**Chapter 49
Heat-related illness**

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**Introduction**

Heat-related illnesses are a spectrum of disorders more commonly seen when the patient is in a warm environment, has underlying comorbidities, is physically active, and/or has attire which does not readily permit easy removal of body heat (such as firefighter “turnout gear”). If not diagnosed and managed appropriately, significant morbidity and mortality may result. This chapter will discuss the physiology of thermoregulation and the various heat-related illnesses, including management strategies.

According to the Centers for Disease Control and Prevention (CDC), an average of 688 people die annually from heat-related illness [1]. Between 1999 and 2003, an estimated 3,442 deaths were attributed to heat injury. Males accounted for 66% of the deaths. Fifty-three percent of those who died were between the ages of 15 and 64 years, while 40% were greater than 64 years of age.

Emergency medical services protocols should reflect the most up-to-date science on the management of patients with heat-related illness. The best management strategies incorporate expected transport times given the acuity of the patient’s illness, local geography, weather, hospital proximity, and local traffic patterns.

**Physiology of thermoregulation**

Normal oral temperature has been demonstrated to be 33.2–38.2 °C [2]. Rectal temperature averages 34.4–37.8 °C, while the normal tympanic temperature is 35.4–37.8 °C.

The human body generates heat through metabolism. Basal metabolic rate is a measure of the number of calories expended at rest while sedentary. The maintenance of body temperature to a narrow window, or thermoregulation, is a complex task controlled by the hypothalamus. The body’s extremities have a greater variation in temperature, dependent on environmental factors, including clothing. Generally, the extremities tend to be cooler than the rest of the body, while the core temperature fluctuates very little.

While metabolism of the various body tissues generates heat, the majority of body heat comes from skeletal muscle activity. Endocrine function can also affect metabolic rate and increase temperature. For example, epinephrine and norepinephrine can increase basal metabolic rate and subsequent heat generation. Similarly, thyroid hormones can produce an elevation of metabolic rate as well as an increase in body temperature.

Body temperature homeostasis occurs with heat generation in balance with heat dissipation. The narrow homeostatic range prevents enzymatic and cellular dysfunction or injury. Several reflexes or semi-reflexes help to maintain temperature. The reflexes or semi-reflexes activated by cold include shivering, hunger, increased voluntary activity, curling up, decreased heat loss, cutaneous vasoconstriction, epinephrine and norepinephrine release, and erection of the short body airs (i.e. “goose bumps”). The reflexes or semi-reflexes activated by heat include cutaneous vasodilation, sweating, decreased voluntary movement, anorexia, decreased heat production, and increased respiration. The posterior hypothalamus controls the reflexes activated by cold, while those for warmth are located in the anterior hypothalamus. Sweating and cutaneous vasodilation occur with activation of the anterior hypothalamus [3]. Sensors in the spinal cord, skin, deep tissues, hypothalamus, and extrahypothalamic regions of the brain provide feedback to the hypothalamus on body temperature [3]. The hypothalamus is activated by core temperature increases of less than 1 ºC [4]. Lesions of the anterior area of the hypothalamus cause hyperthermia.

Heat is dispersed from the body by several different mechanisms including conduction, convection, evaporation, and radiation. Heat loss by conduction occurs through direct contact with an object or environment that is cooler. Skin temperature greatly affects the amount of body heat lost or gained through conduction. The amount of heat dispersed from the body’s core to the skin is reliant on cutaneous blood flow. When the cutaneous vessels dilate, more blood flows to the skin, allowing for greater heat transfer from the deeper tissues (tissue conductance). Horripilation is the erection of the cutaneous hairs which helps to trap air near the skin and inhibit heat transfer from the skin. Clothing also inhibits tissue conductance by limiting transfer of heat from the skin to the environment. Convection refers to the removal of heat as cooler air passes over exposed skin. The more air passes over the skin (e.g. from a fan), the more heat can be dispersed. Evaporation is the heat lost via converting a liquid to a gas. In the human body, about 600 kcal/hour in ideal conditions can be removed through evaporation [5]. Radiation is the transfer of heat by infrared electromagnetic waves. Approximately 250–300 kcal/hour can be transferred to the human body by solar radiation, with clothing acting as a barrier and providing some protection, estimated at 100 kcal/hour [6]. Additionally, the body disperses heat by radiating to cool objects in the vicinity [3].

At an ambient temperature of 21 °C and while at rest, heat loss from the body occurs through radiation and conduction (70%), evaporation (27%), respiration (2%), and urination and defecation (1%) [3]. As the ambient temperature increases, radiation losses decline and heat loss through evaporation increases [3]. At higher ambient temperatures, evaporation plays a more critical role in body heat removal. The removal of heat is reliant upon the gradients of moisture and temperature. As the environmental temperature and humidity increase, the exchange of heat becomes impaired. Therefore, hot humid environments confer the greatest risk to patients for heat-related illness ([Figure 49.1](https://jigsaw.vitalsource.com/books/9781118990827/epub/OPS/c49.xhtml#c49-fig-0001)).



[**Figure 49.1**](https://jigsaw.vitalsource.com/books/9781118990827/epub/OPS/c49.xhtml#R_c49-fig-0001) NOAA’s National Weather Service Heat Index.

Source: National Weather Service National Oceanic and Atmospheric Administration: [www.nws.noaa.gov/os/heat/images/heatindex.png](http://www.nws.noaa.gov/os/heat/images/heatindex.png)

The body adapts over time to more efficiently manage heat stress, primarily through salt retention and increased fluid secretion from sweat glands to increase the rate of evaporation [6]. Other adaptations include increased circulating plasma volume, improved renal filtration, and increased resistance by the kidney to exertional rhabdomyolysis [7]. Adaptation through production of acute-phase reactants also protects tissues from heat stress [8]. Individual cells produce intracellular heat shock proteins which protect them from sudden heating [9,10]. The mechanism is believed to occur through the binding of heat shock proteins to cellular proteins which inhibit the cellular proteins from denaturing (or unfolding) in hot environments.

**Pathophysiology**

Regardless of the etiology, if hyperthermia is not addressed, tissue and cellular swelling and disruption will occur with widespread hemorrhage. Heat injury causes denaturation of proteins, a severe inflammatory response, and disruption of the coagulation cascade. The denaturation of proteins causes direct injury to the cells and cellular function. Pyrexia above 41.6 °C for a few hours can cause cellular damage. Temperatures above 49 °C can cause nearly immediate cell death. Organs most susceptible to apoptosis secondary to hyperthermia include the mucosa of the small intestine, thymus, lymph nodes, and spleen. Injury caused by inflammatory response is due to the release of several inflammatory cytokines including tumor necrosis factor-alpha, interleukin (IL)-1 (beta), and interferon gamma. Several anti-inflammatory cytokines are also released during heat stress, including IL-6, IL-10, and tumor necrosis factor receptors p55 and p75. Animal models with injection of IL-1 and tumor necrosis factor-alpha have demonstrated changes similar to heat stroke [11]. Heat injury causes activation of the coagulation cascade, injury to the vascular endothelium, and increased permeability of the vasculature. This has been demonstrated through surrogate markers of endothelial damage or activation as seen in heat stroke patients. Some of these markers include von Willebrand factor antigen, endothelin, and intracellular adhesion molecule 1 [11–14].

Minute ventilation, heart rate, and cardiac output increase in response to elevated body temperature, while at the same time, perfusion to the viscera decreases. Medications may impede body heat removal. For example, vasoconstrictors and beta-blockers can prevent the body from transferring warm blood from the core to the skin for dissipation [13,15,16].

Underlying chronic medical conditions can also predispose individuals to heat illness. The elderly are at risk as they often have reduced cardiopulmonary reserve. Additionally, they are more often prescribed medications that put them at risk, tend to be less mobile, and are often volume depleted secondary to a decreased thirst mechanism. Age-related decreases in heat shock proteins, lessening their ability to tolerate increased temperatures, also put the elderly at increased risk [14]. Chronic metabolic conditions (e.g. thyrotoxicosis) predispose individuals to heat-related illness. Obesity increases hyperthermic risk, as adipose tissue impedes cooling, and decreases cardiac reserve. Those who are physically active in hot and humid environments are also at risk. For example, those in the military, athletes (e.g. high school athletes have an increased risk of heat illness over other sports by about 10 fold), and outdoor workers are at increased risk for heat-related illness (e.g. 20-fold increased rate of heat-related death compared to individuals in other employment) [1,17–19].

**Heat edema**

In the continuum of heat-related illness, heat edema is one of the mildest forms. Presenting as edema of the hands and/or feet, heat edema is caused by vasodilation and pooling of interstitial fluid, usually in the dependent extremities [12,17]. Limited to the extremities, the patient’s core temperature is unaffected. Heat edema is most commonly observed in the elderly and those unacclimatized to hot environment.

Heat edema is managed symptomatically. Elevation of the affected extremities and compression stockings are the mainstay of treatment, in addition to moving the patient to a cooler environment [20]. Medication interventions are not recommended. Use of diuretics in these patients should be avoided, unless they present with signs of heart failure, as the edema is from heat stress and not from heart failure.

**Heat syncope**

Heat syncope is constellation of symptoms that can include syncope, dizziness, and orthostatic hypotension. These symptoms are related to venous pooling and peripheral vasodilation [17]. The patients often experience syncope after standing following exertion, or quickly changing their body position during exertion (e.g. standing after completing heavy bench presses). Patients suffering from this ailment usually have an unremarkable core temperature.

The mainstay of treatment for this ailment is to move the patient to a cooler environment and provide intravenous normal saline. Usually an initial infusion of 20 mL/kg is sufficient to alleviate symptoms, with an initial maximum bolus of 1 liter of normal saline.

**Heat tetany**

Heat tetany refers to spontaneous muscle spasm, such as carpopedal spasm or laryngospasm. Often a Chvostek or Trousseau sign may be elicited. This arises from a respiratory alkalosis from hyperventilation due to heat stress [21]. Patients may also complain of circumoral paresthesias. The tetany appears to be related to the rate of PCO2 change more than the absolute pCO2 change [22]. The core temperature in these patients may be either normal or elevated.

Treatment for heat tetany consists of removal from the warm environment. Once the heat stress is removed, the compensatory hyperventilation, subsequent hypocarbia, and associated spasms will resolve. Using a paper bag for rebreathing may also help resolve the symptoms, but does create the risk of hypoxemia [6,23]. Given this risk, the use of a paper bag is generally not recommended. The recommended management strategy to alleviate these symptoms is simply removal from the heat stress.

**Heat cramps**

The cramping of skeletal muscle during or after exertion in a warm environment is called heat cramps. The muscles most commonly involved include the legs (typically the larger muscle groups of the legs such as the quadriceps), calves, abdomen wall, and least commonly the arms. Heat cramps arise from heavy sweating and repletion with hypotonic fluids, causing a dilutional hyponatremia. Core temperature is either normal or may be elevated, but usually not in excess of 40 °C.

Treatment consists of rehydration with oral electrolyte solutions or intravenous normal saline as well as stretching and massage of the affected muscle and rest. Gradual rehydration is preferred, as an aggressive rehydration strategy might worsen hyponatremia. Oral rehydration with electrolyte solutions may be of benefit in preventing additional cramping of the same or other muscles [17,20]. In the past, salt tablets were used for both treatment and prevention. Currently, their use is not recommended during the acute management stage of this disease process [12,13,15,23].

**Heat exhaustion**

Some overlap between heat exhaustion and heat stroke occurs and unlike the previously described milder forms of heat-related illness, these patients display systemic symptoms. Heat exhaustion signs and symptoms may include anorexia, dizziness, fatigue, headache, malaise, nausea, sweating, visual changes, weakness, anxiety, confusion, diaphoresis, fever, hypotension, oliguria, skin flushing, tachycardia, or vomiting.

Typically, the core temperature is elevated, but is usually less than 40 °C. Unlike heat stroke, heat exhaustion patients present with near-normal mental status (although in some cases mild confusion is present which resolves after a short course of treatment). Seizures or coma are not part of the heat exhaustion spectrum.

Treatment consists of cooling. Moving the patient out of the hot environment is important (e.g. moving to a shaded or air-conditioned area). Removal of unneeded clothes will facilitate cooling. Cooling by immersing in cool water, running cold water over the patient, or evaporative cooling are all effective cooling methods. Rehydration is also important, with oral rehydration preferred as long as the patient is conscious, able to safely swallow, and does not have vomiting or diarrhea.

## Heat stroke

The most serious of the heat-related illnesses is heat stroke. While there is some overlap between this entity and heat exhaustion, these patients are hyperthermic with central nervous dysfunction. In population studies, this diagnosis carries up to a 10% mortality risk [24]. A medical emergency, heat stroke may present with anorexia, dizziness, fatigue, headache, malaise, nausea, visual changes, or weakness. More concerning symptoms include anhydrosis, cardiac dysrhythmias, hepatic failure, hyperthermia, neurological signs (e.g. ataxia, coma, confusion, irritability, seizures), pulmonary edema, renal failure, rhabdomyolysis, shock, tachycardia, and tachypnea [25]. Core temperature is often between 40 °C and 44 °C. Temperatures may be higher in some cases. Although anhydrosis is described in the classic case of heat stroke, this is not a reliable sign and patients may be diaphoretic and still have this illness.

When severe, heat stroke can lead to multiorgan dysfunction. Factors associated with high case fatality rates include delay in presentation, delay in initiation of treatment, and increased disease severity upon presentation [25].

Heat stroke is classified in two forms: classic and exertional. Classic heat stroke often occurs during heat waves, is more common in the elderly and debilitated, and typically develops over days, not hours or minutes. The inciting etiology is due to external heat stress. Physical activity, or exertion, is usually not a contributing factor to development of classic heat stroke. Patients are often anhydrotic due to the time period over which this condition develops. Exertional heat stroke often occurs in healthy, young adults who are physically active in hot and humid environments without sufficient acclimatization [26]. Developing secondary to internal heat generation (i.e. body metabolism), it is more commonly seen in athletes, firefighters, foundry workers, and military recruits.

Treatment for heat stroke is similar to that for heat exhaustion. Cooling the patient is critical. Moving the patient from the warm environment to a cooler one is important (e.g. moving to a shaded or air-conditioned area). Removal of excess clothing will help facilitate cooling. Cooling by any of the previously mentioned methods will work (e.g. immersing in cool water, running cold water over the patient, or mist sprayer and fans). Rehydration is also important, with intravenous rehydration preferred given the patient’s neurological changes. Seizures should be managed according to protocol, usually with benzodiazepines.

## Cooling techniques

One of the key elements in treating patients with heat exhaustion and heat stroke is rapid cooling. Decreased mortality and improved outcomes have been observed with rapid cooling to a temperature of 38.3 °C [27–29]. Evaporation combined with convection (e.g. mist spray and a fan) is practical in a clinical setting, commonly employed, and efficient, but does depend on ambient humidity [30]. Ice water immersion is an efficient way of rapidly cooling patients and a metaanalysis supported its use with heat stroke [31]. There are, however, practical challenges when using ice water immersion which include difficulty in monitoring and difficulty in obtaining intravenous access after immersion. There are also several commercial body cooling units available. Ice packs to the groin and axilla may have utility when used in combination with evaporative cooling to lower body temperature [32].

In the prehospital environment, ice water immersion is often impractical due to equipment requirements, difficulty in moving a wet patient, placing defibrillation pads, monitoring the patient, maintaining the airway (i.e. aspiration risk), and obtaining intravenous access. Commercial cooling units are also impractical in the prehospital setting. They require special equipment which does not readily fit into an ambulance, can be cost prohibitive, and adds another piece of equipment with components that have a limited shelf-life.

There are few studies comparing the various methods of rapid cooling. Most have methodological challenges. Given the practical challenges (including expense) with the various cooling methods in the prehospital environment, evaporative cooling with convection may be the best alternative, especially in a patient with altered mental status [31]. In an ambulance, this could entail cool mist spray with air conditioning. Supplementary cooling with ice packs to the groin and axilla may also help expedite cooling. Simply removing the patient from the warm environment to the shaded ambulance will also help reduce the patient’s heat stress.

## Conclusion

The heat-related illness continuum ranges from minor illness with mild discomfort to life-threatening heat stroke. The human body is able to acclimatize to heat stress but once those adaptive strategies have been overwhelmed, illness ensues. Early recognition by EMS providers, in combination with patient treatment protocols that the medical director creates incorporating state, regional, and local practices, will help to reduce patient discomfort, morbidity, and mortality in the spectrum of disease. Management usually involves removal from the heat stress, cooling, and hydration as appropriate.

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