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from frostbite. This vesiculation rapidly progresses to ulceration and liquefaction gangrene. Consider administering amitriptyline early for pain. Patients with milder cases report hyperhidrosis, cold sensitivity, and painful ambulation for many years.

TREATMENT Peripheral Cold Injuries When frostbite accompanies hypothermia, hydration may improve vascular stasis. Frozen tissue should be thawed rapidly and com pletely by immersion in circulating water at 37°–40°C (99°–104°F) for 30–60 min and not by using hot air. Rapid rewarming often produces an initial hyperemia. The early formation of large clear distal blebs is more favorable than that of smaller proximal dark hemorrhagic blebs. A common error is the premature termination of thawing, since the reestablishment of perfusion is intensely pain ful. Parenteral narcotics will be necessary with deep frostbite. If cyanosis persists after rewarming, the tissue compartment pressures should be monitored carefully. Many antithrombotic and vasodilatory treatment regimens have been evaluated. The prostacyclin analogue iloprost given within 48 h after rewarming is an option. There is no conclusive evidence that sympathectomy, steroids, calcium channel blockers, pentoxifylline, or hyperbaric oxygen salvages tissue. Patients who have deep frostbite injuries with the potential for significant morbidity should be considered for intravenous or intraarterial thrombolytic therapy. Angiography, fluorescence microangiography, or pyrophosphate scanning may help evaluate the injury and monitor the progress of tissue plasminogen activator therapy. Heparin is recommended as adjunctive therapy. Intraarte rial thrombolysis may reduce the need for digital and more proxi mal amputations when administered within 24 h of severe injuries. A treatment protocol for frostbite is summarized in Table 477-4. PART 15 Disorders Associated with Environmental Exposures Unless infection develops, any decision regarding debridement or amputation should generally be deferred. Angiography or tech netium-99 bone scan may assist in the determination of surgical margins. Magnetic resonance angiography may also demonstrate the line of demarcation earlier than does clinical demarcation. TABLE 477-4 Treatment for Frostbite BEFORE THAWING DURING THAWING AFTER THAWING Remove from environment. Consider parenteral analgesia and ketorolac. Gently dry and protect part; elevate; place pledgets between toes, if macerated. Prevent partial thawing and refreezing. Administer ibuprofen

(400 mg PO). If clear vesicles are intact, aspirate sterilely; if broken, debride and dress with antibiotic or sterile aloe vera ointment. Stabilize core temperature and treat hypothermia. Immerse

part in 37°–39°C (99°–102.2°F) (thermometer-monitored) circulating water containing an antiseptic soap until distal flush (10–45 min). Leave hemorrhagic vesicles intact to prevent desiccation and infection. Protect frozen part—no friction or massage. Encourage patient to gently move part. Continue ibuprofen

(400–600 mg PO [12 mg/kg

per day] q8 to 12h). Address medical or surgical conditions. If pain is refractory, reduce water temperature to 35°–37°C (95°–99°F) and administer parenteral narcotics. Consider tetanus prophylaxis; elevate part. Administer hydrotherapy at 37°C (99°F). Consider dextran or phenoxybenzamine or, in severe cases, thrombolysis rt-PA (IV or intraarterial). Abbreviation: rt-PA, recombinant tissue plasminogen activator.

The most common symptomatic sequelae reflect neuronal injury and persistently abnormal sympathetic tone, including paresthe sia, thermal misperception, and hyperhidrosis. Delayed findings include nail deformities, cutaneous carcinomas, and epiphyseal damage in children. Management of the chilblain syndrome is usually supportive. With refractory perniosis, alternatives include nifedipine, steroids, and limaprost, a prostaglandin E1 analogue. ■ ■FURTHER READING Dow J et al: Wilderness medical society clinical practice guidelines for the out-of-hospital evaluation and treatment of accidental hypother mia: 2019 update. Wilderness Environ Med 30(4S):S47, 2019. Pasquier M et al: Hypothermia outcome prediction after extracorpo real life support for hypothermic cardiac arrest patients: An external validation of the HOPE score. Resuscitation 139:321, 2019. Pasquier M et al: On-site treatment of avalanche victims: Scoping review and 2023 recommendations of the international commission for mountain emergency medicine (ICAR MedCom). Resuscitation 184:109708, 2023. Snijders B et al: Incidences of underlying causes of hypothermia in older patients in the emergency department: A systemic review. Eur Geriatr Med 14:411, 2023. Takauji S et al: Outcome of extracorporeal membrane oxygenation use in severe accidental hypothermia with cardiac arrest and circulatory instability: A multicentre, prospective, observational study in Japan (ICE-CRASH study). Resuscitation 182:109663, 2023. Teien HK et al: Training videos to prevent cold weather injuries. Int J Circumpolar Health 82:2195137, 2023. Zafren K et al: Induced hypothermia to 4.2°C with neurologically intact survival: A forgotten case series. Wilderness Environ Med 31:367, 2020. Daniel F. Danzl

Heat-Related Illnesses Climate change is globally increasing heat-related morbidity and mor tality. Extreme heat events are more frequent and are occurring in more widespread locations. Heat-related illnesses include a spectrum of disorders ranging from heat syncope, muscle cramps, and heat exhaus tion to medical emergencies such as heatstroke. The core body tem perature is normally maintained within a very narrow range. Although significant levels of hypothermia are tolerated (Chap. 477), multiorgan dysfunction occurs rapidly at temperatures >40.5°C (104.9°C). In con trast to heatstroke, the far more common sign of fever reflects intact thermoregulation. ■ ■THERMOREGULATION Humans are capable of significant heat generation. Strenuous exercise can increase heat generation twentyfold. The heat load from metabolic heat production and environmental heat absorption is balanced by a variety of heat dissipation mechanisms. These central integrative dis sipation pathways are orchestrated by the central thermostat, which is located in the preoptic nucleus of the anterior hypothalamus. Efferent signals sent via the autonomic nervous system trigger cutaneous vaso dilation and diaphoresis to facilitate heat loss.

Normally, the body dissipates heat into the environment via four mechanisms. The evaporation of skin moisture is the single most efficient mechanism of heat loss but becomes progressively ineffective as the relative humidity rises to >70%. The radiation of infrared elec tromagnetic energy directly into the surrounding environment occurs

continuously. (Conversely, radiation is a major source of heat gain in hot climates.) Conduction—the direct transfer of heat to a cooler object—and convection—the loss of heat to air currents—become ineffective when the environmental temperature exceeds the skin temperature. Factors that interfere with the evaporation of diaphoresis signifi cantly increase the risk of heat illness. Examples include dripping of sweat off the skin, constrictive or occlusive clothing, dehydration, and excessive humidity. While air is an effective insulator, the thermal con ductivity of water is 25 times greater than that of air at the same tem perature. The wet-bulb globe temperature (WBGT) is a commonly used index to assess the environmental heat load. The WBGT is superior to the “heat index,” which is only based on air temperature and humidity. The WBGT also incorporates radiant heat and wind speed. The regulation of this heat load is complex and involves the cen tral nervous system (CNS), thermosensors, and thermoregulatory effectors. The central thermostat activates the effectors that produce peripheral vasodilation and sweating. The skin surface is in effect the radiator and the principal location of heat loss, since skin blood flow can increase 25–30 times over the basal rate. This dramatic increase in skin blood flow, coupled with the maintenance of peripheral vasodila tion, efficiently radiates heat. At the same time, there is a compensatory vasoconstriction of the splanchnic and renal beds. Acclimatization to heat reflects a constellation of physiologic adap tations that permit the body to lose heat more efficiently. This process often requires 1 to several weeks of exposure and work in a hot envi ronment. During acclimatization, the thermoregulatory set point is altered, and this alteration affects the onset, volume, and content of diaphoresis. The threshold for the initiation of sweating is lowered, and the amount of sweat increases, with a lowered salt concentration. Sweating rates can be 1–2 L/h in acclimated individuals during heat stress. Plasma volume expansion also occurs and improves cutane ous vascular flow. The heart rate lowers, with a higher stroke volume. After the individual leaves the hot environment, improved tolerance to heat stress dissipates rapidly, the plasma volume decreases, and deacclimatization occurs within weeks. ■ ■PREDISPOSING FACTORS AND

DIFFERENTIAL DIAGNOSIS When there is an excessive heat load, unacclimated individuals can develop a variety of heat-related illnesses. Heat waves exacerbate the mortality rate, particularly among the elderly and among persons lack ing adequate nutrition and access to air-conditioned environments. Secondary vascular events, including cerebrovascular accidents and myocardial infarctions, occur at least 10 times more often in conditions of extreme heat. Exertional heat illness continues to occur when laborers, military personnel, or athletes exercise strenuously in the heat. In addition to the very young and very old, preadolescents and teenagers are at risk since they may use poor judgment when vigorously exercising in high humidity and heat. Other risk factors include obesity, poor condition ing with lack of acclimatization, and mild dehydration. Cardiovascular inefficiency is a common feature of heat illness. Any physiologic or pharmacologic impediment to cutaneous perfusion impairs heat loss. Many patients are unaware of the heat risk associ ated with their medications. Anticholinergic agents impair sweating and blunt the normal cardiovascular response to heat. Phenothiazines and heterocyclic antidepressants also have anticholinergic properties that interfere with the function of the preoptic nucleus of the anterior hypothalamus due to central depletion of dopamine. Calcium channel blockers, beta blockers, and various stimulants also inhibit sweating by reducing peripheral blood flow. To maintain the mean arterial blood pressure, increased cardiac output must be capable of compensating for progressive dehydration. A variety of stimulants and substances of abuse also increase muscle activity and heat production. Careful consideration of the differential diagnosis is important in the evaluation of a patient for a potential heat-related illness. The clinical setting may suggest other etiologies, such as malignant

hyperthermia after general anesthesia. Neuroleptic malignant syn drome can be triggered by certain antipsychotic medications, includ ing selective serotonin reuptake inhibitors. A variety of infectious and endocrine disorders as well as toxicologic or CNS etiologies may mimic heatstroke (Table 478-1).

■ ■MINOR HEAT-EMERGENCY SYNDROMES Heat edema is characterized by mild swelling of the hands, feet, and ankles during the first few days of significant heat exposure. The prin cipal mechanism involves cutaneous vasodilation and pooling of inter stitial fluid in response to heat stress. Heat also increases the secretion of antidiuretic hormone and aldosterone. Systemic causes of edema, including cirrhosis, nephrotic syndrome, and congestive heart failure, can usually be excluded by the history and physical examination. Heat TABLE 478-1 Heat-Related Illness: Predisposing Factors and Differential Diagnosis ILLNESS PREDISPOSING FACTORS Cardiovascular inefficiency Age extremes Beta/calcium channel blockade Congestive heart failure Dehydration Diuresis Obesity Poor physical fitness Central nervous system illness Cerebellar injury Cerebral hemorrhage Hypothalamic cerebrovascular accident Psychiatric disorders Status epilepticus CHAPTER 478 Impaired heat loss Antihistamines Heterocyclic antidepressants Occlusive clothing Skin abnormalities Heat-Related Illnesses Endocrine and immune-related illness Diabetic ketoacidosis Multiple-organ dysfunction syndrome Pheochromocytoma Systemic inflammatory response syndrome Thyroid storm Excessive heat load Environmental conditions Exertion Fever Hypermetabolic state Lack of acclimatization Infectious illness Cerebral abscess Encephalitis Malaria Meningitis Sepsis syndrome Tetanus Typhoid Toxicologic illness Amphetamines Anticholinergic toxidrome Cocaine Dietary supplements Hallucinogens Malignant hyperthermia Neuroleptic malignant syndrome Salicylates Serotonin syndrome Strychnine Sympathomimetics Withdrawal syndromes (ethanol, hypnotics)

edema generally resolves without treatment in several days. Simple leg elevation or compression stockings will usually suffice. Diuretics are not effective and, in fact, predispose to volume depletion and the development of more serious heat-related illnesses.

Prickly heat (miliaria rubra, lichen tropicus) is a maculopapular, pruritic, erythematous rash that commonly occurs in clothed areas. Blockage of the sweat pores by debris from macerated stratum cor neum causes inflammation in the sweat ducts. As the ducts dilate, they rupture and produce superficial vesicles. The predominant symptom is pruritus. In addition to antihistamines, chlorhexidine in a light cream or lotion provides some relief. In adults, localized areas may ben efit from 1% salicylic acid TID, with caution taken to avoid salicylate intoxication. Clothing with breathable fabric should be clean and loose fitting, and activities or environments that induce diaphoresis should be avoided. Heat syncope (exercise-associated collapse) can follow endurance exercise or occur in the elderly. Other common clinical scenarios include prolonged standing while stationary in the heat and sudden standing after prolonged exposure to heat. Heat stress routinely causes relative volume depletion, decreased vasomotor tone, and peripheral vasodilation. The cumulative effect of this decrease in venous return is postural hypotension, especially in nonacclimated elderly individu als. Many of those affected also have comorbidities. Therefore, other cardiovascular, neurologic, and metabolic causes of syncope should be considered. After removal from the heat source, most patients will recover promptly with cooling and rehydration. Hyperventilation tetany occurs in some individuals when exposure to heat stimulates hyperventilation, producing respiratory alkalosis, paresthesia, and carpopedal spasm. Unlike heat cramps, heat tetany causes very little muscle-compartment pain. Treatment includes pro viding reassurance, moving the patient out of the heat, and addressing the hyperventilation. PART 15 Disorders Associated with Environmental Exposures ■ ■HEAT CRAMPS Heat cramps (exercise-associated muscle cramps) are intermittent, painful, and involuntary spasmodic contractions of skeletal muscles. They typically occur in an unacclimated individual who is at rest after vigorous exertion in a humid, hot environment. In contrast, cramps that occur in athletes during exercise last longer, are relieved by stretch ing and massage, and resolve spontaneously. Of note, not all muscle cramps are related to exercise, and the dif ferential diagnosis includes many other disorders. A variety of medica tions, myopathies, endocrine disorders, and sickle cell trait are other possible causes. The typical patient with heat cramps is usually profusely diaphoretic and has been replacing fluid losses with copious water or other hypo tonic fluids. Roofers, firefighters, military personnel, athletes, steel workers, and field workers are commonly affected. Other predisposing factors include insufficient sodium intake before intense activity in the heat and lack of heat acclimatization, resulting in sweat with a high salt concentration. The precise pathogenesis of heat cramps appears to involve a relative deficiency of sodium, potassium, and fluid at the intracellular level. Coupled with copious hypotonic fluid ingestion, large amounts of sodium in the diaphoresis cause hyponatremia and hypochloremia, resulting in muscle cramps due to calcium-dependent muscle relax ation. Total-body depletion of potassium may be observed during the period of heat acclimatization. Rhabdomyolysis is very rare with rou tine exercise-associated muscle cramps. Heat cramps that are not accompanied by significant dehydra tion can be treated with commercially available electrolyte solutions. Although the flavored electrolyte solutions are far more palatable, two 650-mg salt tablets dissolved in 1 quart of water produce a 0.1% saline solution. Individuals should avoid the ingestion of undissolved salt tablets, which are a gastric irritant and may induce vomiting. ■ ■HEAT EXHAUSTION The physiologic hallmarks of heat exhaustion—in contrast to heat stroke—are the maintenance of thermoregulatory control and CNS

function. The core temperature is usually elevated but is generally <40.5°C (<105°F). The two physiologic precipitants are water depletion and sodium depletion, which often occur in combination. Laborers, athletes, and elderly individuals exerting themselves in hot environ ments, without adequate fluid intake, tend to develop water-depletion heat exhaustion. Persons working in the heat frequently consume only two-thirds of their net water loss and are voluntarily dehydrated. In contrast, salt-depletion heat exhaustion occurs more slowly in unaccli mated persons who have been consuming large quantities of hypotonic solutions. Heat exhaustion is usually a diagnosis of exclusion because of the multitude of nonspecific symptoms. If any signs of heatstroke are present, rapid cooling and crystalloid resuscitation should be initi ated immediately during stabilization and evaluation. Mild neurologic and gastrointestinal influenza-like symptoms are common. These symptoms may include headache, vertigo, ataxia, impaired judgment, malaise, dizziness, nausea, and muscle cramps. Orthostatic hypoten sion and sinus tachycardia develop frequently. More significant CNS impairment suggests heatstroke or other infectious, neurologic, or toxicologic diagnoses. Hemoconcentration does not always develop, and rapid infusion of isotonic IV fluids should be guided by frequent electrolyte determina tions and perfusion requirements. Most cases of heat exhaustion reflect mixed sodium and water depletion. Sodium-depletion heat exhaustion is characterized by hyponatremia and hypochloremia. Hepatic ami notransferases are mildly elevated in both types of heat exhaustion. Urinary sodium and chloride concentrations are usually low. Some patients with heat exhaustion develop heatstroke after removal from the heat-stress environment. Aggressive cooling of non responders is indicated until their core temperature is 39°C (102.2°F). Except in mild cases, free water deficits should be replaced slowly over 24–48 h to avoid a decrease of serum osmolality by >2 mOsm/h. The disposition of younger, previously healthy heat-exhaustion patients who have no major laboratory abnormalities may include hospital observation and discharge after IV rehydration. Older patients with comorbidities (including cardiovascular disease) or predisposing factors often require inpatient fluid and electrolyte replacement, moni toring, and reassessment. ■ ■HEATSTROKE The clinical manifestations of heatstroke reflect a total loss of thermo regulatory function. Typical vital-sign abnormalities include tachy pnea, various tachycardias, hypotension, and a widened pulse pressure. Although there is no single specific diagnostic test, the historical and physical triad of exposure to a heat stress, CNS dysfunction, and a core temperature >40.5°C (104.9°C) helps establish the preliminary diagno sis. Some patients with impending heatstroke will initially appear lucid. The definitive diagnosis should be reserved until the other potential causes of hyperthermia are excluded. Many of the usual laboratory abnormalities seen with heatstroke overlap with other conditions. If the patient's mental status does not improve with cooling, toxicologic screening may be indicated, and cranial computed tomography (CT) and spinal fluid analysis can be considered. The premonitory clinical characteristics may be nonspecific and include weakness, dizziness, disorientation, ataxia, and gastrointestinal or psychiatric symptoms. These prodromal symptoms often resemble heat exhaustion. The sudden onset of heatstroke occurs when the main tenance of adequate perfusion requires peripheral vasoconstriction to stabilize the mean arterial blood pressure. As a result, the cutaneous radiation of heat ceases. At this juncture, the core temperature rises dramatically. Since many patients with heatstroke also meet the criteria for systemic inflammatory response syndrome (SIRS) and have a broad differential diagnosis, rapid cooling is essential during the extensive diagnostic evaluation. Heat-induced SIRS reflects the responses of both the innate and the adaptive immune systems (Table 478-1). There are two forms of heatstroke with significantly different mani festations (Table 478-2). Classic (epidemic) heatstroke (CHS) usually occurs during long periods of high ambient temperature and humid ity, as during summer heat waves. Patients with CHS commonly have

TABLE 478-2 Typical Manifestations of Heatstroke CLASSIC EXERTIONAL Older patient Younger patient Predisposing health factors/ medications Healthy condition Epidemiology (heat waves) Sporadic cases Sedentary Exercising Anhidrosis (possible) Diaphoresis (common) Central nervous system dysfunction Myocardial/hepatic injury Oliguria Acute renal failure Coagulopathy (mild) Disseminated intravascular coagulation Mild lactic acidosis Marked lactic acidosis Mild creatine kinase elevation Rhabdomyolysis Normoglycemia/calcemia Hypoglycemia/calcemia Normokalemia Hyperkalemia Normonatremia Hyponatremia chronic diseases that predispose to heat-related illness, and they may have limited access to oral fluids. Heat dissipation mechanisms are overwhelmed by both endogenous heat production and exogenous heat stress. Patients with CHS are often compliant with prescribed medications that can impair tolerance to a heat stress. In many of these dehydrated CHS patients, sweating has ceased and the skin is hot and dry. The duration of core temperature elevation directly impacts mor bidity and mortality. If cooling is delayed, severe hepatic dysfunction, renal failure, disseminated intravascular coagulation, and fulminant multisystem organ failure may occur. Hepatocytes are very heat sensi tive. On presentation, the serum level of aspartate aminotransferase (AST) is routinely elevated. Eventually, levels of both AST and alanine aminotransferase (ALT) often increase to >100 times the normal val ues. Coagulation studies commonly demonstrate decreased platelets, fibrinogen, and prothrombin. Most patients with CHS require cau tious crystalloid resuscitation, electrolyte monitoring, and—in certain refractory cases—consideration of central venous pressure (CVP) measurements and point of care ultrasonography (Chap. 493). Hyper natremia is secondary to dehydration in CHS. Many patients exhibit significant stress leukocytosis, even in the absence of infection. Patients with exertional heatstroke (EHS), in contrast to those with CHS, are often young and previously healthy, and their diagnosis is usually more obvious from the history. Athletes, laborers, and mili tary recruits are common victims. Unlike those with CHS, many EHS patients present profusely diaphoretic despite significant dehydration. As a result of muscular exertion, rhabdomyolysis and acute renal fail ure are more common in EHS. Studies to detect rhabdomyolysis and its complications, including hypocalcemia and hyperphosphatemia, should be considered. Hyponatremia, hypoglycemia, and coagulopa thies are frequent findings. Elevated creatine kinase and lactate dehy drogenase levels also suggest EHS. Oliguria is a common finding. Renal failure can result from direct thermal injury, untreated rhabdomyolysis, or volume depletion. Common urinalysis findings include microscopic hematuria, myoglobinuria, and granular or red cell casts. With both CHS and EHS, heat-related increases in cardiac biomarker levels may be present and reversible. Heatstroke often causes thermal cardiomyopathy. As a result, the CVP may be elevated despite signifi cant dehydration. In addition, the patient often presents with potentially deceptive noncardiogenic pulmonary edema and basilar rales despite being significantly hypovolemic. The electrocardiogram commonly displays a variety of tachyarrhythmias, nonspecific ST-T wave changes, and heat-related ischemia or infarction. Rapid cooling—not the initial administration of antiarrhythmic medications—is essential. Above 42°C (107.6°F), heat can rapidly produce direct cellular injury. Thermosensitive enzymes become nonfunctional, and eventu ally, there is irreversible uncoupling of oxidative phosphorylation. The

production of heat-shock proteins increases, and cytokines mediate a systemic inflammatory response. The vascular endothelium is also damaged, and this injury activates the coagulation cascade. Significant shunting away from the splanchnic circulation produces gastrointesti nal ischemia. Endotoxins further impair normal thermoregulation. As a result, if cooling is delayed, severe hepatic dysfunction, permanent renal failure, disseminated intravascular coagulation, and fulminant multisystem organ failure may occur.

■ ■COOLING STRATEGIES Endotracheal intubation and continuous core-temperature monitor ing should be considered both at the scene and as cooling is initi ated. Peripheral methods to measure temperature are not reliable. Hypoglycemia is a frequent finding. Since peripheral vasoconstriction delays heat dissipation, repeated administration of discrete boluses of isotonic crystalloid for hypotension is preferable to the administration of α-adrenergic agonists. Patients who require inotropic support after cooling and fluid resuscitation may require norepinephrine, followed by epinephrine and dobutamine. Rapid cooling is essential in both CHS and EHS, and an immedi ate improvement in vital signs and mental status may prove valuable for diagnostic purposes. Cool water (15°C [60°F]) is sprayed on the exposed skin while fans direct continuous airflow over the moistened skin. Cold packs applied to the neck, axillae, and groin are useful cool ing adjuncts. If cardiac electrodes will not adhere, they can be applied to the patient's back. Immersion cooling in ice-cold water may be a preferable option with EHS if appropriate medical staff and materials for aggressive cooling are available on scene until emergency medical services arrival. The initial increase in temperature from peripheral vasoconstriction will rapidly be overcome by the large conductive thermal transfer into cold water. This technique presents significant monitoring and resuscitation challenges in many clinical settings. The safety of immersion cooling is best established for young, previously healthy patients with EHS (but not for those with CHS). To improve patient access and monitoring, an alternative is ice water therapy. Ice cold water is continuously poured over the supine patient lying on a porous stretcher. Muscular massage improves vasodilatation. Another option that allows patient access is ice cold immersion in a commercial "cooling body bag." To avoid hypothermic afterdrop (continued cooling), active cooling should be terminated at ~38°–39°C (100.4°F–102.2°F). CHAPTER 478 Heat-Related Illnesses The rate of cooling with commercial cooling blankets is very slow. Other methods are less efficacious and rarely indicated, such as IV infusion of cold fluids and cold irrigation of the bladder or gastrointes tinal tract. Cold thoracic and peritoneal lavage are invasive and rarely necessary. Endovascular cooling also provides effective cooling. ■ ■RESUSCITATION Aspiration commonly occurs in heatstroke, and endotracheal intuba tion is usually necessary. Depolarizing agents should be avoided. The metabolic demands are high, and supplemental oxygenation is essential due to hypoxemia induced by thermal stress and pulmonary dysfunc tion. The oxyhemoglobin dissociation curve is shifted to the right. Pneumonitis, pulmonary infarction, hemorrhage, edema, and acute respiratory distress syndrome occur frequently in heatstroke patients. Seizures are common and can occur during therapeutic cooling. Coldinduced tonic-clonic muscular rigidity mimics seizure activity. Refrac tory seizures may require monitoring with an electroencephalogram. The circulatory fluid requirements, particularly in CHS, may be deceptively modest. Aggressive cooling and modest volume repletion usually elevate the CVP to 12–14 mmHg. The reading, however, may be deceptive. Many patients present with a thermally induced hyperdy namic circulation accompanied by a high cardiac index, low peripheral vascular resistance, and an elevated CVP caused by right-sided heart failure. In contrast, most patients with EHS require far more zealous isotonic crystalloid resuscitation. The hypotension that is initially common among patients with heat stroke results from both dehydration and high-output cardiac failure caused by peripheral vasodilation. Inotropes causing α-adrenergic

stimulation (e.g., norepinephrine) can impede cooling by causing significant vasoconstriction. Vasoactive catecholamines such as dopa mine or dobutamine may be necessary if the cardiac output remains depressed despite an elevated CVP, particularly in patients with a hyperdynamic circulation.

A wide variety of tachyarrhythmias are routinely observed on presentation and usually resolve spontaneously during cooling. The administration of atrial or ventricular antiarrhythmic medications is rarely indicated during cooling. Anticholinergic medications (includ ing atropine) inhibit sweating and should be avoided. With a car diac rhythm that sustains perfusion, electrical cardioversion of the hyperthermic myocardium should be deferred until the myocardium is cooled. Significant shivering, discomfort, or extreme agitation is preferably mitigated with short-acting benzodiazepines or propofol. On the other hand, chlorpromazine may lower the seizure threshold, has anticholinergic properties, and can exacerbate the hypotension or cause neuroleptic malignant syndrome. Coagulopathies more commonly occur after the first day of ill ness. After cooling, the patient should be monitored for disseminated intravascular coagulation, and replacement therapy with fresh-frozen plasma and platelets should be considered. Consider a dose of empiric antibiotics after culturing during cooling if the etiology of the hyper thermia remains unclear. There is no therapeutic role for antipyretics in the control of envi ronmentally induced hyperthermia; these drugs block the actions of pyrogens at hypothalamic receptor sites. Salicylates can further uncouple oxidative phosphorylation in heatstroke and exacerbate coagulopathies. Acetaminophen may further stress hepatic func tion. Dantrolene is ineffective when the temperature elevation is not caused by malignant hyperthermia. Although aminocaproic acid impedes fibrinolysis, it may cause rhabdomyolysis and is not recom mended in heatstroke. PART 15 Disorders Associated with Environmental Exposures ■ ■DISPOSITION Most patients with minor heat-emergency syndromes (including heat edema, heat syncope, and heat cramps) require only stabilization and

treatment with outpatient follow-up. Although there are no decision rules to guide disposition choices in heat exhaustion, many of these patients have multiple predisposing factors and comorbidities that will require prolonged observation or hospital admission. Essentially all patients with actual heatstroke require admission to a monitored setting, and most require intensive care. There are reports of very high survival rates of patients following prehospital immersion cooling without intensive care. Most or all of these patients appear to have had heat exhaustion. Many actual heatstroke patients also require prolonged tracheal intubation, invasive hemodynamic monitoring, and support for various degrees of multiorgan dysfunction syndrome. The prognosis worsens if the initial core temperature exceeds 42°C (107.6°F) or if there was a prolonged period during which the core temperature exceeded this level. Other features of a negative prognosis include acute renal failure, massively elevated liver enzymes, and sig nificant hyperkalemia. As expected, the number of dysfunctional organ systems also correlates directly with mortality risk. ■ ■FURTHER READING Bouchama A et al: Classic and exertional heatstroke. Nat Rev Dis Primers 8:1, 2022. Epstein Y, Yanovich R: Heatstroke. N Engl J Med 380:2449, 2019. Filep E et al: Exertional heat stroke, modality cooling rate, and survival outcomes: a systematic review. Medicina 56:589, 2020. Kaewput W et al: Inpatient burden and mortality of heatstroke in the United States. Int J Clin Pract 75:e13837, 2020. Lipman GS et al: Wilderness Medical Society practice guidelines for the prevention and treatment of heat-related illness: 2019 Update. Wilderness Environ Med 30:S33, 2019. Platt M et al: Heat illness, in Rosen’s Emergency Medicine: Concepts and Clinical Practice, 10th ed. Walls RM et al (eds). Philadelphia, Elsevier, 2023, pp. 1771-1780. Rublee C et al: Evidence-based heatstroke management in the emer gency department. West J Emerg Med 22:186, 2021. Sorensen C, Hess J: Treatment and prevention of heat-related illness. N Engl J Med 387:1404, 2022.

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