



Heat illness: Tips for recognition and treatment

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ABSTRACT

Heat stroke, an acute, life-threatening emergency, results from an overload or impairment of heat-dissipating mechanisms. At risk are the elderly, infants, the obese, people with hyperthyroidism, and those taking certain drugs. Early recognition and rapid cooling are essential—the more rapid the cooling, the lower the mortality.

IF HEAT ILLNESS can be caught early, the morbidity and mortality can be significantly reduced. Heat illnesses are a spectrum of syndromes that range from minor conditions such as muscle cramps to the acute, life-threatening emergency of heat stroke. Each year, more than 4,000 people in the US die of heat stroke.¹

This paper deals only with heat exhaustion and heat stroke, as they pose the highest risk of morbidity and mortality to our patients.

HEAT EXHAUSTION VS HEAT STROKE

Heat exhaustion and heat stroke are often discussed separately, but this division can be misleading because they represent a continuum.

Heat exhaustion often presents with flu-like symptoms that may include headache, nausea, vomiting, malaise, muscle cramps, and dizziness (TABLE 1). The temperature is typically normal but can be elevated; it is usually less than 41°C (106°F) if elevated. Clinical signs and symptoms of dehydration are almost always present in the form of tachycardia, hypotension, and diaphoresis.

Heat stroke must be considered in anyone who presents with hyperthermia and

TABLE 1

Heat exhaustion vs heat stroke

	HEAT EXHAUSTION	HEAT STROKE
Symptoms	Flulike: headache, nausea, vomiting, cramps, dizziness	Same
Central nervous system symptoms	Not present	Present, including inappropriate behavior, confusion, delirium, ataxia, seizure, coma
Temperature	Typically < 41°C, usually normal	Typically > 41°C
Sweating	Present	May be absent

altered mental status.

The key point in differentiating heat stroke from heat exhaustion is that central nervous system dysfunction is present in heat stroke but not in heat exhaustion. Central nervous system symptoms classically seen in heat stroke include confusion, delirium, ataxia, seizure, and coma. The cerebellum is most sensitive to heat, and ataxia may be an early sign. Any evidence of central nervous system dysfunction requires aggressive cooling.

Dry, hot skin is not required for the diagnosis of heat stroke. In a prospective study of military recruits,² 50% of patients presenting with full-blown heat stroke maintained the ability to sweat.

The exact temperature at which cellular damage starts to occur is not clear; however, uncoupling of oxidative phosphorylation occurs at 42°C (107.6°F), thus stopping adenosine triphosphate (ATP) production by the mitochondria. The cellular damage is believed to be a function of both the maximum temperature reached and the exposure time at that temperature. Therefore, the faster a patient is cooled the better the prognosis.

Heat stroke is an acute, life-threatening emergency

Suspect heat stroke in anyone with hyperthermia and altered mental status

■ PHYSIOLOGIC RESPONSE TO HEAT

There are a multitude of physiologic responses to heat stress. The cutaneous vessels dilate to increase the surface area for cooling, and the splanchnic vessels constrict to shunt more blood to the periphery. To maintain blood pressure in the face of this greatly decreased peripheral vascular resistance, cardiac output may double, triple, or even quadruple. The volume of sweat increases, as do levels of antidiuretic hormone and aldosterone.

■ HEAT ACCUMULATION AND DISSIPATION

Basal metabolism alone can generate up to 100 kcal/hour.³ Solar radiation can add 100 to 150 kcal/hour, and moderate work can add 300 to 600 kcal/hour. In the absence of heat-dissipating mechanisms, the addition of 70 kcal can increase the core temperature by approximately 0.8°C (1.4°F).

The body has several mechanisms to dissipate this heat to the environment, including radiation, conduction, convection, and evaporation. Of these, radiation and evaporation are the most important.

Radiation, or the transferring of heat from the body to the cooler external environment, accounts for 65% of cooling as long as the air temperature is lower than the body temperature. Once the external temperature reaches 95°F, however, the body can dissipate heat only by evaporation.

Normally, 30% of cooling is from evaporative heat loss. When the humidity level exceeds 75%, however, the potential for evaporative heat loss begins to decrease. In fact, sweat that drips from the skin does not provide any cooling benefit and only exacerbates dehydration. As a result, the combination of high temperature and high humidity essentially blocks the two main physiologic mechanisms that the body uses to dissipate heat, thus placing the patient at risk for heat stroke.

■ RISK FACTORS FOR HEAT STROKE

Extremes of age. The elderly are less able to increase their cardiac output for heat dissipation and are more often relatively dehydrated. They also commonly have comorbid con-

ditions such as coronary artery disease, congestive heart failure, or previous myocardial infarction that might limit their ability to compensate for peripheral vasodilatation.

Neonates are at risk because they lack thermoregulatory and sweating capabilities.

Obesity is a risk factor for heat stroke because these individuals have more insulation and a lower surface area-to-volume ratio and therefore less capacity for dissipating heat.

Certain dermatologic conditions as well as burns that affect a large surface area can limit heat dissipation by sweating, thus placing these patients at risk for heat stroke.

Hyperthyroidism can markedly increase the metabolic rate, thus effecting a dramatic rise in temperature from increased endogenous heat production.

Various medications and drugs, many of which are quite common, may predispose a patient to heat exhaustion and heat stroke.

- Beta-blockers inhibit the compensatory increases in cardiac output.
- Diuretics produce a relative state of dehydration that in turn affects both central thermoregulation and sweating. A mere 2% decrease in body weight through fluid loss increases the heart rate and body temperature and decreases plasma volume.
- Anticholinergic drugs such as tricyclic antidepressants, antihistamines, antispasmodics, phenothiazines, and lithium can disrupt hypothalamic function and also reduce sweating.
- Drugs of abuse such as cocaine; amphetamines and their derivatives such as MDMA ("ecstasy") and MDEA ("Eve"); phencyclidine (PCP); lysergic acid diethylamide (LSD); and other stimulants can increase muscular and endogenous heat production. Amphetamines and LSD also act directly on the hypothalamus to raise temperatures.

■ COMPLICATIONS OF HEAT STROKE

Multiple organ systems are affected by elevated core temperature. The mechanism of injury at the cellular level is multifactorial. Oxidative phosphorylation uncouples, enzyme systems stop functioning, proteins denature, and membranes become more permeable.



The central nervous system is particularly affected; this phenomenon prompted the statement by Hamilton, "It does not take long either to boil an egg or to cook neurons."²

The kidneys are affected by decreased renal perfusion, which can lead to acute tubular necrosis and renal failure.

Damage to muscle and rhabdomyolysis can produce myoglobinuria and exacerbate the ensuing nephropathy.

Hepatocellular injury often occurs and is reflected by markedly elevated liver enzyme levels. Aspartate aminotransferase (AST), alanine aminotransferase (ALT), and lactate dehydrogenase (LDH) levels may rise into the tens of thousands of units per liter if the patient survives the first 24 hours. Even in heat exhaustion, elevations of liver enzyme levels as high as several thousand are often seen.

Bleeding due to consumptive coagulopathy may occur. Thermal damage to the vascular endothelial cells leads to exposure of type III basement membrane collagen.

The only organ that appears to be unaffected by heat stroke is the pancreas.

■ TREATMENT OF HEAT EXHAUSTION

Rest and removal from the heat stress is vital. The mainstay of treatment in heat exhaustion is rehydration. If symptoms are mild, oral fluids may be adequate. As the symptoms become more severe, intravenous fluids may be necessary to quickly rehydrate the patient. Conservative measures of cooling can be initiated, but aggressive cooling is not necessary in heat exhaustion.

■ TREATMENT OF HEAT STROKE

Heat stroke is considered an acute life-threatening emergency: several studies showed the mortality rate to be as high as 30% to 80%.⁴ As soon as heat stroke is diagnosed or even suspected, cooling measures should be started.

Aspirin and acetaminophen should *not* be given, as they are ineffective. The hypothalamic setpoint is not elevated as it is in fever. Furthermore, aspirin is contraindicated because of its effect on platelets and clotting.

The optimum method of cooling, while providing the greatest access to the patient, is

enhanced evaporative cooling. While ice baths are effective, they limit the health care provider's access to the patient and are less practical for managing monitors, intravenous lines, and endotracheal tubes.

Evaporative cooling also minimizes avoidance behavior and shivering. If shivering does occur, medications such as meperidine (Demerol) and diazepam (Valium) are very effective in eliminating this unwanted effect.

The patient's clothing should be completely removed, and slightly warm water should be sprayed on the patient to promote evaporative cooling. Ice bags or cold compresses should be placed in areas where large vessels are close to the body surface such as the groin, axilla, and scalp. A fan should be used to maximize evaporation.

Most patients can be cooled to temperatures less than 101°F in less than 40 minutes using this technique. Hyperthermia may recur due to thermoregulatory instability, thus requiring close temperature monitoring and possibly additional cooling at a later time. Urine output and central venous or wedge pressures should guide administration of intravenous fluids.

Initial lab work should include a complete blood count, electrolytes (including calcium), blood urea nitrogen (BUN), serum creatinine, liver enzymes, creatine kinase, the prothrombin time and the partial thromboplastin time, and arterial blood gasses. ■

■ REFERENCES

1. Clowes GH Jr, O'Donnel TF. Heat stroke. *N Engl J Med* 1974; 291:564-567.
2. Hamilton D. The immediate treatment of heatstroke. *Anaesthesia* 1976; 31:270-272.
3. Knochel JP, Reed G. Disorders of heat regulation. In: Kleeman CR, Maxwell MH, Nari RG, editors: *Clinical disorders of fluid and electrolyte metabolism*. New York: McGraw-Hill, 1987.
4. Hubbard RW, Gaffin SL, Squire DL. Heat-related illnesses. In: Auerbach PS, editor: *Wilderness medicine*. St. Louis: Mosby; 2001:195.

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Treatment:
rest, removal
from heat,
rehydration,
evaporative
cooling