Clinical

Managing Heat Illness *in Urgent Care*

Urgent message: The urgent care provider's most critical role in heat illness is to identify risk factors and the cause, to cool and hydrate the patient, assess for complications, and educate the patient in the hope of preventing a more serious exposure.

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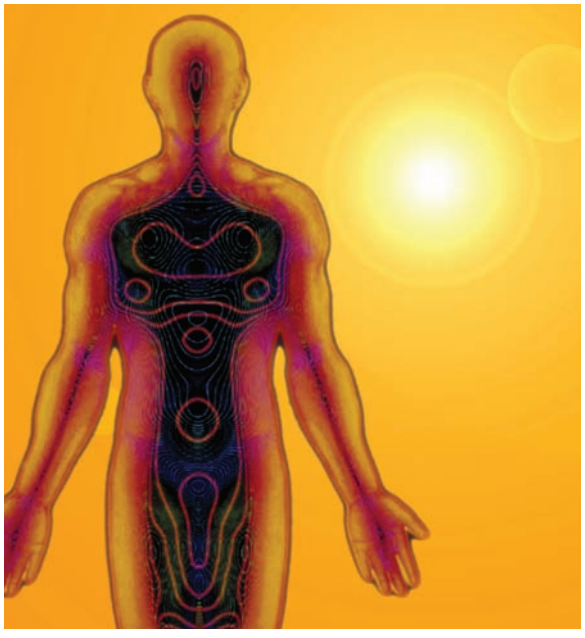
Introduction

Heat illness occurs when external heat conditions and internal heat production overwhelm the ability of the body to dissipate heat. Evaporation of sweat is the most effective way to dissipate heat; when the humidity is high, evaporation is compromised. Calculations that are based on both temperature and humidity, such as the heat index, are a more robust way of determining heat stress than ambient temperature alone.[1] Elevated humidity can cause even moderate temperatures to be dangerous, especially to persons at high risk.



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Internal heat production depends on both the level of exertion and the physiologic characteristics of the patient. In the average adult, exertion can raise the basal metabolic rate from 100 kcal/hr to more than 1000 kcal/hr, 70% to 100% of which is released as heat.[2]

Physiology

As the core temperature exceeds the hypothalamic set point, heat avoidance behavior is induced, and both sympathetic and parasympathetic tones are augmented.

Increased sympathetic tone increases cardiac output, supporting cutaneous and skeletal muscle vasodilatation, allowing for radiation of heat, delivering plasma for sweat and oxygen for exertion, with contraction of splanchnic circulation.

Parasympathetic tone modulates sweating, increasing to 2.5 liters per hour in an acclimated person during strenuous exercise.[3] Heat shock proteins (HSP) act as molecular chaperones that prevent denaturing of other proteins at higher temperatures. Initial heat stress triggers increase HSP expression, which protects cells from a second heat exposure.[4] Any process or comorbidity that interferes with cardiac output, va-

TABLE 1.
Key Risk Factors for Heat Illness or Heat-Related Death[9,10]

Age
- Elderly (>65 years old) or very young (<5 years old)

Chronic disease
- Cardiac
- Mental illness
- Endocrine
- Nutrition
- Infection

Drugs
- Sympathomimetics
- Neuroleptics
- Cardiovascular
- Diuretics
- Alcohol
- Anticholinergics

Behavior
- Failure or inability to seek cool environment
- Left unattended in car
- Low fluid intake
- Drugs and alcohol
- Lack of acclimatization

Living conditions
- Lack of access to air conditioning
- Urban
- Low income
- Social isolation

Prolonged outdoor activities
- Agricultural workers
- Runners
- Child/adolescent athletes
- Laborers
- Undocumented border crossers

TABLE 2.
Therapeutic and Recreational Drugs That May Contribute to Heat Illness—and Their Mechanisms[9,10]

Drug category	Relevant mechanism
Diuretic (e.g., alcohol [ADH suppression], furosemide, HCTZ)	Deplete intravascular volume
Cardiovascular (e.g., beta blockers, calcium channel blockers)	Decreased cardiac output and vascular resistance impairs cutaneous vasodilation
Anticholinergic (e.g., antihistamines, Parkinsonian medications, atropine/ scopolamine, tricyclic antidepressants, neuroleptics)	Inhibit sweating
Sympathomimetic (e.g., cocaine, amphetamines, ephedrine/ pseudoephedrine)	Vasoconstriction, increased muscle activity, increased metabolic rate
Neuroleptic (e.g., phenothiazines, thioxanthenes, butyrophenones)	Inhibit sweating (anticholinergic); inhibit hypothalamus-directed vasodilatation

sodilatation, sweating or sweat evaporation, electrolyte balance, or normal behavioral response can impair temperature homeostasis.

Acclimatization requires one hour daily of moderate exercise for 10 to 14 days in conditions of heat stress.[5] Acclimatized persons have increased sweat gland numbers, sweat volume, and salt reabsorption, leading to enhanced ability to dissipate heat by evaporation, reduced hyponatremia, and increased total body water (TBW). Increased TBW allows increased cardiac output. Dehydration causes a reduction in available cardiac output, and greatly reduces the benefits of acclimitization.[3]

Epidemiology

Heat illnesses are a growing concern. It is estimated that there are 60,000 presentations for heat-related illness a year in the U.S., with an average of 688 deaths per year.[6,7] This figure does not include the deaths of undocumented migrants crossing the border between the U.S. and Mexico, which between 2002 and 2003 was estimated to be 409 persons, just for the border section between Yuma, AZ and El Paso, TX.[8]

Several populations have increased risk of heat illness (**Table 1**). The elderly and the very young, patients with comorbidities, patients taking medications or drugs that interfere with heat homeostasis (**Table 2**), and persons with limited cognitive ability, low socioeconomic status, and mental illness are at elevated risk. Relatively young adults such as athletes, laborers, and border crossers are susceptible during high exertional states, even in moderate temperatures.

Diagnosis and Management

Key elements of the history include exposure, acclimatization status, comorbidities, drug and alcohol history, and a medication history (**Table 3**). Knowledge of patients' social resources is crucial for disposition decisions and preventative counseling.

The physical examination should focus on vital signs, general appearance and mental status, hydration status, skin condition, and the cardiovascular and nervous systems.

Vital signs, including orthostatics to assess volume status, help sort patients into those with relatively minor versus major heat illnesses.

Skin conditions, including heat rash and sunburn, interfere with sweating and evaporative heat losses.[11]

Anhidrosis should be assessed in the axilla, as forehead sweating is an unreliable marker.[5]

Hydration can be assessed from pulse and blood pressure, fontanel, oropharynx mucous membranes, lacrimation, sweating, active vomiting or diarrhea, and urine color and output.

Cardiovascular examination should focus on murmurs, especially in exertion-related syncope, and heart failure signs. In exertional heat illness, it is important to assess for muscle tenderness, considering rhabdomyolysis.

Altered mental status or an abnormal neurologic examination should prompt immediate transfer to a higher level of care, even if the patient's temperature is normal (**Table 4**).

TABLE 3.
Key Elements of History

HPI	Chief complaint, length of exposure to heat stress, exertion, hydration
PMH	Comorbidities, especially cardiac and mental health, acclimatization status, mobility, medications, drugs and alcohol, social support, access to air conditioning
Associated symptoms and ROS	Fainting, weakness, lightheadedness, thirst, nausea, vomiting, diarrhea, urine output and color, muscle cramps and myalgias, headache, seizure, behavior change, rash, sunburn, sweating

TABLE 4.
Key Elements of Physical Examination

Vital signs	Temperature (preferably rectal) Pulse Respiratory rate Blood pressure and orthostatics Pulse oximetry Random fingerstick blood glucose – Diabetics – Suspected altered mental status
General	Mental status, body habitus, ill or not ill, motor activity level
Skin	Color and perfusion, pallor, cyanosis Diaphoresis, axillary anhidrosis miliary rash, sunburn
HEENT	Fontanel level, tears, oropharynx hydration
Pulmonary	Tachypnea, apnea
Cardiovascular	Tachycardia, bradycardia, dysrhythmias, murmur, heart sounds
Abdomen	Ability to tolerate oral fluids, vomiting, diarrhea
GU	Urine color, output
Musculoskeletal	Muscle tenderness or spasm, edema
Neurological	Cognitive ability, focal neurological deficits
Psychiatric	Reality testing, bizarre affect

Heat Syndromes

There are several common entities seen in urgent care centers related to heat exposure, ranging from minor annoyances to higher risk for significant morbidity. These include heat rash, heat edema and heat syncope, heat cramps, and heat exhaustion (**Table 5**).

Heat Rash

Heat rash, also known as prickly heat, lichen tropicus, and miliaria rubra, occurs when sweat ducts are blocked by dead skin. Tiny vesicles form as sweat accumulates under the skin, resulting in pruritis. Chronic vesicles can rupture into the surrounding tissue, causing skin thickening and scarring. Secondary *Staphylococcus* and *Streptococcus* infections can occur.

Treatment includes antihistamines for pruritis, cool baths, and time in a cool environment. Chronic heat rash will need dermatologic follow-up for treatment with salicylate gels to avoid scarring.[7]

Heat Edema

Heat edema occurs mostly in older individuals who are adjusting to an increased heat strain. Vasodilatation, in combination with relative venous stasis, causes blood pooling. No dehydration or salt imbalance is usually present, and diuretics are not warranted. This must be distinguished from more worrisome causes, including deep vein thrombosis (DVT), nephrotic syndrome, liver failure, and congestive heart failure. Heat edema is a be-

TABLE 5.
Heat Stress Syndromes[7,10,12]

	Heat rash	Heat edema	Heat syncope	Heat cramps	Heat exhaustion	Heat stroke
Mechanism	Macerated skin blocks sweat glands, vesicles form Chronic rash when vesicles rupture deep	Pooling of blood with vasodilatation, venous stasis	Vasodilatation and lack of venous return from posture cause transient hypotension	Unclear: Loss of sodium in sweat Dehydration Plain water hydration Spinal reflex[14]	Prolonged exposure over hours or days to heat stress, dehydration Exacerbated by exertion	Heat stress from internal and external sources overwhelms ability of body to dissipate heat; leads to shock
History	Lack of rest in cool environment, sweating	Elderly, not acclimatized	Prolonged standing in heat Responds immediately to lowered posture, cooling	Painful spasm in exerted muscle groups, during or hours after exercise Previous history, not acclimatized, drinking water only, anorexia	Lack of access to cooling Exertion Nonspecific: Headache, malaise, dizziness, nausea, vomiting, irritability	Risk factors AMS Similar to heat exhaustion
Signs	Vesicles Erythematous Pruritic Chronic lichenification scarring	Pitting or nonpitting edema of lower extremities, usually not beyond ankles Hands may be edematous	Transient hypotension Responds rapidly to rest, cooling	Spasm, fasciculation in large muscle groups, usually flexor	Diaphoresis Tachycardia, tachypnea Normal or elevated core temperature	AMS Core temperature >39.5 C Hypotension
Laboratory tests	None	None	None routinely Elderly/comorbid/ exerting need EKG, labs, imaging appropriate for syncope work-up	Electrolytes often normal Hyponatremia Hypokalemia Creatinine kinase	Electrolytes Renal function Creatinine kinase Liver function Coagulation studies	Transfer to higher level of care for extensive laboratory tests
Treatment	Antihistamines Cool environment	Elevation Compression stockings	Fluids Rest Cool environment Acclimatization	Fluids Pain control	Fluids Rest Cool environment Acclimatization Protect from re-exposure	ABCs Begin cooling/hydrating, air conditioning, fan water spray
Concerning differential diagnosis	Secondary infection Heat exhaustion	CHF DVT Liver disease Nephrotic syndrome	Hypertrophic heart ACS Dysrhythmias Stroke Seizure Heat stroke	Rhabdomyolysis Heat exhaustion	Heat stroke: AMS Hypotension Core temperture >39.5° C Rhabdomyolysis Septic shock	Neuroleptic malignant syndrome Serotonin syndrome
Red flags	Inhibits sweating	Should not extend above ankles		Ask about eating disorders	Altered mental status	
Disposition	*Discharge:* Follow up dermatology if chronic	Discharge	*Admit:* Any concern for serious cause *Discharge:* Simple heat syncope *Follow-up:* Cardiology for IHC	*Discharge* Avoid exertion for 1-3days Must re-acclimate	*Admit:* Risk factors No access to air conditioning Not responding to treatment *Discharge:* Responds to cooling and fluids	Medical emergency Transfer to a higher level of care immediately

nign condition that is self-limiting, and may be treated with elevation and compression stockings. Follow-up with a primary care provider is recommended in seven to 10 days (or sooner if the condition does not improve with conservative measures).

Heat Syncope

Heat syncope occurs when peripheral vasodilatation and impedance to venous return caused by posture combine to lower the blood pressure enough to interrupt cerebral blood flow. Classically, military personnel standing at attention with locked knees are at risk.

Persons usually recover promptly with lowered head, elevated lower extremities, and a cooler environment.

Differential diagnosis for syncope includes concerning entitities such as idiopathic hypertrophic cardiomyopathy, dysrhythmias, acute coronary syndrome, and cerebral vascular accident.

In a young, non-exerting, otherwise healthy individual who responds to appropriate therapy, disposition may include discharge home. However, advanced age, comorbidities, and a history of exertion at time of syncope re-

quire further testing. In young patients with exertional syncope, restriction on activity level pending a referral to a cardiologist for further work-up is appropriate. Aggressive search for serious disease must be made in older patients and patients with comorbidities, and admission or transfer to a higher level of care is appropriate.

Heat Cramps

Heat cramps are defined as motor unit hyperactivity in major muscle groups, usually thigh or leg, during or several hours after prolonged exertion under heat stress.

The exact mechanism is not clear. It was originally thought that hyponatremia due to excess sweating and hydrating with water caused heat cramps. However, heat cramps can occur before any rehydration has taken place, and many patients with heat cramps have no electrolyte or serum osmolarity derangements. A spinal reflex caused by overexertion has also been proposed as a mechanism.[13]

The two most useful prophylactic steps to prevent heat cramps are heat acclimatization and consuming adequate water during exercise. Relative muscle dehydration appears before the subject experiences thirst, so the advice should be to consume water at regular intervals during exercise even if the athlete does not feel thirsty.

Treatment for heat cramps includes rehydration with an oral salt solution or IV normal saline, as well as pain control, which may require narcotics. Electrolytes should be checked and replaced as needed, and creatinine kinase levels measured to rule out rhabdomyolysis. Typically, heat cramps respond rapidly to treatment, rarely lasting for more than 15 minutes during a flare-up. They can produce agonizing spasms during a flare-up, and can recur several times over the next 24 to 48 hours. During the recovery period, the patient should avoid exertion since the spasms can be triggered by a normal muscular contraction. Unfortunately, they can be triggered during sleep, and awaken the patient with severe pain. The painful contractions are usually in the flexor muscles, and hyperextension of the involved muscle may overcome the spasm. When they involve the hamstring flexors, extension of the hip and knee are useful, as is standing up, and slow mild pacing.

Icing the involved muscles may provide the patient with pain relief, and mild analgesics are useful when the cramps subside.

In the elderly patient who is perhaps already on a diuretic agent for hypertension, the cramps are worsened

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by any potassium imbalance. Many of these patients will obtain relief from potassium oral supplementation even when the serum electrolyte level of potassium is normal.

Heat Exhaustion

Heat exhaustion is generally a result of prolonged exertion or prolonged exposure to a higher heat index than normal. Symptoms are nonspecific, and can include any of the above syndromes, as well as lightheadedness, malaise, fatigue, headache, nausea, vomiting, decreased urine output, and thirst. Dehydration and electrolyte abnormalities are common but not necessary to the diagnosis, and patients with a history of exertion need to have rhabdomyolysis excluded.

Patients with heat exhaustion need to be in a cool, air conditioned environment, and inappropriate extra clothing should be removed. Hydration can usually be accomplished with oral salt solution or normal saline, with electrolyte correction as necessary. Patients should respond to cooling and hydration; any patient with persistent symptoms or comorbidities should be admitted to the hospital.

Prudent discharge requires that the patient have access to a cool, air conditioned environment for the next 48 to 72 hours, especially for those with risk factors for heat illness.

Elderly patients, patients with limited mobility, and mentally ill or retarded patients need a caretaker or family member to check on them at least twice a day during periods of higher than normal heat or humidity. Close follow-up should be arranged. Workers and athletes likewise require 48 to 72 hours of decreased activity in a cool environment, and must re-acclimate.

Heat Stroke

Heat stroke needs to be considered in patients with core temperatures above 39.5 ºC, anhidrosis, or any alteration in mental status. These patients must be immediately transferred to a higher level of care. This is a true life-threatening emergency.

Prevention

All patients with heat illness are at higher risk for relapse in the short term and for recurrence in the long term. It is important to avoid re-exposure for two to three days, as heat shock proteins and body water composition take time to equilibrate. Athletes and laborers will need to re-acclimatize after a period of rest, and cannot immediately resume their previous level of exertion.

It is appropriate to counsel *all* patients regarding heat illnesses during the summer months, regardless of their presenting complaint.

During the course of weather episodes in which the daily high temperature might exceed 90º F, or 80º F (32 ºC or 27ºC, respectively) with high humidity, urgent care physicians can reduce the burden of heat illness with brief counseling, educational handouts, and posters. Excellent patient information sources are available.[14,15]

Patients should be counseled to hydrate liberally, unless specifically contraindicated. Inactive individuals need four liters of fluids or more daily during heat waves, and the exerting adult may need up to 10 liters daily. Thirst is an unreliable indication of hydration status, as it is mainly stimulated by hypernatremia, and hydration must often be scheduled.

Exerting adults should drink 250 mL of fluid every 15 to 20 minutes during exercise, and children should drink 150 mL. It is often impossible to hydrate enough during exercise, and hydration must begin before activity and continue afterwards.

Thirst is stimulated by eating; hydration at meals in addition to during activities is necessary. Electrolyte solution is generally unnecessary for people consuming a normal diet, and has only been shown to increase exercise tolerance during the first three days of acclimitization.[3] However, if the taste is more tolerable, especially to children, this may encourage hydration. Patients not consuming a normal diet, exerting heavily for prolonged periods of time, or with gastroenteritis will benefit from electrolyte solutions, and there should be no hesitation to use intravenous fluids.

Air conditioning for as little as three hours per day is the only intervention known to be protective against heat stroke during heat waves.[16] For this reason, some urban areas have heat wave response plans that include heat shelters, such as malls. Fans have not been shown to be protective.[17]

Exertion should, if possible, occur in the early morning or late afternoon or evening, avoiding activity during the heat of the day. Outdoor laborers should be given regular rest and hydration breaks; an air conditioned rest area is protective against heat illness and maximizes exercise tolerance.

Summary

Heat illness has an unknown incidence, but heat stroke is the second largest cause of environmental or weather-caused mortality—more than hurricanes, tornados, and lightning combined, second only to hypothermia.[18] It is preventable through public health measures, education, and early intervention.

Most presentations of heat illness can be quickly and adequately treated by the urgent care provider, and serious morbidity and mortality can be avoided by attending to risk factors, excluding serious diagnoses, preventing complications, and promptly recognizing severe disease. Preventative education remains an important part of the urgent care provider's role. ■

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