Heat Illness In The Emergency Department: Keeping Your Cool

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Abstract

Heat illness spans a broad spectrum of disease, with outcomes ranging from benign rash to fatal heat stroke. Heat illness is broadly divided into 2 types: classic and exertional. Both types occur as a result of exposure to elevated temperature with inadequate thermoregulation; however, classic illness occurs without preceding physical activity. Treatment consists of rapid cooling, fluid replacement, and physiologic support. Other milder forms of heat illness include heat fatigue, heat syncope, heat edema, and heat rash. Drugs, drug combinations, drug side effects, and infections can also cause or complicate heat illness and these manifestations may not respond to standard cooling maneuvers and treatments alone; each requires specific additional therapy or antidotes to reverse the cycle of heat and organ damage. This review examines the physiology, diagnosis, and treatment of exertional, classic, and drug-induced hyperthermia. Field and prehospital diagnosis and treatment are also reviewed, with recommendations for rehydration and monitoring in rhabdomyolysis.

Case Presentations

On a late summer afternoon shift, a 16-year-old adolescent boy presents to the ED via EMS after collapsing during football practice. The medics state that he complained of dizziness and staggered to the edge of the field before he fell to the ground. The medics call out vital signs: blood pressure, 102/60 mm Hg; heart rate, 120 beats/min; and respiratory rate, 16 breaths/min. They did not measure a temperature, but state that he feels hot to the touch. The patient is brought to the resuscitation room where you begin your evaluation. The patient is awake, but very lethargic and unresponsive to verbal commands. You notice he is flushed, his skin is hot to the touch, and he is sweating. The rectal temperature is 40.3°C. You begin volume resuscitation with normal saline, remove his athletic equipment and clothing, and order laboratory studies. As the nurse tells you that the patient is no longer responding to verbal or noxious stimuli, you wonder if you should intubate him. You consider the quickest and most effective way to cool the patient with the resources available in your department. As you assess what other treatments the patient may need during his resuscitation, the nurse tells you there is a new patient in the next room.

As you walk into the next room, you see a 35-year-old woman with her husband at her bedside. He says his wife was treated for nausea and vomiting at an urgent care center the previous day. When she woke up today, she was confused, sweating, and her arms and legs seemed "stiff." As you begin your evaluation, you ask the ED clerk to obtain the records from the outside facility. On examination, the patient is febrile at 39.4°C, diaphoretic, and disoriented. Her heart rate is 133 beats/min and her blood pressure is 170/110 mm Hg. On motor examination, she exhibits cogwheel rigidity. You place an IV and administer a benzodiazepine, which does little to alleviate her symptoms. You ask yourself: What next?

Introduction

Heat-related illnesses exist along a continuum from the less severe symptoms of heat edema to the potentially fatal syndrome of heat stroke. **(See Table 1.)** Heat-related illnesses are classically defined in review literature by exposure without alteration of hypothalamic thermoregulation.[1,2] In contrast, fever is defined by changes to the hypothalamic set point by pyrogenic cytokines.[1] Heat stroke is a life-threatening condition and requires the highest level of clinical attention. The cardinal sign of heat stroke is a change in mental status and it is associated with a significant mortality. Delirium, convulsions (particularly during cooling), multisystem organ failure, coma, and death can occur. Any substance or situation that alters thermoregulatory function can precipitate severe, life-threatening disease. As there are no tests to confirm the diagnosis of heat stroke, other conditions presenting in a similar way

should be considered and investigated. A list of differential diagnoses is provided in **Table 2, page 3**. Heat-related illnesses occur when the body's cooling mechanisms are unable to control the natural rise in body temperature caused by metabolism, physical activity, or exposure to warm temperature. The true prevalence is difficult to estimate, as many cases go unreported. Heat-related illnesses can be multifactorial, but are most commonly seen in improperly conditioned patients participating in physical activities in environments with hot, humid weather (exertional), and in patients unable to escape a heated environment (classic). The challenge for the emergency clinician is twofold: first, the severity of heat illness must be established and prompt cooling therapy must be started. Second, the emergency clinician must be aware of toxicological substances, infectious processes, and other medical conditions that can cause symptoms that mimic heat illness.

The majority of heat illness cases are benign and easily reversed. After evaluation, diagnosis,

Table 1. Types Of Heat Illnesses[2-9]

Heat edema	Benign, self-limiting condition, usually seen in the lower extremities after heat exposure; secondary to prolonged cutaneous vasodilation.
Heat rash	A dermatologic condition caused by plugging of sweat ducts and the ensuing inflammatory eruption that follows; also known as lichen tropics, prickly heat, miliaria rubra.
Heat cramps	Exercise-associated muscle contractions believed to be secondary to loss of electrolytes (such as sodium), which occurs mainly after cooling.
Heat tetany	Carpopedal spasm and paresthesia secondary to hyperventilation.
Heat syncope	Transient loss of consciousness or collapse during heat exposure followed by a relatively rapid return to baseline; elevated core temperature is not necessary to make the diagnosis.
Heat exhaustion	An illness of mild to moderate severity secondary to loss of salt and water during exposure to heat. Symptoms are wide-ranging and may include fatigue, rapid pulse, profuse sweating, vomiting, and weakness without central nervous system involvement. Core temperature may be low, normal, or high (but < 40°C [104°F]) .
Heat stroke	Core temperature ≥ 40°C (104°F) with central nervous system dysfunction/encephalopathy. Classic: Passive hyperthermia secondary to impaired mechanisms for heat dissipation or inability to escape heated environment (generally develops slowly, over days). Exertional: Hyperthermia associated with participation in sport or occupation leading to heat exposure (develops relatively rapidly).

and cooling treatment, the vast majority of patients presenting with mild to moderate disease can be discharged from the emergency department (ED). Important exceptions include heat stroke and lesser forms of heat illness occurring in patients who are elderly or debilitated or who have multiple comorbidities. Such conditions may result in major physiologic stress in situations of passive ambient heat exposure. This issue of *Emergency Medicine Practice* reviews the types of heat illness, their diagnosis, and their management. Knowing how to rapidly cool the patient, what sequelae to anticipate, and which processes mimic heat stroke can prevent mortality.

Critical Appraisal Of The Literature

A systematic search of the literature through May 2014 was undertaken using PubMed and the Cochrane Database of Systematic Reviews. The search was performed using the terms *heat illness, heat stroke, neuroleptic malignant syndrome, serotonin syndrome, aspirin overdose, sympathomimetic overdose, rhabdomyolysis,* and *lithium toxicity.* Limitations of the studies included poor study design, small sample sizes, and lack of appropriate controls in those studies examining treatment.

Epidemiology

The data that exist on the epidemiology of nonexertional and exertional heat illness are imprecise,

Table 2. Differential Diagnosis In Hyperthermia[1,2,4-6]

Origin/Cause	Diagnosis
Endocrine	• Pheochromocytoma • Thyroid storm
Infectious (including central nervous system)	• Brain abscess • Encephalitis • Malaria • Meningitis • Sepsis • Tetanus • Typhoid fever
Neurologic	• Cerebrovascular accident • Status epilepticus/seizures
Toxicological	• Alcohol withdrawal (delirium tremens) • Anticholinergic toxidromes • Aspirin overdose • Complex drug interactions (PCP, heroin, MDMA, cocaine, amphetamines) • Malignant hyperthermia • MAO inhibitors • Neuroleptic malignant syndrome • Serotonin syndrome

Abbreviations: MAO, monoamine oxidase; MDMA, 3,4-methylene-dioxymethamphetamine; PCP, phencyclidine

with inconsistent definitions of important terms. However, there are identifiable trends across all sources that are helpful in interpreting meaning. Nonexertional heat-related illness is noted most frequently during heat waves with high peak temperatures and extended exposure.[10-12] Risk factors for heat illness may include low socioeconomic status, inability to leave home, mental illness and other comorbidities, unavailable or nonfunctioning air conditioning systems, and age > 65 years.[10-19] Over the past several decades, many cities and countries have achieved significant success in the implementation of prevention and education plans aimed at reducing the occurrence of heat-wave–related heat illness.[11,14,20]

Fewer epidemiological data are available with regard to exertional heat illness, but the studies that have been conducted present consistent risk factors such as young age and participation in sport or exercise in hot environments. Nelson et al performed a retrospective analysis of exertional heat illness in the United States from 1997 to 2006. They identified 54,983 patients treated in EDs.[21] Patients aged ≤ 19 years represent the largest portion of these cases (47.6%) and the majority of these were related to sport or exercise. Fortunately, 90.4% were successfully treated and discharged from the ED. The remainder required hospital admission; no deaths were reported.[21] Kerr et al also addressed heat illness in the athlete, noting that football players were 11 times more likely to develop exertional heat illness than players of all other sports combined.[22] Rav-Acha et al reviewed 34 cases of exertional heat illness and reported that delayed recognition and treatment contributed to 6 fatalities; 4 of the 6 decedents died within 1 hour of collapse.[23]

Physiology

The widely accepted definition of *thermoregulation* is the physiologic response to internal and external thermal stimuli. It is the process by which the human body regulates heat production and dissipation to maintain core temperatures that are consistent with life. Hyperthermia occurs when the natural thermoregulatory processes are overwhelmed or are no longer functional.

Mechanisms Of Heat Transfer

There are 4 mechanisms by which the human body regulates temperature: radiation, convection, conduction, and evaporation. **(See Table 3, page 4.)** Radiation is heat transfer that occurs through emission of particles or waves. It plays a relatively minor role in transferring heat away from the body. Convection is heat transfer by a moving liquid or gas, such as occurs from flowing blood to surrounding tissues. Conduction is heat transfer through direct contact

between surfaces and is, therefore, surface-area dependent. In the ED, conduction can be a major mechanism of heat transfer in treating the hyperthermic patient. Lastly, evaporation, the change of a liquid to a gas, is the primary method by which the human body cools itself. It occurs naturally through sweating or therapeutically by misting the skin with water. Evaporative heat transfer becomes less effective with rising ambient humidity.[24]

Two-thirds of the human body is composed of water; for the average 70 kg body, this represents approximately 50 L. Evaporative loss of water depends on a number of physiologic and environmental factors, and is only possible if humidity is low enough to allow sweat production and permit evaporation.[25] Evaporation is dependent on wind velocity and vapor pressure between the water on the skin and the atmosphere. To maintain thermal equilibrium, the body increases the rate of sweat output to compensate for increases in body temperature.[24] When sweat is produced more rapidly than it can evaporate, it becomes less effective as a means of cooling.[24] Sweating rates are also dependent on the density of activated sweat glands in a given region of the body and the sweat output per gland, both of which differ depending on the part of the body.[26] Ultimately, if water cannot evaporate or if water is not replaced, core temperature will rise and hyperthermia may ensue.

Thirst has not been shown in the heat-illness literature to be an effective mechanism for monitoring fluid balance. For this reason, hydration regimens that encourage fluid and electrolyte intake beyond the normal thirst sensation are widely accepted as effective in preventing heat illness.[18,27-30] However, no large randomized controlled studies support this practice.

Thermoregulation

Thermoregulation in humans occurs by both voluntary and involuntary mechanisms. Voluntary responses to heat arise in the cerebral cortex and include seeking shade, resting, drinking water, and cooling oneself. Involuntary responses to heat are regulated by the hypothalamus and involve complex cutaneous, cardiovascular, and neurologic systems.[31-33]

The preoptic anterior hypothalamus is the area of the brain where temperature sensation and regulation intertwine. It receives input from the somatosensory thermoreceptors in the skin and spinal cord and then regulates changes by altering skin blood flow, cutaneous vasodilation, and blood pressure through the baroreceptor reflex.[45,31,33-36] During exercise, competing reflexes in this system will activate both vasoconstriction (in response to physical activity) and vasodilation (in response to the heat produced).[33,36] This results in an increase in skin blood flow and, thus, heat dissipation through the surface of the skin. Skin blood flow can increase to 8 L/min during exercise and is essential to heat transfer through increased convection and evaporation.[34,35,37] However, a cardiac output of 20 L/min is needed to maintain skin blood flow at that rate, so any impairment of this system through cardiovascular disease, dehydration, electrolyte abnormalities, medications, infection, or age may alter the evaporative cooling mechanism.[5,34,35,38,39] In hyperthermia, the peripheral mechanisms that help the body maintain temperature at its normal set point are lost.

Prevention And Acclimatization

Prevention and acclimatization are important in decreasing heat illness morbidity and mortality, as adverse effects appear more likely when behavioral responses are restricted. Although good physical conditioning through training helps decrease the risk of heat illness, it does not entirely prevent it.[18,27,40,41] Assessment of the thermal environment should include factors such as air temperature, humidity, situational environment, clothing/armor, and the diversity of activity being performed. The most commonly used assessment of environmental heat safety is the wet bulb globe temperature (WBGT) index. Developed by the United States military in the 1950s, it was originally created to assess a soldier's risk for developing heat-related illness and guide the work/rest and hydration practices of military personnel. Briefly, it is an equation that takes into account heat (dry bulb temperature), humidity (wet bulb temperature), and ambient temperature, calculated to include wind and reflected heat (black

Table 3. Cooling Mechanisms And Methods

Cooling Modality	Mechanism	Dissipation/Absorption	Cooling Method
Conduction	Direct contact between surfaces of divergent temperatures	Both minimal	Cooling blankets
Convection	Transfer to moving liquid or ambient air	Both moderate	Mist/fan, water immersion
Radiation	Transfer via electromagnetic waves	Both minimal	Not applicable
Evaporation	Change of state from liquid to vapor or water	Dissipation only	Sweating, fan, misting

Adapted from *Emergency Medicine Clinics of North America*, Vol. 31, Edition 4, Walter F. Atha, Heat-Related Illness, Pages 1097-1108, Copyright 2013, with permission from Elsevier.

globe temperature).[27,40,42] Currently, both military and civilian groups (including the American College of Sports Medicine), use the WBGT index to reduce the risk of heat illness and injury. A comparison of the military and American College of Sports Medicine guidelines is found in **Table 4**.

The temperature at which the WBGT index reflects individual guidelines can be widely variable. This may be secondary to different measurement techniques used by each group. However, there are additional limitations to using the WBGT index as a predictor of heat illness: calibration techniques, WBGT formula variation, nonstandard instrumentation, lack of consideration of clothing type/armor, and other oversights.[40-43] Because of these variations, several researchers have attempted to develop mathematical models to estimate WBGT but there is no consensus regarding their use.[44,45]

Acclimatization has been described as daily exposure to high temperatures over a 1- to 2-week period.[2,3,46] After several days of a daily regimen of physical exertion in high temperatures, the body is said to adapt by increasing plasma volume, sweating at lower temperatures, increasing sweat volume, lowering the electrolyte concentration of sweat, lowering heart rate with exertion, and increasing stroke volume.[47,48] There is not a large body of literature to support this definition, although it is accepted in clinical practice.

Mass Gatherings, Field Care, And Prehospital Care

Heat illness considerations play an important part in planning and managing mass-gathering events. Large retrospective studies of general population cohorts and of mass-gathering cohorts demonstrate that, as ambient temperature rises, the incidence of heat illness increases proportionately. Heat illness cases are rare in noncompetitors and civilians at temperatures of 60°F (15.5°C) or below.[49-51]

A variety of prediction tools exist, including the heat index and the more comprehensive WBGT (which takes into account wind velocity and radiant heat).[40,42] The WBGT is used extensively in the United States military, but there are no guidelines as to when to cancel an event. The medical director should consider recommending cancellation or modification of a mass event if the expected heat will be too high or if the resources to prevent heat illness appear insufficient.[42]

Measurement Of Patient Core Temperature In The Field

Ideally, an accurate reading of a patient's core temperature should obtained, but direct core body temperature measurements are often impractical. Core temperatures are most often obtained through rectal temperature measurement, but esophageal or bladder temperature reading may also be obtained. Methods of external measurement include oral, axillary, skin, and tympanic readings. However, these modalities are inaccurate and cannot be used reliably as an indicator of core temperature because skin cooling occurs more rapidly than core cooling.[52,53] Pryor et al examined 4 methods of external temperature measurement (temporal, tympanic, forehead, and skin) in firefighters who engaged in physical activity while wearing thermal protective clothing. They compared each to the core temperature measured rectally and found that external temperature measurement failed to accurately predict core temperature.[53] Although the Pryor et al study referenced a very specific population, the results are supported by other articles and can be extrapolated to patients with heat illness.[54-57]

While external measurements are not ideal, emergency medical service (EMS) and prehospital providers should use what is available and initiate treatment in a timely fashion. If external temperature measurement is all that is available, the temperature trends can be used as a precursor to core measurement. The recommendation to "cool first, transport

Table 4. Wet Bulb Globe Temperature Categories And Recommended Activity Levels

WBGT Category	ACSM Guideline	US Military Guideline	Risk Level And Recommendations
1 (white flag)	< 50°F (10°C)	78°F-81.9°F (25.6°C-27.7°C)	Very low risk. Use caution and preventive measures during physical activity.
2 (green flag)	< 65°F (18.3°C)	82°F-84.9°F (27.8°C-29.4°C)	Low risk. Use caution and preventive measures during physical activity.
3 (yellow flag)	65°-73°F (18.3°-22.8°C)	85°F-87.9°F (29.4°C-31.1°C)	Moderate risk. Curtail strenuous outdoor activity for all persons not acclimatized.
4 (red flag)	73°-82°F (22.8°-27.8°C)	88°F-89.9°F (31.1°C-32.2°C)	High risk. Use extreme caution and preventive measures during physical activity.
5 (black flag)	> 82°F (27.8°C)	> 90°F (32.2°C)	Extreme risk. Restrict physical activity to air-conditioned or climate-controlled environments.

Abbreviations: ACSM, American College of Sports Medicine; WBGT, wet bulb globe temperature.

second" is based on the fact that timely treatment is essential in decreasing mortality, and it underscores the goal of treating patients within the "golden hour."[52,58,59] This approach prioritizes appropriate cooling measures in the first 30 to 60 minutes after recognition, when interventions are most likely to improve survival. The exact initial temperature is thought to be less important than the time to reduce the core temperature to < 40°C. Military and sports literature has shown that when core temperature is decreased to < 40°C within 30 minutes of recognition, the mortality rate approaches zero.[52,58]

Field And Prehospital Treatment

The principles and modalities of prehospital cooling are similar to inhospital treatment. After initial prehospital triage and intravenous access are obtained, cooling and hydration should begin. The patient with significantly elevated temperatures and central nervous system abnormalities should be placed in a cold water immersion bath (when available), as such patients are likely suffering from heat stroke. If this is not available, misting the patient with tepid water, combined with fanning, promotes rapid evaporative and convection cooling. Additionally, ice packs to the neck, groin, and axillae, or near great vessels, provide the highest-volume exposure to cooling.[3,52,60]

It is also essential to start hydration judiciously. The main therapeutic effect of intravenous fluids is to re-establish circulating volume and to promote sweating. Fluids can be given orally or intravenously in the prehospital setting; clinical condition will likely dictate the route. Patients suffering from moderate to severe exertional heat illness are more likely to present with concurrent hypovolemia and dehydration, although this may not always be the case. (See the Treatment section, page 8).

Sporting events, especially endurance events, make for special prehospital circumstances as the athletes are usually prepared for heat illness and have attempted to replace their fluid losses. This can result in hyponatremia if the patient has attempted to rehydrate with very hypotonic fluids. The 2007 Statement of the Second International Exercise-Associated Hyponatremia Consensus Development Conference recommends withholding hypotonic or isotonic fluid administration in any patient who appears to have symptomatic hyponatremia (confusion, seizures, weakness, nausea, or vomiting) until a blood sodium level can be obtained.[61] Many endurance events now supply point-of-care devices to check serum sodium. A prospective study of 766 runners participating in the Boston Marathon (2002) looked at blood samples from 488 runners who met specific physical examination and history criteria on a survey.[62] Of this group, 13% had sodium values < 135 mEq/L, while 0.6% had sodium values < 120

mEq/L and were defined as critical.

Patients with moderate symptoms (headache, nausea, or vomiting) should only be treated with hypertonic sodium if serum or plasma levels have been identified as critical (< 120 mEq/L). Patients with severe symptoms (seizure or confusion) should receive hypertonic saline in the field and be immediately transported to a hospital. Recommendations for hypertonic saline treatment, where indicated in moderate and severe hyperthermia, are: up to 3 doses of a 100-mL bolus of 3% saline given 10 minutes apart, followed by transport to a hospital.[61] While the withholding of intravenous fluids may seem counterintuitive, it is important to remember that intravenous fluids in the field are not as important or efficacious for rapid core temperature reduction as are active cooling maneuvers such as cold water immersion, misting and fanning, or ice packs. In cases where prehospital sodium levels are unknown, presenting symptoms should guide treatment.

Emergency Department Evaluation

History

Patients presenting to the ED with hyperthermia pose a diagnostic challenge because of the broad array of conditions associated with elevated core temperature. **(See Table 2, page 3.)** It is important to obtain information related to environmental heat exposure as well as the patient's activities prior to the onset of symptoms. History of exercise, recreational activity, and outdoor work may help to narrow the differential.[6,63] Because multiple drug classes are associated with hyperthermic toxidromes, it is vital to get an accurate account of the patient's use of medication or illicit substances as part of the initial assessment. **(See Table 5.)** Drugs can also exacerbate heat-related illness associated with ambient heat exposure. Evaluation of elderly, debilitated, and nonambulatory patients should include an assess-

Table 5. Drugs Associated With Hyperthermia

Prescription Drugs	Nonprescription Drugs
• Anticholinergics	• Amphetamines
• Antihistamines	• MDPV ("bath salts")
• Beta blockers	• Cocaine
• Calcium-channel blockers	• Ecstasy
• Diuretics	• Jimson weed
• Laxatives	• Mushrooms
• Lithium	• Phencyclidine
• Neuroleptics	
• Phenothiazines	
• Salicylates	
• Serotonergics	
• Tricyclic antidepressants	

Abbreviation: MDPV, 3,4-methylenedioxypyrovalerone.

ment of their living conditions and, specifically, their access to air conditioning.

To institute proper treatment, it is important to recognize other manifestations of heat-related illnesses in the ED:

- Patients with new-onset lower extremity edema should be questioned about heat exposure, living conditions, and medication use.
- Prickly heat or heat rash should be considered in patients who present with a burning rash during times of high ambient temperatures and humidity. This should especially be considered in children.
- Patients evaluated for muscle cramps and carpopedal spasm should be asked about activity prior to arrival, as these conditions may also result from heat exposure.
- Patients presenting with syncope should be evaluated for cardiac dysfunction, symptoms of stroke, medication mismanagement, glucose imbalance, and heat illness.
- In patients with altered mental status, use of illegal substances and exposure to toxins should be explored.

Physical Examination

Because heat stroke can compromise both the respiratory and the circulatory systems, evaluation of the airway, respiratory rate, and circulation is crucial.[5,9,64] Patients should be undressed and placed on a monitor, and core temperature should be obtained. As discussed previously, a rectal measurement provides the most accurate temperature.[52,53,65]

Central Nervous System

Physical examination findings suggestive of central nervous system involvement and, therefore, heat stroke, include irritability, confusion, bizarre behavior, ataxia, coma, and seizure activity. Additional findings (such as hyperreflexia) may suggest other diagnoses, such as lithium toxicity.[66] Heat exhaustion presents with milder symptoms (such as dizziness, weakness, malaise, and headache).[3]

Cardiovascular System

Healthy adult patients with heat illness may present with sinus tachycardia.[67] However, medications and medical devices may hinder this response, causing a normal heart rate. Hypotension is also common and is usually associated with dehydration.

Emergency clinicians should be aware of other etiologies of hyperthermia associated with cardiac abnormalities. Anticholinergic toxicity, serotonin syndrome, and neuroleptic malignant syndrome may also cause tachycardia. Tachycardia with hypertension is common in serotonin syndrome.[68]

Lungs

Physical examination of the lungs is likely to be unremarkable, although tachypnea and hypoxia may be seen, especially if the patient develops pulmonary edema from aggressive fluid resuscitation. Rales may be heard on physical examination in this case.

Abdomen

The abdominal examination will likely be nonspecific. A urinary catheter should be placed in patients with heat stroke to monitor urine output.[69] Urine output may be decreased or normal, depending on the severity of disease and end-organ involvement. In cases of hyperthermia caused by anticholinergic toxicity, urinary retention may be observed.[68]

Skin

Findings upon examination of the skin can vary. Most patients present with warm, flushed skin, which may be wet or dry. Generally, exertional heat stroke is associated with wet skin, while classic heat stroke is associated with anhidrosis, although this is not always the case.[4] Evidence of pruritus or erythematous papules and vesicles may indicate miliaria or heat rash.

Diagnostic Studies

Heat stroke is a clinical diagnosis, and there is no single diagnostic test to identify it. Nonetheless, diagnostic studies are essential in ruling out other conditions with a presentation similar to heat stroke and with differing management. **(See Table 2, page 3.)**

A full set of vital signs should be obtained on all patients, and a rapid blood glucose test and 12-lead electrocardiogram (ECG) obtained. A complete blood count (CBC) and complete metabolic panel with liver enzymes, lactate, urinalysis, and creatine phosphokinase (CPK) level should also be obtained in all patients with suspected heat stroke (both exertional and classic). These tests should be considered in patients with milder forms of heat illness as well. If appropriate, testing for cardiac enzymes, arterial blood gas, and thyroid-stimulating hormone levels, as well as a toxicological screen, should also be considered.

A chest x-ray should be obtained to evaluate for pneumonia and pulmonary edema, the latter of which is associated with heat stroke.[2]

The emergency clinician should consider a head computed tomography (CT) if there are any neurologic deficits, or if there is evidence or history of trauma. A CT may also be considered in patients who do not return to baseline with appropriate treatment. A lumbar puncture should be considered in hyperthermic patients when central nervous system infection is suspected.

Treatment

The urgency and level of treatment in heat illness depends on the severity of the disease. It is therefore crucial to determine how impaired the hyperthermic patient is so proper therapy can be initiated. Most important is the neurologic examination, as patients who are altered or have neurologic findings or deficits likely have heat stroke.

Heat stroke, whether exertional or classic, has the highest mortality rate of all the heat illnesses. However, in exertional heat stroke, prompt and appropriate cooling yields survival rates of 90% to 100%, whereas classic heat stroke patients tend to have more severe underlying comorbidities, resulting in a significantly higher mortality rate, ranging from 14% to 63%.[20,70-73] Delayed or improper treatment can cause fatalities secondary to end-organ damage in both classic and exertional heat stroke.[6,74] Rapid reduction of core temperature and support of organ system function are paramount to reducing morbidity and mortality, especially in exertional heat stroke[5,52,58,59,69,75] and, to a lesser extent, in classic heat stroke.[20] Previous research has determined that death from classic heat stroke is primarily associated with multisystem organ failure, cardiovascular collapse, and neurologic damage.[59,64,70]

Exertional Heat Stroke Treatment
Rehydration
Contrary to the belief that exertional heat stroke patients are dehydrated (as they present with a history of physical activity), these patients may, in fact, be euvolemic and may suffer harm from massive and rapid rehydration. In an observational study by Seraj et al of 34 patients during the pilgrimage season (*Hajj*) in Saudi Arabia, 22 patients were found to have a normal to above-normal central venous pressure (CVP), while 12 patients were found to have low CVP.[76] Of the normal- to high-CVP group, 6 patients were found to have a CVP elevated such that providing a significant amount of fluid could have precipitated acute congestive heart failure or pulmonary edema. According to this study, an average of 1 L of crystalloid was adequate to normalize CVP in patients with exertional heat stroke.[76]

There is an emerging standard of bedside ultrasound to guide fluid resuscitation, although there is no clinical literature to guide fluid resuscitation specifically in heat illness. It is, therefore, reasonable to base resuscitation efforts on sepsis guidelines.[77] The authors' recommendation for rehydration includes initial 500-mL aliquots of normal saline in mild cases and more aggressive fluid resuscitation in hemodynamically unstable patients, while monitoring blood pressure, urine output, CVP, lactate, and CPK levels. If rhabdomyolysis is present, urine output should be maintained at 50 to 100 mL/h or 2 mL/kg/h. If rhabdomyolysis is not present, a goal of 0.5 to 1 mL/kg/h is accepted by present practice.

One review article states broadly that cooled intravenous fluids are not recommended, as they may induce shivering.[2] However, there is a large body of evidence that shows positive outcomes for cardiac arrest patients on a hypothermia protocol that includes cooled intravenous fluids. A recent case report by Hong et al illustrates the use of a therapeutic hypothermia protocol in a heat stroke patient with multiorgan failure. The subject had a full recovery at 1 year.[78] In a thorough discussion of the pathophysiology, Wilson et al and Nielsen et al further supported the consideration of cooled fluids, given that controlling hyperthermia aids in preservation of neurologic function in cardiac arrest,[79,80] which is very similar to the decrease in perfusion of end organs that is seen in severe heat stroke. While there is no consensus in the literature, cooled intravenous fluids may be considered in the patient with severe heat stroke.[81]

Preventing End-Organ Damage
End-organ damage in exertional heat stroke is similar to that seen in classic heat stroke. Rhabdomyolysis, which can be defined as a CPK level > 5 times the upper limit of normal, is related to muscle breakdown and hypovolemia in heat stroke patients. CPK levels will likely be higher because of increased muscle breakdown in exertional heat stroke when compared with classic heat stroke.[82] Resuscitation efforts are aimed at normalizing intravascular volume to prevent ischemic damage of the renal system while preventing progression to acute renal failure. The literature warns that CPK levels of 5000 IU/L to 10,000 IU/L raise concern for progression to acute kidney injury.[83] Initial management for resuscitation will be guided by the degree of injury and underlying medical condition. Although there are no comparative studies of the exact amount of fluid required in heat illness, 1 to 2 L of normal saline should be started once elevation of CPK is identified. Additional fluid boluses should be given in patients with CPK > 10,000 IU/L unless there are contraindications. CPK, urine output (maintain at 2 mL/kg/h), and renal function should be monitored closely for 24 to 72 hours, during which time the CPK should peak.[83,84]

Case reports have documented possible stress-induced cardiomyopathies such that, if ECG or enzyme abnormalities are seen, further workup is clinically indicated.[85] If patients present with hypotension that is refractory to fluid resuscitation, vasoactive medications may be required.[86,87] Use of such medications may decrease peripheral circulation and heat dissipation. Although several studies mention that dysrhythmias should resolve as the patient is cooled, and cardioversion should be withheld, there is little pri-

mary literature to guide this decision. Clinical practice supports unstable patients being cooled while Advanced Cardiac Life Support (ACLS) is initiated, if indicated. Special attention should also be given to hepatic injury, which, according to several case reports, may be more common than originally thought. Hypotheses for cause of injury include direct thermal insult and hypoxia.[64,88] Although a majority of these injuries resolve and fewer than 10% of patients go on to require transplant,[89] there is little literature to direct treatment; standard monitoring and treatment should be followed. Patients who are admitted with elevated liver enzymes should have the enzymes monitored and trended. The literature does not guide the interpretation of elevated liver enzymes in patients with milder forms of heat illness; these patients may be good candidates for discharge to home. The authors of this review recommend admission if liver enzymes are elevated more than 2 to 3 times the upper limit of normal. Patients with less severely elevated liver enzymes may be discharged, but only after levels have been rechecked and found to be stable or decreasing.

Active Cooling Techniques
Water Immersion Therapy
Water immersion therapy is highly successful in cooling patients, as water distributes and dissipates heat very rapidly and serves as an efficient heat sink or capacitor. This method provides conduction cooling over a large surface area, so the patient should have at least his torso submerged and, if possible, his full body (excluding head).[52] Many earlier studies recommended using cool or tap water (not ice water) for immersion. It was thought that ice water resulted in shivering and cold-induced vasoconstriction that would decrease heat loss and increase metabolic heat production.[58] However, there are several newer studies with opposite findings suggesting that ice water is superior.[90-94] In a review of recent literature, a range of cooling rates has been identified, with ice water immersion providing the most rapid cooling. **(See Table 6.)** To prevent hypothermia, patients should be removed from water immer-

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Table 6. Immersion Cooling Rates, By Water Temperature

- Cooling rates with water immersion temperature < 3°C (ice water) ranged from 0.12°C to 0.35°C/min[92-95]
- Cooling rates with water immersion temperature > 8°C (cold water) ranged from 0.04°C to 0.25°C/min[90-92,95-97]
- Cooling rates in temperate water immersion ranged from 0.10°C to 0.19°C/min[90,92,98]

Note: Factors that affected the above rates and help explain differences include: (1) circulation of water in immersion tub, (2) the amount of body surface immersed, and (3) the location where cooling took place.

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sion therapy when they reach a core temperature of 38.8°C (102°F) to 38.6 (101.5°F).[52]

Although some of the studies that favor ice water immersion reported little to no observed shivering, vasoconstriction could not be directly measured. Casa et al argued that the shorter duration of ice water immersion required to decrease the subject's temperature to a safe level minimizes the effect of increased heat production by shivering.[52] Proulx et al looked at cooling rates of hyperthermic patients (core body temperature > 40°C) when submerged in water ranging from 2°C to 20°C until rectal temperatures reached 37.5°C. They were able to obtain the fastest recorded rate of cooling (0.35°C/min) when subjects were submerged in water at 2°C. Of note, no cold-shock responses (such as hyperventilation and cardiac arrhythmias) were noted. No shivering was recorded by either direct observation or by increase in heart rate.[92] In contrast, a study by Clements et al found there was no difference in cooling rates between ice water (5°C) and cold water (14°C) immersion. This finding may be caused, in part, by vasoconstriction. There is no mention of shivering in this study and the patients were only submerged up to the torso.[96]

Vigorous massage during ice water immersion, spraying immersed patients with 40°C water, and blowing air warmed to 45°C over the submerged patient have all been suggested to help decrease vasoconstriction, but there is no strong evidence to support such practice. Benzodiazepines have been reported to help decrease shivering; however, these medications should be reserved for extreme shivering, and short-acting agents are advised.[99]

Mist-And-Fan Technique
Although it is not as rapid as water immersion therapy, based on review literature, evaporative cooling with misted water and fanning has been shown to successfully cool hyperthermic patients. Mist-and-fan techniques have been used successfully for over 30 years in Saudi Arabia during the *Hajj*. Each year, millions of people make the multiday pilgrimage in extreme environmental conditions. Most of the research by Weiner and Khogali was published in the early 1980s and has flaws in study design, study population, or calculation of cooling rates. Their cooling technique involved misting an undressed patient with water cooled to 15°C while fanning the patient with air warmed to a temperature of 45°C, thus increasing evaporation. In their controlled study, cooling rates reached 0.31°C/min.[100] On repeat studies, cooling rates were found to be closer to 0.05°C/min.[101,102] A more recent observational study conducted by Hadad et al found cooling rates as high as 0.14°C/min when a patient was soaked with tap water and placed in an open, moving vehicle.[103] In the military literature, Poulton et al found a cool-

ing rate of 0.1°C/min when using a helicopter rotary blade.[104] Research in this area lacks rigor, and studies applying very different cooling methods are difficult to compare with one another. Overall, the clinically accepted trend in mist-and-fan cooling suggests fully exposing the patient and using tap water and a fan. Across published literature, rates of cooling appear to be slower than immersion therapy.

Ice Packs And Internal Cooling
Ice packs applied to the groin, axillae, neck, and head, and placement of wet sheets over the patient are other modalities that may be used if preferred cooling techniques are unavailable. This method is inferior to both immersion and the mist-and-fan techniques[3,105] (although there are no additional comparative trials). If external cooling is not effective, internal cooling methods such as gastric, bladder, and rectal lavage can be considered. However, these modalities are poorly studied in humans, may cause water intoxication, and the available literature conflicts.[106] Cardiopulmonary bypass has been reported in extreme cases and does provide effective cooling.[3]

Classic Heat Stroke Treatment
Rehydration
Recommendations for rehydration for classic heat stroke are similar to recommendations for exertional heat stroke: initial 250- to 500-mL aliquots of room-temperature normal saline in mild cases, more aggressive rehydration in severe cases, and monitoring of urine output, CVP, lactate, and CPK levels. This recommendation is based on sepsis literature, as there is a lack of evidence to guide heat illness resuscitation. Urine output should be maintained at 50 mL to 100 mL/h, or 2 mL/kg/h in rhabdomyolysis and 0.5 to 1 mL/kg/h without rhabdomyolysis. Dehydration is more likely in exertional heat illness than in classic heat stroke, but individual methods for monitoring fluid status are the same as in exertional heat stroke. Caution should be exercised in patients with congestive heart failure, renal dysfunction, lung disease, and fluid-overloaded states such as pulmonary edema.

Classic Heat Stroke–Associated Sodium Abnormalities
Hypernatremia and hyponatremia have both been associated with heat stroke in the literature. As patients with classic heat illness are more likely to take multiple medications and have more complex underlying comorbidities, other electrolyte abnormalities may also exist. A study by Hausfater et al looked at sodium measurements in 1263 patients presenting with heat stroke. Hyponatremia was seen in one-third of patients and hypernatremia in just under one-fifth. According to their risk-of-death score, patients

with hypernatremia had a significantly lower 1-year survival rate than the hyponatremia patients. Hypernatremia was found to be independently associated with elevated creatinine and blood urea nitrogen.[30]

Preventing End-Organ Damage
Prevention of end-organ damage is paramount to survival in classic heat stroke. In a retrospective study of 28 patients who had been diagnosed with heat stroke, > 75% of patients progressed to multiorgan dysfunction, with 85% of those patients having respiratory dysfunction.[64] The highest mortality rates were seen in patients with CPK > 1000 IU/L and metabolic acidosis and liver enzymes elevated more than twice normal, which correlated with a mortality rate of 87%.[64] To prevent such outcomes, organ dysfunction must be identified and corrected, if possible.

Rhabdomyolysis is related to muscle breakdown and has been associated with high mortality rates, as described by Varghese.[64] Treatment is aimed at normalizing CPK levels and intravascular volume to prevent ischemic damage of the renal system while preventing progression to acute renal failure. In general, CPK levels should be trended for 24 to 72 hours until they peak, and resuscitation efforts should maintain a urine output of 2 mL/kg/h.[83,84]

Cardiovascular dysfunction was seen in 54% of patients with heat stroke in the retrospective study by Varghese. The definition of cardiovascular system dysfunction used was broad, and included bradycardia, hypotension, and dysrhythmias.[64,107] If ECG or enzyme abnormalities are seen, further workup is clinically indicated, as with cardiovascular dysfunction in exertional heat stroke. Vasoactive medications should be used in refractory hypotension and ACLS should be initiated, if indicated. Hepatic function must also be monitored because, in a minority of patients, direct thermal insult and hypoxia may cause hepatic injury that requires transplant.[64,88] Although a majority of these injuries resolve, there is little literature to direct treatment, so standard monitoring and treatment should be followed.

From a review of the literature, there is little evidence that compares treatment and prevention of end-organ damage in classic heat stroke with that of exertional heat stroke. Many of the treatment recommendations follow standard practice for the specific organ damaged and have not been directly studied in heat illness.

Cooling Techniques
Cooling literature in classic heat stroke is not as robust or recent as that of exertional heat stroke. A 1986 retrospective study of 39 patients by Vicario et al found there to be no significant difference in survival between patients who were rapidly cooled and those who had a delay in cooling > 1 hour.[72] A

2009 study by Pease et al noted that the cooling time required was significantly shorter in survivors. This time discrepancy makes it unclear whether time to cooling is as important to survival in classic heat stroke as it is in exertional heat stroke and highlights an area where research is needed.

Cooling techniques in classic heat stroke are similar to those used in exertional heat stroke. **(See page 8.)** However, in classic heat stroke, immersion therapy may have more barriers to success than in exertional heat stroke. Evaporative cooling techniques with mist and fan or cool gauze and fan may be better tolerated by older individuals, and they allow for easier monitoring. In a case-control study by Hart et al, patients were cooled by ice water immersion or brisk ice massage.[70] Mortality was reported in 14% of patients, and cooling mechanisms were only described as part of overall treatment, so causality cannot be identified. Of note, all patients were cooled within 1 hour of arrival.[70] There are no randomized trials to compare true immersion cooling with evaporative cooling.

As time to cooling may, in fact, improve survival, it is our recommendation that cooling therapy should be initiated as rapidly as possible.[70] Patients may undergo immersion therapy or evaporative cooling depending on comorbidities, extent of illness, and availability of monitoring capabilities. Further research is needed.

Other Cooling Modalities

External cooling devices (such as cooling blankets) may be considered if no other options are available.[78] Devices that adhere to the skin and cover large surface areas have better efficacy in reducing core temperature[108] when compared with cooling blankets, which rely on conduction and provide low and inconsistent surface-area coverage. At this time, no pharmacologic agents have been found to be effective in cooling patients with heat stroke.[3,5] As discussed previously, more invasive internal cooling modalities (such as gastric, bladder, and rectal lavage) lack quality human research and are associated with significant side effects.[106] Cardiopulmonary bypass may be used in extreme circumstances; however, there is a general lack of evidence to guide treatment in the setting of heat illness.[3] Nonetheless, its use is not unseen in emergency medicine.

Risks Of Cooling

There are risks associated with cooling the hyperthermic patient, regardless of whether the patient is diagnosed with classic or exertional heat stroke. Emergency clinicians must be careful not to overcool patients and induce hypothermia. Patients must be monitored to ensure that, once cooling has ceased, their core temperature stabilizes. These patients should be kept on a monitor during observation.

Putting It All Together

Although few hospitals have water immersion facilities, water immersion therapy is accepted by present practice to be the most efficient cooling technique available.[90,92] However, in the present literature, there is a lack of quality trials with adequate numbers of participants that compare different methods of cooling. When cooling rates of individual studies are compared, there appears to be a slight advantage in cool water immersion when compared with the mist-and-fan technique, although there is no randomized comparative research to support it.[52,90,109]

When available, immersion up to the torso or of the full body (excluding the head) is the most effective method for rapid cooling, and ice water should be used. Rectal temperatures need to be monitored frequently to prevent overcooling. Cooling should be stopped when core temperature reaches 38.6°C (101.5°F).[52]

Treatment Of Other Heat-Related Conditions

Prickly Heat (Miliaria)

As this is a disease characterized by incomplete formation of sweat glands, children are most commonly affected. Treatment for associated pruritus may include calamine lotion, cold compresses, and topical steroids. If there is a suspicion of infection, antibiotics should be considered.[110]

Heat Edema

Based on the pathophysiology, this is not a fluid-overloaded state, but a state of vasodilation for whole-body heat reduction. For this reason, there is no role for diuresis. Symptomatic treatment includes cooling and oral hydration.

Heat Tetany And Cramps

Treatment includes oral hydration and symptomatic treatment (ie, stretching and massage). No evidence to support the use of benzodiazepines has been reported. If the patient is found to have renal dysfunction, hyponatremia, or an elevated CPK, intravenous fluids should be given according to the level of associated dehydration, electrolytes should be addressed, and admission should be considered.[111]

Special Populations

The Very Young

Children are at greater risk for heat injury for several reasons. They have a greater surface area to body mass ratio than adults, allowing for more environmental heat transfer. They have slower sweat rates, and they begin to sweat at higher core temperatures than adults. Slower acclimatization to high ambient temperature and a blunted thirst response are also contributing factors. Between 1997 and 2006, nearly one-half of reported exertional heat illnesses were in

Clinical Pathway For Differential Diagnosis Of Heat Illness

Consider potentially life-threatening causes:
- Heat stroke
- Neuroleptic malignant syndrome
- Serotonin syndrome
- Anticholinergic OD
- Sympathornimetic OD
- Aspirin OD
- Sepsis

Also consider:
- CNS infection
- Other medications

← **YES** — **Altered mental status?** — **NO** → **Mild to moderate heat illness (heat stroke unlikely)**

Exertional or classic heat stroke? — **YES** → **Mental status normalizes with euthermia?** — **YES** → **Heat illness confirmed** ← **YES** — **Symptoms resolve with euthermia?**

NO (Exertional or classic heat stroke?) →
NO (Mental status normalizes with euthermia?) →
NO (Symptoms resolve with euthermia?) →

Consider alternate diagnosis by symptoms

Symptoms	Diagnosis
Altered mental status, muscle rigidity, elevated CPK, diaphoresis, NL pupils, phenothiazine exposure	NMS
Altered mental status, myoclonus, HTN, hyperreflexia, increased HR, diaphoresis, dilated pupils, SSRI use	Serotonin syndrome
Altered mental status (confusion); increased HR; dry, hot flushed skin; urinary retention; quiet abdomen; dilated pupils	Anticholinergic OD
Altered mental status (manic), increased HR, HTN, diaphoretic, dilated pupils, recreational sympathomimetic risk	Sympathomimetic OD
Altered mental status, hyperventilation, respiratory alkalosis or metabolic acidosis, tinnitus, increased HR, vomiting, history of ingestion	Aspirin OD
Mixed, other symptoms: consider other causes. Poison control center call is helpful for all cases	Consider other causes

Abbreviations: CPK, creatine phosphokinase; CNS, central nervous system; HTN, hypertension; HR, heart rate; NMS, neuroleptic malignant syndrome; OD, overdose; SSRI, selective serotonin reuptake inhibitor.
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Clinical Pathway For Treatment Of Noninfectious Heat Illness

Patient presents with heat illness

↓

Initiate passive cooling maneuvers:
- Remove from environment
- Place in cool, shaded area
- Remove extra clothing

↓

Altered mental status? — **YES** → Heat stroke

Altered mental status? — **NO** → Mild or moderate heat illness (ie, heat exhaustion or less severe condition)? → MILD (ie, heat tetany or less severe condition)

Heat stroke branch:

Initiate active cooling:
- IV fluids, mist-and-fan
- Ice packs
- Immersion if available and patient would tolerate
- More invasive methods in severe and high-risk cases only

↓

Resolves with cooling? — **YES** → Admit vs selective discharge

Resolves with cooling? — **NO** → Consider more severe or atypical process

Mild or moderate branch:

Initiate active cooling:
- IV fluids, mist-and-and fan
- Ice packs
- Immersion if available and patient would tolerate
- More invasive methods in severe and high risk cases only

↓

Resolves with cooling? — **YES** → Initiate passive cooling:
- PO fluids
- Observe vs discharge home when feeling better

Resolves with cooling? — **NO** → Consider more severe or atypical process

MILD branch:

Initiate passive cooling:
- PO fluids
- Observe vs discharge home when feeling better

Consider more severe or atypical process

NMS	Administer bromocriptine, dantrolene **(Class III)**
Serotonin syndrome	Administer benzodiazepine, cyproheptadine **(Class III)**
Anticholinergic OD	Administer physostigmine (monitor for dysrhythmia) **(Class III)**
Sympathomimetic OD	Administer benzodiazepines, sedation (avoid phenothiazines) **(Class III)**
Aspirin OD	Administer alkalinization, hemoperfusion **(Class III)**
Other medication (eg, lithium)	Administer drug-specific antidote (toxicology consult) **(Class III)**
Inadequate resuscitation	Administer continued therapy and evaluation **(Class III)**
Consider other	Administer infectious disease consult, toxicology consult, sepsis screen **(Class III)**

For Class of Evidence definitions, see page 14.

Abbreviations: CPK, creatine phosphokinase; CNS, central nervous system; HTN, hypertension; HR, heart rate; NMS, neuroleptic malignant syndrome; OD, overdose; PO, by mouth; SSRI, selective serotonin reuptake inhibitor.

Adapted with permission from SafetyCore™. © SafetyCore™ 2014.

children and adolescents.[6] Additionally, very young children may not be able to remove themselves from hot environments. Every year, children die from heat stroke after being left in unattended vehicles. Heat stroke and death in these patients can occur even at relatively mild ambient temperatures.[112]

The Very Old

The elderly are more susceptible to death from heat stroke during heat waves. Aging patients are not as capable of increasing cutaneous blood flow or increasing cardiac output when exposed to high temperatures. These mechanisms are critical for heat dissipation during periods of exposure to passive thermal stress. The decrease in cardiac output results from a diminished stroke volume and a reliance on increasing heart rate to preserve cardiac blood flow. In an elderly patient with cardiovascular disease, an already-strained left ventricle may not be able to tolerate these physiologic changes, leading to death from cardiovascular events.[113] Other factors that predispose the elderly to death during heat waves include medication use (eg, beta blockers), debilitation, and volume depletion.[6]

The Agitated Patient

The agitated patient presenting to the ED should be evaluated promptly for hyperthermia. Excited delirium syndrome is a constellation of symptoms and behaviors usually associated with abuse of sympathomimetic drugs or long-term use of psychiatric medication. The syndrome typically presents with bizarre, hyperaggressive behavior, along with profound hyperthermia and tachycardia. There is often a struggle with law enforcement or healthcare professionals, during which the patient encounters a noxious physical or chemical stimulus. This is followed by a quiescent period, after which sudden death may ensue. The mechanism of sudden death in these patients is poorly understood and is likely multifactorial. The syndrome can be differentiated from other clinical entities because delirium is accompanied by extremely aggressive and violent behavior. Treatment includes prompt chemical sedation to minimize physical struggle with the patient and initiation of active external cooling maneuvers.[114]

Patients With Drug-Induced Hyperthermia

Patients taking neuroleptics and serotonin reuptake inhibitors, as well as other psychoactive medications (such as lithium), may develop dangerous hyperthermia via differing mechanisms. Central nervous system dopamine-receptor antagonists can cause neuroleptic malignant syndrome, which results in muscle rigidity and generation of peripheral heat energy as well as disturbance of central temperature regulation mechanisms in the hypothalamus.[115] The mechanism of action for neuroleptic malignant syndrome from lithium use has been poorly defined, but emergency clinicians should be aware that lithium causes neuroleptic malignant syndrome independent of other agents frequently implicated in the disease.[116] Serotonin reuptake inhibitors (used in the treatment of depression and other psychiatric conditions) can cause serotonin syndrome with resultant hyperthermia after increased dosing, addition of other serotonergic agents, overdose, or substance abuse.[117] Serotonin-induced hyperthermia results from increased muscle tone and rigidity, leading to generation of heat that the body is unable to dissipate.[68]

If such medications are suspected to have caused hyperthermia, treatment will differ from that of heat stroke. Precipitating medications should be stopped, which may be all that is required if the illness is mild. Neuroleptic malignant syndrome may require intravenous fluids, bromocriptine, or dantrolene. Serotonin syndrome may be treated with intravenous fluids and benzodiazepines.[118] Patients with lithium toxicity should be given intravenous fluids and, if critical levels are met, they should undergo hemodialysis.[119]

Patients who abuse sympathomimetic drugs are at risk of developing hyperthermia. The cause

Class Of Evidence Definitions

Each action in the clinical pathways section of *Emergency Medicine Practice* receives a score based on the following definitions.

Class I
- Always acceptable, safe
- Definitely useful
- Proven in both efficacy and effectiveness

Level of Evidence:
- One or more large prospective studies are present (with rare exceptions)
- High-quality meta-analyses
- Study results consistently positive and compelling

Class II
- Safe, acceptable
- Probably useful

Level of Evidence:
- Generally higher levels of evidence
- Nonrandomized or retrospective studies: historic, cohort, or case control studies
- Less robust randomized controlled trials
- Results consistently positive

Class III
- May be acceptable
- Possibly useful
- Considered optional or alternative treatments

Level of Evidence:
- Generally lower or intermediate levels of evidence
- Case series, animal studies, consensus panels
- Occasionally positive results

Indeterminate
- Continuing area of research
- No recommendations until further research

Level of Evidence:
- Evidence not available
- Higher studies in progress
- Results inconsistent, contradictory
- Results not compelling

is multifactorial and depends on the mechanism of action of the drug being misused. Sympathomimetic drug use may result in vasoconstriction from direct alpha-receptor stimulation, impairing the ability of the body to lose heat through cutaneous blood flow. These substances also cause psychomotor agitation with increased heat production.[120] Commonly abused sympathomimetics include cocaine, ecstasy (MDMA), 3,4-methylenedioxypyrovalerone (MDPV, "bath salts"), amphetamines, and phencyclidine (PCP). Cocaine use with concomitant elevated ambient temperature has been shown to increase cocaine abuse–related deaths.[121] This suggests that in warmer climates, patients who use dangerous sympathomimetics are at higher risk of hyperthermia and heat stroke.

Anticholinergics remain a cause of both accidental and intentional intoxication.[34] Jimson weed and other naturally occurring plants and mushrooms

contain anticholinergic compounds. Hyperthermia results from blockade of both peripheral and central muscarinic acetylcholine receptors. Peripheral blockade prevents sweating and cutaneous heat loss. Central muscarinic blockade leads to agitation and increased heat production.[115] When compared with the sympathomimetics, anticholinergic agents often induce milder hyperthermic effects.[120]

Controversies And Cutting Edge

The current management of heat illness is relatively noncontroversial. There is widespread agreement that therapy should be initiated as rapidly as possible, including in the prehospital environment. Current research has refocused attention on established cooling techniques, such as cold water immersion. The general consensus is that immersion is effective. The data stating that immersion causes shivering and worsening hyperthermia is scant and anecdotal. No modality, to date, has been shown to cool more quickly than cold water immersion, probably because of the large contact area during immersion and the high heat capacitance of water. The application of surface cooling devices, such as those used in hypothermia protocols, also presents a cutting-edge technology. Surface cooling devices can be used on patients who cannot get in and out of an immersion tank, and on patients who need to remain in a bed.[78,122]

Of special concern is rhabdomyolysis, an area in which there has been a recent increase in research. This is important for management of heat illness because rhabdomyolysis often accompanies moderate to severe heat illness. The main complication of rhabdomyolysis is acute kidney injury, but there has not been a great deal of research toward establishing exactly what level of rhabdomyolysis is associated with acute kidney injury. This question is answered indirectly by looking at other factors. What may be most important is monitoring the absolute CPK level, considered in conjunction with associated findings (such as acidosis, myoglobin level, and hyperkalemia).

Disposition

The majority of patients with mild to moderate heat illness can be safely discharged to home. Factors associated with improved outcome include younger age, fewer medical problems, working air conditioning in the home, adequate social contact, availability of a cool environment, and resources to bathe more often.[123] Criteria for safe discharge include re-establishment of adequate intravascular volume, euthermia, ability to tolerate fluids, and absence of major laboratory abnormality.

Time- And Cost-Effective Strategies

- Begin cooling immediately. Cooling can begin in the prehospital phase, and aggressive cooling can begin even as the history and physical examination are being performed. Strategic ice pack placement and misting and spraying are effective and inexpensive cooling modalities. Simply getting the core temperature to ≤ 40°C quickly can reverse much of the pathophysiology of heat illness.
- Fluid repletion (oral or intravenous) is a mainstay of therapy and re-establishes the body's ability to thermoregulate by circulation and sweating.
- Blood tests to determine end-organ damage should be considered in the management of heat illness, especially if it is severe or if the patient is very old or debilitated, has multiple comorbidities, or appears otherwise ill after cooling and rehydration.
- There is no test for heat stroke, but the cardinal sign is central nervous system alteration. The emergency clinician can be confident that central nervous system alteration represents a potentially life-threatening situation. The presence of altered mental status should prompt the emergency clinician to perform 4 critical tasks: (1) cool the patient immediately; (2) obtain a CBC with differential, basic metabolic panel, CPK level, lactate, ECG, and chest x-ray; (3) plan for admission; and (4) screen for diseases that can present with hyperthermia (such as meningitis, drug-induced hyperthermia, withdrawal syndromes, and intracranial processes).

A large retrospective study concluded that patient characteristics associated with need for admission included advanced age, comorbidities, male sex, and low socioeconomic status.[124] The majority of deaths occur in patients aged > 65 years, patients with cardiovascular disease, and patients with mental illness.[15] Additional factors associated with death included bedridden status, lack of daily excursion outside the home, and inability to care for self.[123] Based on the evidence, the authors recommend a cautious approach in patients with these risk factors. With regard to the severity of the episode, any patient with severe heat stroke, regardless of age or comorbidities, should be observed at least until the altered mental status resolves. Significant laboratory abnormalities that warrant admission until resolution include renal failure, hyponatremia, cardiac ab-

normalities, and markedly elevated CPK. Although the literature varies, a CPK > 5000 IU/L is considered severe, and a level > 10,000 IU/L is highly correlated with acute kidney injury, as are metabolic acidosis, hypoalbuminemia, elevated liver function tests, and hyperkalemia.[125] Myoglobin level > 600 ng/mL has also been shown to be a useful predictor of which patients will go on to develop acute kidney injury.[126] Upon hospital admission, acuity should be dictated by the clinical picture; persistent acidosis, hypotension or tachycardia, or severe elevation of liver function tests warrants placement in a higher acuity area of the hospital.

The best evidence guiding the disposition of the young athlete or military personnel with exertional heat illness comes from the consensus statement of the American College of Sports Medicine posi-

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Risk Management Pitfalls For Heat Illness

1. **"The patient did not respond to multiple doses of diazepam and he required general anesthesia and intubation."**
Persistent agitation after control of temperature or inability to control temperature should prompt consideration of other causes and contributors to heat illness. Sympathomimetic intoxication associated with heat illness will respond well to benzodiazepines, but anticholinergic syndrome and neuroleptic malignant syndrome will not. Before moving to general anesthesia, emergency clinicians should consider specific antidotes and treatments for unusual causes of hyperthermia.

2. **"The patient came in febrile and altered, but now he is better. He can definitely go home."**
Before discharging anyone with heat illness or heat stroke, consider the factors that are associated with mortality (such as advanced age, comorbidities, immobility, social isolation, cardiovascular disease, and male sex). Also remember that rhabdomyolysis is a frequent complication of heat illness and that factors such as CPK > 10000 IU/L, hyperkalemia, and metabolic acidosis are associated with a higher likelihood of acute kidney injury. Assess a checklist of criteria that are associated with safe discharge, including working air conditioning and social support. Be sure that symptoms are resolved and that any marked vital sign or laboratory abnormality has normalized before discharge.

3. **"Haloperidol, lorazepam, and cooling maneuvers could not reverse the heat stroke and heat-induced psychosis; I have no idea why the patient died."**
Benzodiazepines are a good starting choice when sedation is necessary. Phenothiazines, because of their potential extrapyramidal side effects and their anticholinergic effects, are not a good choice in hyperthermia and agitation. The anticholinergic effects can markedly decrease the ability of the body to sweat and the extrapyramidal effects can worsen hyperthermia. They can also cause neuroleptic malignant syndrome.

4. **"The patient was given naloxone, and thereafter developed severe agitation. He must have been doing a speedball. Just keep giving him diazepam."**
A speedball is a mixture of heroin and cocaine. When the heroin is reversed with naloxone, the sympathomimetic effects are unmasked and the patient may become agitated. Occasionally, heroin is mixed with an anticholinergic such as scopolamine. In this case, when the heroin is reversed, a full-blown anticholinergic syndrome may result. You can distinguish between sympathomimetic and anticholinergic toxidromes by remembering how they differ. The anticholinergic patient will tend be more profoundly delirious, and will have dry, flushed skin, urinary retention, and a quiet abdomen, whereas the patient abusing sympathomimetic drugs will tend to be manic, will tend to sweat profusely, and will respond better to benzodiazepines.

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tion paper.[93] The authors agree with the American College of Sports Medicine recommendation that patients with heat exhaustion abstain from training and competition for 1 to 2 days, and that patients who have suffered from heat stroke abstain for at least 1 week. There is evidence that patients who return to their activities prematurely after heat exhaustion or heat stroke are at higher risk of recurrence. In the opinion of the authors, 24-hour follow-up on any patient discharged with heat exhaustion or heat stroke is prudent.

Case Conclusions

You intubated your 16-year-old patient with exertional heat stroke and gave him 2 L of room-temperature normal saline. It wasn't possible to immerse him in cold water with the equipment available in your department, so you placed ice packs on his groin, axillae, and neck, and asked the nurses to dampen his skin with tepid water. You found a fan in the nurses' break room and placed it at the patient's bedside to take advantage of evaporative and convective cooling. You continued to monitor the patient's rectal temperature, which dropped to 38.8°C (102°F), but you continued to cool him with a goal of 38.6°C (101.5°F) in mind. The patient's laboratory results revealed a CPK of 4800 IU/L and a serum creatinine of 1.6 mg/dL, so you continued to volume resuscitate him with normal saline to keep his urine output at 50 to 100 mL/h, and the ICU team was notified.

For your second patient, the hyperthermic 35-year-old woman with mental confusion and rigidity, you decided to administer an additional dose of IV benzodiazepine. Just as the nurse administered the second dose,

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Risk Management Pitfalls For Heat Illness (Continued from page 16)

5. **"Please give the febrile marathoner with heat exhaustion 3 L of normal saline and then recheck his vital signs."**
Healthy patients who participate in marathons or other high-endurance events or scenarios can develop hyponatremia. In the exertional heat illness group, it is important to check serum sodium level before administering a large amount of saline.

6. **"But I treat all my agitated patients with haloperidol."**
Sedation of the patient with heat illness merits careful consideration. Benzodiazepines are safe, but they may obscure the real cause of hyperthermia. It is important to try to establish the likely cause of hyperthermia before sedating the patient. If you do judge that a sedative is necessary, benzodiazepines are the safest and most generally useful class.

7. **"The patient is altered because he is old, febrile and demented."**
It may be the case that a fever can, by itself, cause alteration of mental status in an elderly patient, but bear in mind that elderly patients with immobility and dementia are at increased risk for heat stroke. Additionally, the elderly are more likely to be on complex combinations of medications, which may predispose them to heat-related illness.

8. **"Please give that 98-year-old a quick 3 L of normal saline and when you are done, let me know."**
The elderly, in general and especially with heat illness, can become hyponatremic. If the sodium level is very low and it is corrected too quickly, central pontine myelinolysis can occur. To avoid complications of hyponatremia and hypernatremia reversal, a sodium level should be obtained as quickly as possible so that sodium derangement may be corrected appropriately. Additionally, conditions such as congestive heart failure or renal disease can thwart the ability of the elderly patient to tolerate rapid resuscitation even if the fluid deficit is fairly large. Careful but continuous fluid administration with frequent rechecking is prudent.

9. **"Her legs were swollen so I prescribed furosemide. I had no idea she would get dehydrated after discharge, fall, and break her hip."**
Heat edema occurs frequently, especially early in the heat season and it often resolves with acclimatization. Treatment emphasizes support stockings and elevating the extremities, not on diuretics.

10. **"He looked great at discharge. I couldn't believe he died the next day at football practice."**
Young patients with exertional heat exhaustion or heat stroke can quickly recover and appear well. However, their ability to regulate temperature may be impaired, and they should be returned to play slowly over time, with close monitoring.

the clerk arrived with the records you requested from the urgent care center. You noticed right away that she was treated for her nausea and vomiting the previous morning with promethazine and prochlorperazine. You remembered that the phenothiazine antiemetics are associated with neuroleptic malignant syndrome and that symptoms can begin within 24 hours of administration of a precipitating agent. You undressed the patient and placed ice packs on her groin and bilateral axillae to cool her, and you began fluid resuscitation with normal saline. You contacted the ICU team and administered 2.5 mg of bromocriptine orally and continued to monitor her core temperature and hemodynamics. You considered giving the patient IV dantrolene for her marked rigidity, but to your relief, her rigidity began to subside with your treatments.

References

Evidence-based medicine requires a critical appraisal of the literature based upon study methodology and number of subjects. Not all references are equally robust. The findings of a large, prospective, randomized, and blinded trial should carry more weight than a case report.

To help the reader judge the strength of each reference, pertinent information about the study will be included in bold type following the reference, where available. In addition, the most informative references cited in this paper, as determined by the authors, will be noted by an asterisk (*) next to the number of the reference.

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CME Questions

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1. **The feature that differentiates heat stroke from heat exhaustion is:**
   a. Temperature > 40°C (104°F)
   b. CPK > 10,000 U/L
   c. Mental status alteration
   d. Elevated anion gap and hyperthermia

2. **Which of the 4 heat transfer mechanisms is the primary method the human body uses to cool itself?**
   a. Conduction
   b. Convection
   c. Radiation
   d. Evaporation

3. **Wet bulb globe temperature is:**
   a. A laboratory test for establishing the sodium content of serum
   b. A temperature that factors ambient temperature, radiant heat, humidity, and wind speed
   c. The wind chill factor
   d. A measurement of ocular temperature to determine blood flow

4. **Active cooling maneuvers do not need to be instituted in exertional heat stroke unless the patient remains hyperthermic after 1 hour in the ED.**
   a. True
   b. False

5. **All of the following drugs may increase susceptibility to hyperthermia EXCEPT:**
   a. Lithium
   b. Tricyclic antidepressants
   c. Benzodiazepines
   d. Anticholinergics

6. **Chilled fluids and cold water immersion are contraindicated because they cause shivering, which will elevate the patient's temperature.**
   a. True
   b. False

7. **You volunteer to be a medical director for a local half-marathon with 3000 runners. The temperature is expected to be in the low 80s °F. The sponsors tell you about an old immersion tank the previous medical director said to put in storage, and it costs $200 to deliver it and $50 to keep it filled with ice to maintain a temperature of about 65°F (18.3°C). It leaks and the temperature won't stay any lower than about 70°F. Your response is:**
   a. Immersion tank treatment is unlikely to be effective unless the temperature of the water is 60°F or less.
   b. Cold water tank immersion causes shivering and makes heat illness worse.
   c. Heat illness and heat stroke are definitely possible at this temperature and immersion tank treatment is effective, but it should be used sparingly because of its side effect profile.
   d. Heat illness and heat stroke are definitely possible at this temperature, immersion tank treatment is effective, and it is worth the cost to have on hand.

8. **You have a somewhat frail elderly patient with heat stroke, a CPK of 3050 IU/L, a slightly elevated creatinine, serum sodium of 126 IU/L, potassium of 2.9 IU/L, a mildly elevated anion gap, and no changes in mental status. You call the hospitalist to arrange admission and monitoring for heat stroke and rhabdomyolysis. The hospitalist replies that: (1) the patient does not have heat stroke if there are no mental status changes, and (2) that only a CPK ≥ 10,000 U/L is associated with progression to acute kidney injury, so the patient should not be admitted. Which of the hospitalist's assertions are correct?**
   a. He is correct about heat stroke, but he is incorrect about rhabdomyolysis. A CPK of < 10,000 U/L is more likely to cause acute kidney injury if there are associated comorbidities and acid/base or electrolyte abnormalities.
   b. He is incorrect about heat stroke, but he is correct about rhabdomyolysis. It takes a CPK of ≥ 10,000 U/L to cause acute kidney injury.
   c. Neither
   d. Both

9. **An affluent-appearing woman is brought in by EMS for suicide attempt by pill ingestion. It is July and the patient was found in a room alone, semiconscious with a note stating she was depressed and wanted to "end it all." En route, she vomited, and the EMS crew administered promethazine. On arrival, she is hyperthermic and altered. You appropriately intubate the patient for failure to protect the airway and you begin active cooling maneuvers, but the patient fails to respond and is increasingly altered. You remember reading an *Emergency Medicine Practice* issue on drug-induced hyperthermia, and that:**
   a. Altered mental status, muscle rigidity, diaphoresis = aspirin overdose
   b. Altered mental status, hyperventilation, myoclonus, dilated pupils, and respiratory alkalosis = neuroleptic malignant syndrome
   c. Red as a beet, hot as a hare, goofy as a dog, dry as a bone = serotonin syndrome
   d. Altered mental status, myoclonus, hypertension, hyperreflexia, increased heart rate, diaphoresis, and dilated pupils = serotonin syndrome

10. **In heat stroke, liver functions tests are of little prognostic value.**
   a. True
    b. False

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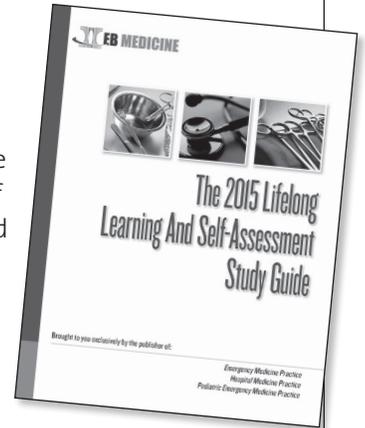
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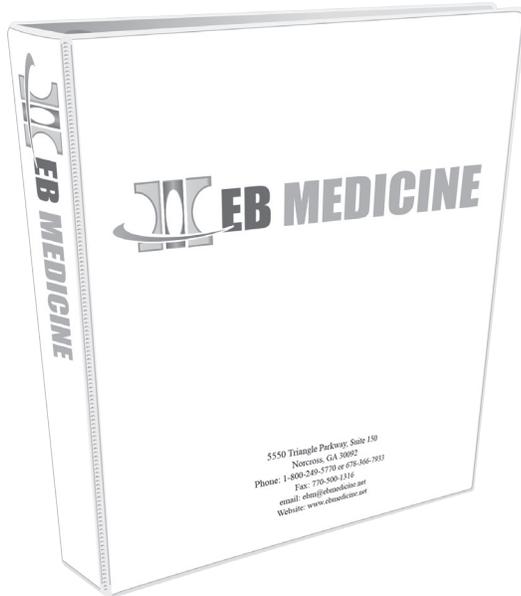
Physician CME Information

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Accreditation: EB Medicine is accredited by the Accreditation Council for Continuing Medical Education (ACCME) to provide continuing medical education for physicians. This activity has been planned and implemented in accordance with the Essential Areas and Policies of the ACCME.

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Needs Assessment: The need for this educational activity was determined by a survey of medical staff, including the editorial board of this publication; review of morbidity and mortality data from the CDC, AHA, NCHS, and ACEP; and evaluation of prior activities for emergency physicians.

Target Audience: This enduring material is designed for emergency medicine physicians, physician assistants, nurse practitioners, and residents.

Goals: Upon completion of this article, you should be able to: (1) demonstrate medical decision-making based on the strongest clinical evidence; (2) cost-effectively diagnose and treat the most critical ED presentations; and (3) describe the most common medicolegal pitfalls for each topic covered.

Objectives: Upon completion of this article, you should be able to: (1) recognize and treat the spectrum of heat illness, including heat stroke; (2) follow standard and best practices for rapid cooling of patients with heat illness; (3) recognize when heat illness is caused by factors other than exertion and exposure; and (4) apply the respective therapies for these atypical causes of heat illness.

Discussion of Investigational Information: As part of the newsletter, faculty may be presenting investigational information about pharmaceutical products that is outside Food and Drug Administration-approved labeling. Information presented as part of this activity is intended solely as continuing medical education and is not intended to promote off-label use of any pharmaceutical product.

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