Heat-related illness

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Abstract: This article concisely overviews heat-related illnesses, emphasizing their significant impact on public health. It explores the pathophysiology of conditions ranging from mild heat cramps to life-threatening heat stroke, highlighting key heat transfer mechanisms and the importance of environmental factors. Differential diagnosis considerations, prevention strategies, and nursing implications are discussed, underscoring the need for prompt recognition and intervention in managing these conditions.

Keywords: exercise-induced hyponatremia, heat cramps, heat edema, heat exhaustion, heat-related illnesses, heat stroke, heat syncope, hyperthermia, nursing implications

On average, there are more than 700 deaths from excessive heat exposure per year in the US.[1] Heat illness is a leading cause of mortality and morbidity in US high school athletes.[1] Heat-related illnesses range from mild conditions, such as heat cramps and heat syncope, to more severe heat exhaustion and potentially fatal heat stroke. The human body constantly interacts with the surrounding environment and is perpetually exchanging heat.

Mediation of heat transfer between the body and the environment is critical to maintaining a stable core temperature. There are four processes through which this occurs. Understanding these mechanisms will help to explain the pathophysiology and treatment of heat-related illnesses.

A temperature gradient between the core and the skin surface allows for heat loss when the core temperature is greater than the surface temperature. Heat dissipation is therefore reduced if core temperature and environmental temperature increase. When the ambient temperature exceeds the core temperature, the body gains heat through convection, conduction, and radiation, leaving evaporation as the only means of cooling (see *Heat transfer mechanisms*). Humidity reduces the effectiveness of evaporative cooling, as the skin/air vapor pressure differential is low.[3] As ambient temperature and relative humidity increase, evaporation and convection cooling become less effective.

These cooling mechanisms are critical to maintaining normothermia, which is set by the hypothalamus. Humans and other warm-blooded animals can maintain a consistent body temperature through metabolic activity. The body, therefore, has mechanisms to maintain normothermia throughout a range of environmental temperatures. Examples of the clinical manifestations of these mechanisms include flushing versus mottling due to vasodilation or vasoconstriction of the skin blood vessels, sweating in the heat, and shivering in the cold.

Hyperthermia is an increase above the normal physiologic hypothalamic setpoint and is considered separate from heat illness. Hyperthermia is an expected response to exposure to elevated ambient temperatures or increased metabolic activity. Disease, medications, or other toxidromes may also cause hyperthermia. Core temperatures may elevate during physical activity without causing signs or symptoms. Therefore, a set temperature threshold alone should not be routinely used as an indicator when assessing asymptomatic individuals.[3] Heat illness may occur when core body temperature rises from increased metabolic activity and environmental exposure faster than the body's ability to dissipate heat. Risk factors for heat-related illnesses include lack of heat acclimatization, poor conditioning, high temperatures and humidity, certain chronic diseases and medications, dehydration, and extremes of age.

Early recognition and treatment are a priority, as outcomes are directly related to the magnitude and duration of hyperthermia. If rapid cooling is not promptly initiated for patients with heat stroke, morbidity and mortality can reach 30% to 80%.[3]

Pathophysiology

A rise in core temperature of less than 1 °C (1.8 °F) can lead to an increase in cutaneous blood flow of up to 8 L/min and a sixfold increase in blood flow to glabrous skin surfaces, such as the face, ears, palms, and soles, through sympathetic vasodilation. This shunting of blood to the periphery leads to a decrease in splanchnic and renal perfusion by 30%.[3] The increased demand resulting from peripheral vasodilation leads to increased cardiac output. Increased core temperature induces enzyme denaturation, leading to coagulopathy and cellular apoptosis. An inflammatory response can lead to increased mucosal permeability and cause gastrointestinal toxins to enter the systemic circulation, causing further endothelial injury, loss of thermoregulation, multisystem organ failure, and shock. Permanent central nervous system injury occurs in 20% of cases.[3] Morbidity and mortality are directly related to the magnitude and duration of hyperthermia (see *Heat illness types*).

Differential diagnosis

It is important to consider other potential conditions when assessing a patient with hyperthermia. An elevated core temperature with confusion could also be caused by sepsis, meningitis, encephalitis, drug ingestion, medications, thyroid storm, and toxic ingestions (see *Substances that can contribute to hyperthermia*).[3] Consider the history of the event, the patient's health history, as well as the medications or drugs the patient may be taking.

In a patient who recently underwent surgery, consider malignant hyperthermia. When a medication or drug overdose is suspected, consider serotonin syndrome, anticholinergic toxicity, and sympathomimetic toxicity. For example, if a patient was found to be hyperthermic after emergency medical services (EMS) found them as the driver of a vehicle off of the road, consider what circumstances may have preceded the event. Such a patient may have had a stroke, myocardial infarction, seizure, dysrhythmia, or hypoglycemia that caused them to swerve off the road, and then develop a concomitant heat illness from environmental exposure.

Patients participating in endurance events who drink only water, with no food or electrolyte supplements may develop exercise-associated hyponatremia (EAH). These events are often held in high temperatures, and the signs and

Heat transfer mechanisms[2]

- Conduction: Transfer of heat from warmer to cooler objects in direct contact. Heat loss by conduction increases when in contact with a cold surface, when clothing is wet (5x), and with immersion (25x).
- Convection: Transfer of heat to or from a liquid or gas in motion. Convective heat loss increases with wind speed or water flow.
- Radiation: Transfer of heat in the form of electromagnetic energy. In general, this accounts for the most significant heat loss.
- Evaporation: Change of state from liquid to a gas. Circulation of air over the skin is necessary for evaporation. Causes loss only (no heat gain).

Heat illness types

Condition	Signs and symptoms
Heat stroke	Core temperature over 40 °C (104 °F) and central nervous system dysfunction, including confusion, seizures, and coma
Heat exhaustion	Nausea, dizziness, fatigue, headache, extreme thirst, and myalgia
Heat syncope	A transient loss of consciousness in the setting of heat exposure, with a rapid return to normal baseline function[3]
Heat cramps	Painful involuntary muscle spasms usually occurring in the lower extremities and abdomen associated with dehydration and electrolyte imbalances[3]
Heat edema	A benign and self-limited accumulation of interstitial fluid in dependent extremities related to cutaneous vasodilation and vascular leak[3]
EAH	Mild symptoms of EAH are similar to those of heat exhaustion. These include malaise, fatigue, headache, nausea/vomiting, and lightheadedness.[4] Severe signs mimic exertional heat stroke with altered mental status, delirium, seizures, or coma.[4]

symptoms of EAH and heat illnesses are similar, so these conditions may easily be misidentified.[4] See the section about EAH following the Case 3 discussion.

Prevention

Reviewing weather forecasts is important when planning outdoor activities. The Wet-Bulb Globe Temperature Index (WBGT) measures temperature, humidity, and exposure to solar radiation. The WBGT is used by the military and research groups but is not easily accessible to civilians. A well-known alternative is the Heat Index.[3] Individuals and medical personnel planning outdoor activities should consider these factors, as well as sun exposure, proper water supplies, and appropriate clothing for specific environments.

The general health of individuals participating in outdoor activities should also be considered. Athletes should undergo a preparticipation physical exam.

Proper heat acclimatization helps to reduce the incidence of heat-related illnesses by increasing heat tolerance and heat dissipation.[3] Individuals should acclimatize over 10 to 14 days. Heat acclimatization involves progressively increased intensity and duration of heat-exposed exertion. The benefits are lost within 3 weeks if this acclimatization is not maintained.[5] Hydration status is the most easily altered risk factor. Dehydration leads to higher physiologic strain and reduced ability to sweat. At the same time, overhydration does not decrease the incidence of heat illnesses and may predispose one to EAH.[5] The goal should be euhydration, with a recommendation to drink to thirst.[3]

Case 1

On an afternoon in early September, a high school football team was practicing in full gear. A 17-year-old male player began stumbling and fell to the ground. The team's athletic trainer immediately assessed him and found he was not confused but complained of feeling "dizzy and nauseated." He also mentioned a headache and leg cramps. The athletic trainer moved the player to the shade, removed his helmet and pads, and called 911. The trainer fanned him and gave him a sports drink, but he vomited after only a few sips. EMS arrived, applied ice packs, and obtained peripheral venous access to begin I.V. fluids. He was then transported to the ED for further care.

Substances that can contribute to hyperthermia[3]

Class	Examples
Alcohol	
Amphetamines	amphetamine, dextroamphetamine, lisdexamfetamine, methamphetamine
Anticholinergics	atropine, benztropine, oxybutynin
Antihistamines	diphenhydramine, loratadine, cetirizine
Antipsychotics	haloperidol, chlorpromazine
Benzodiazepines	alprazolam, lorazepam, clonazepam
Beta blockers	atenolol, metoprolol, propranolol
Calcium channel blockers	amlodipine, diltiazem
Cocaine	
Diuretics	torsemide, furosemide, bumetanide, hydrochlorothiazide
Laxatives	lactulose, polyethylene glycol, docusate
Phenothiazines	prochlorperazine, promethazine
Thyroid agonists	levothyroxine
Tricyclic antidepressants	amitriptyline, doxepin

Case 1 discussion

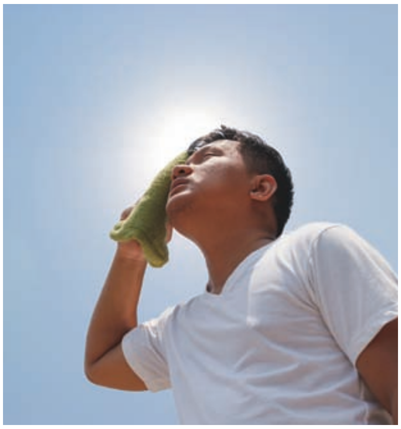
Heat exhaustion results from exposure to high ambient temperature, strenuous exercise, or both. This heat illness manifests as various symptoms, including nausea, dizziness, fatigue, headache, extreme thirst, and myalgia. If left unrecognized and untreated, it can quickly progress to heat stroke.[3]

The mainstays of treatment are cooling and hydration. A person experiencing heat exhaustion should immediately cease physical exertion, move to an area of shade, remove heavy clothing, and be provided oral hydration. 911 should be called.

Evaporative cooling can be performed by dousing the patient with cool water and fanning them.

If in an austere setting with prolonged extrication and delayed EMS arrival, a nurse may treat the patient with conductive cooling. This can be accomplished by carefully immersing the patient in a body of water, preferably with gently moving water. The nurse should sit in the water with the patient resting in their lap to protect the patient's airway.[3]

EMS should assess vital signs, perform a primary and secondary survey, establish I.V. access, and continue evaporative cooling. EMS may also lower the internal temperature of the ambulance by increasing the air conditioning. Ice packs may be applied. A study by Dykstra et al.[6] showed that packs of wetted cubed ice provide the greatest cooling benefit. The effective cooling period of chemical cold packs lasts approximately 5 minutes, while ice packs can effectively cool a patient for more than 25 minutes. When comparing chemical cold packs to ice packs of similar size, ice packs confer a significantly greater temperature change. A study by Lissoway et al.[7] showed that cold packs applied to glabrous skin



Heat exhaustion manifests as various symptoms, including nausea, dizziness, fatigue, headache, extreme thirst, and myalgia.

surfaces (cheeks, palms, and soles) are more effective than the traditional neck, groin, and axillae locations. If cold I.V. fluids are available, they may be used. Cold I.V. fluids may supplement other methods of cooling but, alone, are insufficient.

On arrival to the ED, the care team should obtain a thorough handoff from EMS, including care provided by bystanders. Rectal temperature should be measured promptly. During the physical exam, close attention to the neurologic system is crucial. If neurologic deficits are noted, the patient should instead be considered to have heat stroke. Evaporative cooling should be continued, and ice packs applied as mentioned previously.

There are no specific target temperatures for heat exhaustion treatment, although it is reasonable to follow the evaporative cooling goals of heat stroke treatment. Therefore, evaporative cooling may be discontinued when the core temperature reaches 38 °C (100.4 °F).[3] Core temperature will continue to drop after cooling measures are stopped. Cooling to normothermia may lead to hypothermia and induce shivering. Lab studies may include complete blood cell (CBC) count, basic metabolic panel (BMP), hepatic function panel, urinalysis, creatine kinase (CK), prothrombin time (PT), activated partial thromboplastin time (aPTT), international normalized ratio (INR), and ECG. Additional testing should be tailored to the specific case, evaluating for other possible diagnoses. Patients should be observed in the ED until signs and symptoms improve, and may then be discharged. Patients should be instructed to avoid physical exertion for at least the next 24 hours.[3]

Case 2

It is July in Arizona, and EMS brings a 78-year-old male with altered mental status to the ED. EMS reports that a neighbor found him lying on the floor, unresponsive in his home. The home had no air conditioning, and EMS described the environment as "unbearably hot." EMS providers state that they attempted to initiate cooling with chemical cold packs, obtained I.V. access, and started I.V. fluids. The patient is hot to the touch. He is somnolent, mumbling incoherently when stimulated. He is tachycardic, with a heart rate of 120 beats/minute, respiratory rate of 22, BP of 105/75, and a rectal temperature of 41.5 °C (106.7 °F).

Case 2 discussion

Heat stroke is a medical emergency that requires prompt recognition and treatment. It is characterized by a core temperature over 40 °C (104 °F) and central nervous system dysfunction, including confusion, seizures, and coma.[3] Heat stroke is generally divided into two categories. Classic heat stroke is due to passive exposure to high temperatures. This is typically seen with extremes of age, chronic medical conditions, and those lacking air conditioning or adequate shelter. Exertional heat stroke occurs with strenuous exertion in hot environments.[3]

Similar to heat exhaustion, prompt recognition and treatment by bystanders and EMS are key to treating heat stroke. If heat stroke is suspected, the person should immediately cease all physical activity, be moved to a cool shady area, and remove any heavy clothing. Core temperature may be assessed if a thermometer is readily available; however, clinical assessment alone is enough to initiate treatment. 911 should be called while continuing to assess and treat the individual.

One may douse a patient with heat stroke with water and fan them to perform evaporative cooling. In an austere setting, if near a body of water, conductive cooling can be performed as detailed above in the Case 1 discussion. This can be continued until EMS arrives. More equipment and personnel are often available on the sidelines or nearby medical tents at athletic events.

Conductive cooling may be performed using the tarp-assisted cooling with oscillation method (TACO method).[5] This involves placing the patient on a tarp, holding up the edges, and immersing the patient in a bath of ice water. To provide additional convective cooling, gently rock the tarp back and forth.

In many scenarios, a thermometer may not be readily available; therefore, someone exhibiting neurologic signs and symptoms who has been exerting themselves in a hot environment should be presumed to have a heat stroke.

The distinction between heat exhaustion and heat stroke should not be based solely on the presence or absence of diaphoresis. Patients with heat stroke may be diaphoretic. Basing the diagnosis strictly on this could delay proper treatment and lead to worsened outcomes. Morbidity and mortality are directly related to the magnitude and duration of hyperthermia.

EMS arrival heralds the assessment of vital signs, a complete primary and secondary survey, establishment of I.V. access, and continued cooling efforts. Local protocols largely guide prehospital care by EMS. Although protocols vary, initial treatment focuses on airway, breathing, circulation, and cooling. As the critical treatment for heat stroke is immediate cooling, this will often take precedence over transport to the hospital. Transport may delay cooling due to transport time, triage time, and limited resources in the destination ED. EMS can lower the internal temperature of the ambulance by increasing the air conditioning. Ice packs should also be applied, as mentioned in the discussion of Case 1. Providers could also consider placing large bags of ice over the patient.[8] If cold I.V. fluids are available, they should be used. Cold I.V. fluids may supplement other methods of cooling but, alone, are not considered sufficient.[3] Providers should be prepared for possible complications including seizures, dysrhythmias, and shock.[3]

On arrival to the ED, the care team should obtain a handoff from EMS, including care provided by bystanders. Core temperature should be measured promptly. During the physical exam, particular attention should be paid to performing a thorough neurologic exam. If neurologic deficits are noted, the patient should be considered to have heat stroke. Aim to reach a goal temperature of 39 °C (102.2 °F) within 20 minutes of patient arrival in the ED.[3] Place the patient in a morgue bag with a zipper at the top. Have multiple people working to obtain large amounts of ice and cold water, and fill the morgue bag with enough ice water to immerse the patient and perform conductive cooling. Leave the chest exposed to allow for monitoring leads and evaporative cooling. Administer I.V. fluids for rehydration, opting for cooled fluids, if available. Cooled I.V. fluids do not confer sufficient internal temperature change to serve as a primary means of cooling; instead, they serve as a cooling adjunct. If hypotension persists despite I.V. fluid administration, vasopressors are indicated.[3] Once the goal temperature is achieved, aggressive cooling measures may be discontinued.

The patient's temperature will continue to drop after cooling measures have been stopped. Dropping too low may lead to shivering, which is counterproductive as shivering may raise core temperature.[3] Antipyretics are ineffective due to the physiologic mechanism involved and may be harmful due to hepatic, hematologic, and renal complications. If endotracheal intubation is indicated, the patient should be paralyzed with rocuronium, a non-depolarizing neuromuscular blocker, as prolonged paralysis will prevent shivering, and hyperkalemia is likely to be present.

Avoid depolarizing neuromuscular blockers, like succinylcholine, as this may worsen hyperkalemia, which is common in these patients. Benzodiazepines should be used for sedation and seizure management. Lab work may include CBC, BMP, hepatic function panel, urinalysis, CK, PT/PTT/INR, and ECG. Other testing should be tailored to the specific patient, as needed to assess for other possible causes of hyperthermia.

Patients will require admission to the ICU for further care.

Case 3

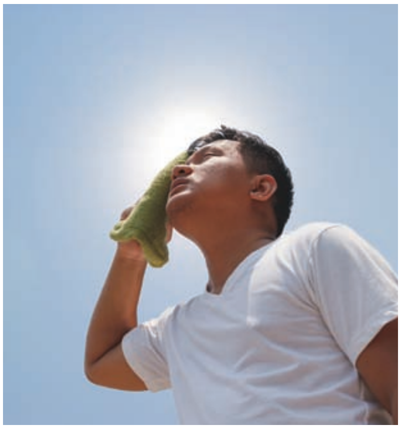
It is a brutally hot August day in Texas, and local healthcare volunteers are providing medical coverage for a marathon. A race worker states over the radio that a runner has collapsed at the halfway point. A group of volunteer healthcare providers hop onto an all-terrain vehicle with their equipment and are transported to the scene. Upon arrival, they find a runner lying on the ground complaining of severe pain in his legs and abdomen. On assessment, the patient is noted to have a rapid pulse and pale, moist skin that is not abnormally hot. A brief history reveals that the patient has not eaten since last night and has been drinking only water throughout the race.

Case 3 discussion

Heat cramps are painful involuntary muscle spasms in the lower extremities and abdomen. They are associated with physical exertion in hot temperatures, stemming from dehydration and electrolyte imbalances. Decreased fitness levels, copious sweating, and poor heat acclimatization all predispose people to heat cramps.[3]

During activity, muscles fatigue and individuals perspire to facilitate evaporative cooling. Oral rehydration with water alone may not replace the electrolyte losses. This combination of electrolyte imbalances, dehydration, and muscle fatigue leads to muscle cramps.[3]

Prehospital treatment of heat cramps focuses on removing the patient from the hot environment, resting, and providing electrolyte oral hydration. Stretching is unlikely to provide much relief.[1] Electrolytes may be provided through commercial sports drinks, soluble rehydration powders, and salty foods.



EAH has become well known in endurance athletics and is the most common medical complication of ultradistance exercise.

Case 3 also suggests EAH, which occurs when the serum sodium concentration is less than 135 mEq/L that occurs during or shortly after prolonged physical activity.[9] EAH has become well known in endurance athletics and is the most common medical complication of ultradistance exercise.[4] This illness can be life-threatening, and clinicians must recognize signs and symptoms and know the treatment. Drinking water or hypotonic fluids in excess is the most common etiology of EAH. The other major factor is the secretion of nonosmotic arginine vasopressin (AVP), which limits renal water excretion and promotes water retention.[4] Stimuli that can cause the release of AVP include pain, emotional stress, physical exercise, vomiting, hypoglycemia, exposure to heat, and nonsteroidal anti-inflammatory drug (NSAID) use.[4] Risk factors that increase the likelihood of developing EAH include poor heat acclimatization, female sex, longer race-completion times, NSAID use, low body mass index, and episodes of nausea (with or without vomiting).[4,10]

Recognition of EAH is essential but can be difficult due to signs and symptoms resembling heat-related illnesses. Mild symptoms of EAH are similar to those of heat exhaustion, such as malaise, fatigue, headache, nausea/vomiting, and lightheadedness.[4] If misdiagnosed as a heat-related illness and treated with a large amount of hypotonic fluids, the athlete can progress to severe signs and symptoms that mimic exertional heat stroke with altered mental status, delirium, seizures, or coma.[4] Sodium point-of-care testing is helpful but not always feasible or available in many situations.

The athlete's fluid, electrolyte, and food intake should be assessed when obtaining a health history. Of note, an athlete should not gain weight during an endurance event. Orthostatic symptoms and thirst indicate heat illness, whereas bloating, weight gain, and lack of thirst suggest EAH.[10] When caring for an athlete with disorientation who is unable to relay history, assessing rectal temperature is essential and will help differentiate between the two etiologies.

Education of participants and staff regarding appropriate hydration is important to reduce the occurrence of EAH. Educating athletes to drink to thirst and limiting the availability of fluids during an event has been shown to reduce the percentage of athletes requiring treatment for hyponatremia from 22% down to 3%.[4] Placing salted snacks at the end of the race and sports drinks could be beneficial.

Case 4

A family has just arrived at an amusement park for a spring break trip. After arriving at the hotel, they immediately visit the park, eager to ride roller coasters and check out the outdoor attractions. In the afternoon, feeling hot and tired, the 46-year-old mother sits down on a sunbaked bench to wait for her family as they ride another coaster. When she sees them exit the ride and walk toward her, she stands up to meet them and immediately appears unsteady. Her family catches her as she loses consciousness and collapses to the ground. By the time EMS arrives, she has regained consciousness and is sitting on the ground talking with her family. During their assessment of the patient, she reports having felt lightheaded and noticing “tunnel vision” immediately upon standing up from the bench, though she feels “back to normal” now.

Case 4 discussion

Heat syncope is a mild form of heat illness. Heat syncope is a transient loss of consciousness during heat exposure with a subsequent rapid return to baseline mental status.[3] It most often occurs with rapid position changes (sitting to standing) and prolonged standing in the heat. Prolonged standing or rapid standing leads to reduced venous return and decreased preload. This, in turn, leads to reduced cardiac output and decreased blood flow. The resulting decreased cerebral blood flow causes syncope. Cerebral blood flow is restored when the patient collapses, and mental status rapidly improves. The patient should rapidly return to baseline mental status within minutes. Risk factors include a lack of heat acclimatization, dehydration, peripheral vasodilation from heat exposure, venous pooling in the lower extremities, advanced age, certain medications, and medical comorbidities such as ischemic heart disease.[3] Consider other potential causes of syncope as well as possible seizures.

Treatment focuses on ceasing physical exertion, rest, and oral rehydration. Elevating the legs while the patient lies supine may help resolve orthostasis symptoms. Patients with heat syncope should avoid further physical exertion for the rest of the day. If syncope is recurrent, symptoms are persistent, or the history and exam are consistent with an alternate diagnosis, call 911 and take the patient to the ED for further evaluation and treatment.

Case 5

It is late morning in August, and a healthy and active 33-year-old male is hiking a popular trail in southwest Virginia. A group of hikers come across him, sitting on a log in the shade. He has removed his socks and shoes and is looking at his hands and feet. The group asks if he is ok, and he reports that he feels fine, other than noticing swelling to his hands and feet. The group sees that he is appropriately dressed for the weather and seems otherwise well. He has an open water bottle and an empty beef jerky bag beside him. He says he has had similar symptoms while hiking and has never had a problem before. The group invited him to join them, and they completed the 7-mile hike together without further issues.

Case 5 discussion

Heat edema is the accumulation of interstitial fluid in dependent extremities, resulting from hydrostatic pressure, vascular leak, and cutaneous vasodilation.[3] This can be treated by elevating the affected extremities. The use of compression stockings may reduce the likelihood and severity of edema. Diuretics are not effective and may lead to dehydration and electrolyte imbalances.[3] The edema should resolve spontaneously within several days.

Nursing implications

Nursing staff must be able to quickly recognize heat illness when evaluating a patient. Nurses should promptly alert the charge nurse and attending physician to expedite care.

Nurses should consider working with colleagues and administrators to develop a heat illness treatment pathway for the facility or organization. A preplanned response for such cases will facilitate the rapid assessment, diagnosis, and treatment of patients with heat-related illnesses. A treatment pathway could include actions such as regular training refreshers with infrequently used equipment, obtaining top-zip morgue bags and large plastic bags to fill with water and ice for rapid cooling, and creating a clinical protocol with descriptions of the location and operation of equipment needed, and steps to be taken following the identification of a heat-related illness.

Special populations

Young children and infants are at particular risk of classic heat stroke as they are unable to affect their environment. Children left in parked cars are at greatest risk of heat stroke, with an average of 37 pediatric deaths each year in the US.[11]

Infants are less able to sweat when hot and therefore have a reduced capacity for heat dissipation. Signs and symptoms of heat illness may be difficult to recognize in infants, with vague signs and symptoms such as restlessness, decreased urine output, tachypnea, irritability, vomiting, and lethargy. Older adults also have a reduced ability to thermoregulate, with sweating thresholds generally elevated in comparison with younger persons. Kuzuya[12] found that those age 65 years or older accounted for 79.3% of heat stroke deaths and that the peak age group for fatal heat stroke is between 75 and 89 years of age.

Conclusion

Heat-related illness occurs when core temperature rises faster than the body can dissipate heat.[3] These illnesses include heat exhaustion, EAH, heat cramps, heat syncope, and heat edema. Special populations, specifically young children, infants, and older adults, are at a higher risk for heat stroke. Expeditious identification and intervention are key to treating heat exhaustion and heat stroke. Rapid cooling initiated by bystanders and EMS is the mainstay of treatment. Nurses must be able to quickly recognize a heat-related illness when evaluating a patient and provide prompt treatment. ■

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