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Every summer children die because of their unrecognized intolerance of heat. Their larger body surface area, lower output of sweat, and greater metabolic heat production place them at increased risk for heat-related illness. Children with special needs (e.g., cardiac disease, seizure disorders) are at increased risk of developing heat exhaustion or heatstroke. The author presents a timely and comprehensive review of heat-related illnesses that includes recognition, management, and prevention.

—The Editor

The Spectrum of Heat Illness in Children

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Introduction

For life on earth, heat is a double-edged sword. Too much or too little can be fatal. When the protections against the heat fail, heat illness develops.

The tragedy of heatstroke is that it often strikes highly motivated young individuals under the disciplines of work, training, and competition. Heatstroke is the second leading cause of death among young athletes in this country, ranking directly behind head and spinal injuries. Other high-risk groups include the chronically ill or bedridden, the mentally ill, those taking antipsychotic or anticholinergic drugs, those who work under high heat loads, and alcoholics.[1] Though heat illnesses are eminently preventable, thousands of people continue to suffer from them each year.[2,3,4]

The Spectrum of Heat Emergencies

Why some children develop heat illnesses and others do not is not yet known, but the operating factors of high temperature, increased metabolic oxygen demands, and relative hypoxia seem to define the damaging factors. Even the healthiest of children can experience heat illness under the right circumstances.

The clinical picture of heat illness is a spectrum ranging from the fatalities and multisystem morbidity of heatstroke to the faints of heat syncope or the pains of heat cramps. In this continuum, heat dissipation reflexes may or may not be lost, depending upon the severity of the disease.

No single identified environmental pattern consistently provokes heatstroke, although most epidemics have occurred when the temperature exceeds 32°C, with relative humidity greater than 50% for two or more days. Under normal conditions, heat is lost from the body by radiation, convection, conduction, and evaporation. The heat control center, located in the hypothalamus, is responsible for overall body heat regulation. The ability of the body to lose heat is primarily dependent on sweating when the ambient air temperature increases. The capacity for sweating also is determined in part by relative humidity of the environment and wind velocity. Thus, the combination of high temperature, humidity, and inadequate ventilation provides the basic conditions for heat illness. However, factors such as conditioning, medications, underlying disease, or previous history of heatstroke play a predictive role in identifying patients at highest risk.

The prevalence of the more minor heat illnesses is unknown and, quite possibly, unknowable. Certainly every exercising young athlete at one time or another has suffered heat cramps and may have felt lightheaded and dizzy after severe exercise in the heat. No one has counted the number of young marching band members who have collapsed during summer practices.

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How Hot is Too Hot?

Some physicians and many laymen feel that there is a set maximum core temperature beyond which heat illness or tissue damage is inevitable. Strangely, difficulty arises in defining exactly when the body temperature is "too high." Young athletes who are in good shape have had measured core temperatures of 41°C and beyond at routine (for them) exercise. One study reported the change of rectal temperatures in eight highly acclimatized Israeli soldiers while carrying loads of 35 kg during a desert march.[8] Five of the eight had rectal temperatures ranging from 41.5°C to 42.4°C upon completion of the march.

Some physicians would consider a patient with such temperatures to have suffered heatstroke by virtue of the measured temperature. These Israeli athletes required no therapy other than oral replenishment of water and electrolytes. The soldiers also suffered no worse central nervous system effects than euphoria when the march was finished.

Other authorities feel that the maximum temperature is less important in a definition of heatstroke. They will include not only the maximum temperature but also the time spent at that temperature.[5,6]

Where hyperthermia and tissue "cooking" have been blamed for all of the damage of heatstroke, volunteers and patients treated for whole body hyperthermia have easily shown that rectal temperatures of 42°C have been endured without any injury. Heatstroke, on the other hand, has been observed at temperatures of only 40°C—a temperature seen in every emergency department (ED) almost every day. Moreover, tissue injury from heatstroke has been shown to continue in about 25% of patients after cooling to a normal body temperature.[7,8]

One hypothesis is that cellular metabolism increases about 13% for each centigrade degree rise in temperature (van Hofft's law). At 40.5°C, the cellular metabolism is 50% above normal. As the core temperature rises, if the oxygen supply does not keep pace with the intracellular needs, the cells will become hypoxic and begin to die. In a conditioned athlete, the heat is dissipated well, and the heart and cardiovascular-vascular system can provide for the cardiovascular load of the heat dissipation, supply necessary oxygen and metabolic substrates, and meet the circulation needs of the exercise. In the poorly conditioned, chronically ill, dehydrated, or the very young, the cardiovascular system is not able to provide for the needs of both circulation and cooling. As the temperature rises and the duration of exposure increases, even the fit and conditioned athlete will experience cardiovascular failure at some point. When this happens, the cardiovascular system is unable to provide both adequate circulation and continue cooling efforts. The result is multi-system breakdown with diffuse cellular death. The cellular breakdown appears to be responsible for some of the delayed morbidity in those patients who survive the emergent phase.

Certain mediators may increase organ damage, including endotoxin, cytokine levels, activated coagulation components, and activated or injured endothelium. In studies, plasma concentrations of tumor necrosis factor (TNF-α), interleukin 1a (IL-la), and lipopolysaccharide (LPS) were elevated in patients with classical heatstroke. The concentrations decreased significantly after cooling but remained above the control values.[9,10] These cellular mediators are being explored, and their roles are not yet clear.[10]

Predisposing Factors

Man is superbly adapted to a hot climate, with the ability to shed large amounts of excess heat efficiently. If the exogenous burden of heat is too great, if endogenous production is too large, or if other factors supervene, then our ability to deal with excess heat will fail. If the exposure is too great, all will have failure of heat regulation; if it is marginal, only a few individuals will experience the failure of the heat elimination processes. A discussion of the factors that predispose to cooling system breakdown follows. *(See Table 1.)*

Exogenous Heat Gain from the Climate

Severe climactic conditions, particularly those with high humidity without wind, predispose to heatstroke. These severe conditions may be due to a generalized climactic change or to the change in a small, local environment (a "microclimate").

Generalized climactic changes (heat waves) are well known to predispose to heatstroke in epidemic proportions. Less well appreciated are the effects of a microclimate on individuals. Incarceration in "The Black Hole of Calcutta," working in mines, around furnaces or boilers, and other similar unusual circumstances, are

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Table 1. Clinical Characteristics of Heatstroke

	CLASSIC	EXERTIONAL
Age group affected	Old/very young	Young/fit
Occur in epidemics	Yes (heat waves)	Yes (group athletes)
Predisposing illness	Yes	No
Prevailing weather	Heat wave	Variable
Sweating	Often absent	Often present
Acid/base change acidosis	Alkalosis	Metabolic acidosis
Alkalosis	Respiratory	Respiratory
Rhabdomyolysis	Uncommon	Common
Acute renal failure	Rare	Common
DIC	Rare	Common
CPK elevation	Mild	Marked
Major organ system Renal/muscle/hematologic	CNS	—
Lactate	Usually low	Usually high

associated with markedly increased incidence of heatstroke and other heat illnesses. These hot and often humid microclimates provide abundant heat gain and little opportunity for heat losses.

Deaths have also been reported in children from saunas, steam rooms, whirlpool baths, and hot tubs. It has been thought that the resulting severe hyperthermia has been induced by the rapid conduction of heat by the water. The peripheral vasodilation, erect posture, and alcohol ingestion that may often accompany the use of hot tubs and saunas may aggravate the hyperthermia. Indeed, some of our "hot tub drownings" may actually have been heat-stress related.

Another special microclimate is the unprotected hot car. Temperatures in automobiles left exposed to the sun can reach up to 60°C. Children (or pets) left in this environment are rapidly overwhelmed by the heat exposure and often die.[11]

There is a very strong inverse relationship between daily hours of home air conditioning and classic heat illnesses.[12,13] Indeed, during the 1980 heat wave, the CDC estimated the risk of dying from heatstroke to be 49 times greater for those persons without home air conditioning than those with 24-hour-a-day air conditioning. The same investigators found that merely spending about two hours per day in an air-conditioned place such as a shopping mall would also decrease the risk of heatstroke during a heat wave.

Increased Endogenous Heat Production

Any factor that increases the amount of body heat to a point beyond the capacity of the body to eliminate it will eventually produce heat illnesses.

Exercise

Extremes of exercise under hot environmental conditions may lead to heatstroke or other heat illness even in normal persons. Core body heat gains of 1°C per 5 minutes are possible with strenuous exercise if the microclimate precludes heat losses. It should be noted that seizures, delirium tremens, combative behavior, and straining at restraints all qualify as extremes of exercise. Although not customarily listed as exercise, vigorous muscular contractions in these activities may produce just as much heat as the marathon runner's exercise.

Febrile Illness

Any child with a febrile illness has a greater chance of developing a heat illness during hot, humid weather. Compared to peers, the febrile patient has an increased metabolic rate that produces an even greater heat burden. Often this heat burden may be combined with the effects of the illnesses, such as vomiting or diarrhea, which may lead to dehydration, or pulmonary dysfunction, which may decrease oxygen intake or cause dermatitis, which prevents effective sweating.

Drugs

Drugs such as cocaine, phencyclidine hydrochloride (PCP), and lysergic acid diethylamide (LSD) that cause a hyperactive state and increase the body's metabolic rate may predispose the patient to development of heatstroke. Needless to say, it does not matter whether the drug causes the hyperactivity by direct action, such as strychnine, or by indirect central nervous system (CNS) effects, such as PCP.

Tricyclic antidepressants and amphetamines can also boost heat production by both stimulating the hypothalamus and increasing muscle activity. Several reports have implicated amphetamines and cocaine in the pathogenesis of hyperthermia in association with massive rhabdomyolysis. The threshold of damage may be easily surpassed when one is under the influence of a drug that blunts the recognition of fatigue and increases the motor activity.

Impairment of Heat Dissipation

Clothing. Heat-retaining clothing will cause the effective absence of sweating as a cooling force by complete inhibition of evaporation, in the most extreme case. Fatal heatstroke has occurred in men exercising in plastic sweat suits, even when the temperature is as low as 26.6°C. Multilayer, occlusive, protective garments (chemical or fire protection) can cause heatstroke even with short exposures. Young wrestlers who are trying to make weight limits prior to a match have died due to the combination of occlusive garments and heat stress.

Obesity. Overweight children are less heat tolerant than children of normal weight.[14] During strenuous exercise, they are at a much higher risk for heat illness. It is not apparent whether the obesity or the accompanying lack of physical conditioning is the problem. Certainly, fat is an insulator and decreases heat loss to the environment. The obese child may also be unable to attain the cardiovascular output necessary for effective cooling because of lack of conditioning.

Dehydration. Dehydration blunts the response to heat stress in at least two ways. First, the depleted blood volume means that less fluid can be lost as sweat before terminal reflexes decrease perspiration. Second, the lower blood volume means that the cardiovascular system can less easily afford to shunt blood to the periphery to dump heat by radiation.

Progressive dehydration may occur during a long run or other prolonged vigorous exercise, even though the athlete drinks during the exercise. Hot, exercising athletes simply do not ade-

quately rehydrate themselves voluntarily. Thirst is not a reliable indicator of dehydration; children must be made to "over drink."

Age. Certain characteristics of children make them less efficient at thermoregulation than adults. Heat intolerance in children exercising in hot areas is attributable to greater surface-area-to-mass ratios, lower sweating rates, and greater metabolic heat production.[30]

Their larger body surface area per kilogram promotes greater heat exchange between the skin and the environment. If the ambient temperature exceeds skin temperature, the net result is heat gain.

Children sweat less than adults due to a lower output of each sweat gland. In children, the sweating set point (i.e., the change in rectal temperature at which sweating starts) is higher.

Children produce more metabolic heat per kilogram of body weight at a given speed of walking or running than adolescents or adults. This places a greater strain on thermoregulatory mechanisms. The child's lower cardiac output at any given metabolic level places them at risk when exercising in the heat. A decreased blood supply to the skin means less convection of heat from the body core to the periphery.

These characteristics are accentuated when the child is exposed to extreme weather conditions or has underlying dehydration. Hyperthermia during extreme hot weather is particularly likely to develop in children with vomiting or diarrhea, respiratory tract infections, or neurologic illness.[15,16]

Chronic Disabling Diseases. Chronic disabling diseases of virtually any form will increase the risk of heatstroke. Important among these diseases are diabetes, cardiovascular disease, malnutrition, and disorders that impair the sweating responses.

Skin Diseases. Any skin disease that will impair sweating will decrease the ability of the body to compensate for a heat load. This not only includes such diseases as scleroderma and extensive burn scarring, but also new burns and diaper rash. Extensive sunburn will also destroy the ability to sweat.

Drugs. There are five major mechanisms by which drugs may affect the ability to dissipate heat to control the body temperature:

1. Depression of the thermoregulatory center of the anterior hypothalamus can affect the control of the body temperature "set point" probably through interference with or by depletion of dopamine. (Phenothiazines are prime examples of drugs with this effect.)[17]
2. Anticholinergic agents can block the sweating response and affect the body's ability to maintain normal body temperature by inhibition of sweating. (Atropine, tricyclic antidepressants, and a large group of other medications have some effects here.)[18]
3. Any drug that causes cutaneous vaso-constriction will markedly decrease the body's ability to lose heat. These medications include the sympathomimetics (e.g., norepinephrine or pseudoephedrine).
4. Diuretics may cause relative dehydration by decreasing total body water stores. If the patient is dehydrated, sweating may be diminished or abolished, and cardiovascular responses to heat are markedly hampered.
5. If the cardiovascular responses to heat stress are blocked, the body is less able to tolerate increased heat. If beta blockers or calcium channel blockers are prescribed, the diminished cardiovascular response to stress puts the patient at greater risk from heat stress.

Additionally, the diagnosis of heatstroke may be confused with neuroleptic malignant syndrome. This syndrome has been associated with the high-potency antipsychotic drugs, such as haloperidol or fluphenazine. Muscular rigidity, altered consciousness, autonomic instability, and high temperatures characterize the neuroleptic malignant syndrome. It should be considered in anyone taking antipsychotic drugs whether or not there are environmental heat stresses. Although the syndrome may occur on hot days, it has not been associated with high environmental temperatures. Core body temperatures higher than 41°C have not been reported.

Miscellaneous Factors

Prior Heatstroke. Current evidence suggests that those persons who have had a prior history of heatstroke are at an increased risk of another attack. The feeling is that the genetic or physiologic predisposition of the person is the reason for this increased incidence and not some damage induced by the prior heatstroke.

Prior malignant hyperthermia or neuroleptic malignant syndrome may predispose to heatstroke. These reports, however, are scanty and lack pathologic confirmation of the underlying diseases.

Minor Heat Emergencies. If the rate of heat gain exceeds the heat loss by only a small amount, the heat loss mechanisms may not be overwhelmed but may not completely protect the patient. This leads to a minor heat emergency.

Heat Edema

Swelling of the feet and ankles is often reported during the summer months. This heat edema is worse during the first few days of heat exposure and is often asymptomatic.

The edema is usually minimal, is not accompanied by any significant impairment in function, and often resolves after several days of acclimatization. It is presumed that cutaneous and muscular vasodilatation, combined with venous stasis, lead to a vascular leak and accumulation of interstitial fluid in the lower extremities.

The most important reason to be aware of this clinical syndrome is to prevent overly vigorous treatment in patients with this problem. For heat edema, brief diagnostic evaluation is in order, but invasive diagnostic or therapeutic intervention is clearly inappropriate for this self-limited disease. A characteristic history and otherwise normal examination is usually all the diagnostic work-up requires.

Therapy consists of reassurance. There are no studies to support or refute the use of mild diuretics if the edema is bothersome. Certainly, if diuretics are administered, strict attention should be paid to fluid status, and dehydration must be prevented.

Accurate and detailed assessment of this problem's pathophysiology has not been established (and may not be needed), as the problem resolves without any therapy.

Heat Cramps

Heat cramps are painful, involuntary spasms of major muscles used in intense exercise. They usually occur in unacclimated persons and are more common after profuse sweating. Body temperature is usually normal with heat cramps; other, minor heat syndromes may accompany the heat cramps. Fortunately, heat cramps are limited both in duration and morbidity.

The cause of any cramp is usually not clear, and heat-related cramps are no exception. Heat cramps are common in hot weather and are believed to be from the sodium and water losses described above. The cramps are presumably due to a rapid

change in the extracellular osmolality. Other factors, such as alkalosis, which decreases the ionized calcium, or diuresis, which causes a loss of magnesium or calcium, may play a role.

Massaging and stretching will help to alleviate the pain and may allow the athlete to resume competition. Muscle cramps also can be treated with simple oral fluid replacement. In severe cases, intravenous fluid replacement and judicious doses of diazepam may be useful. Magnesium sulfate and calcium chloride have been considered, but there is no study to refute or support their use.

Heat Syncope

If we force a person to stand for prolonged periods of time in one position or strap them to a tilt table, we can cause almost any person to faint. High temperatures and vasodilation potentiate this loss of normal vasomotor control. Dehydration due to sweating will further decrease the blood return to the peripheral circulation. The result of these three mechanisms is the heat syncope. The young marching band member in a parade on a sweltering Memorial Day is an ideal candidate for this form of heat illness.

Treatment. Treatment of heat syncope is simple and quite physiologic. Remove the child from the heat stress. Keep the child in a recumbent position. The child should receive oral fluids and be encouraged to avoid prolonged standing in the near future.

If the child does not recover from the faint promptly, then transport to a medical facility is indicated. If many children or adults in a group start to suffer from heat syncope, the group leader should recognize that the necessary conditions for production of heatstroke are present and reduce the activity accordingly.

Prevention. Patients should be instructed about the cause of the syncope and how to prevent repeated episodes. An appreciable amount of venous circulation can be maintained with minimal knee bending or isometric maneuvers when at the attention position.

If these maneuvers fail, or if the patient is not in parade type activities, he or she should be encouraged to assume a horizontal or head between the knees position whenever he or she recognizes the symptoms of this postural syncope.

Heat Exhaustion

Heat exhaustion is an intermediate step from the minor heat illnesses to the overwhelming catastrophe of heatstroke. In terms of morbidity, it falls midway between the previously described disorders and heatstroke. Generally, heat exhaustion is thought to involve reversible heat overload, while heatstroke has irreversible damage to tissues. Unfortunately, the signs and symptoms of early heatstroke, particularly exercise-related heatstroke have considerable overlap with the signs and symptoms of heat exhaustion. The low mortality of heat exhaustion may represent these misdiagnoses.

Heat exhaustion originally was thought to represent either a water or salt depletion secondary to sweating; neither assertion has been conclusively proven. Certainly, acute dehydration is responsible for many symptoms similar to heat exhaustion, but is not a reliable finding. A more constant definition of heat exhaustion is overwhelming of the heat compensation mechanisms without tissue damage or permanent sequelae. Heatstroke would then include all cases of heat illness that cause permanent damage to end organs such as the liver, heart, kidneys, or CNS.

Heat exhaustion is considered by most authorities to be a precursor to heatstroke and, thus, warrants vigorous treatment. Certainly, the climate where heat exhaustion is found is also clearly conducive to heatstroke. If multiple people in an area start to have symptoms, suspicions for heatstroke should be markedly raised. Coaches, trainers, or group leaders should decrease or postpone training and increase water and rest breaks.

Making the Diagnosis. Vague symptoms and signs make heat exhaustion a diagnosis of exclusion. In addition to the temperature elevation, patients often have nausea, vomiting, and intense fatigue. Hyperventilation is typical. Identification of a precipitating exposure to heat and exclusion of other underlying illnesses are essential to the diagnosis.

Although measurement of rectal temperature has been touted as the only reliable way to differentiate heat exhaustion from heatstroke, it is actually quite unreliable. As previously noted, normal marathon runners may have exercise-related temperature elevations well in excess of 40°C. If the patient has just stopped intense activity, the temperature may transiently exceed 41.1°C.

The neurological picture should be relatively normal. Profound, disturbing mental status changes should not be found, although the patient may complain of a headache or be mildly confused, dizzy, or weak. If a patient with heat exhaustion loses consciousness, heatstroke is a distinct possibility and needs to be aggressively pursued.

Since children with exertional heatstroke are often sweating, skin moisture and skin color are also poor ways to differentiate heatstroke from heat exhaustion. The classic hot, dry skin found in heatstroke is found only in *classic* heatstroke.

The only reliable way to differentiate heat exhaustion from heatstroke is to measure enzymes that illustrate either muscle or liver damage. CPK, LDH, and SGOT elevations should prompt the clinician to consider that heatstroke has occurred. Unfortunately, these laboratory tests are not available instantaneously. In all cases of heatstroke, rapid treatment is necessary to prevent progression of damage, and one should not wait for confirmation from diagnostic tests.

Treatment. The severity of the patient's symptoms dictates the nature and rapidity of the therapy. The amount of fluid replacement required depends on the degree of hypovolemia. In mild cases, repletion with oral fluids such as water, diluted orange juice, or any of the sport drinks (e.g., Gatorade or Exceed) is appropriate. More serious cases require fluid repletion with intravenous fluids.

Resting in a cool, shaded place is essential. External cooling is helpful, and either evaporation or ice may be used. If the patient's temperature remains elevated, then tissue damage can be presumed to be progressive.

Patients should be observed for any of the signs of heatstroke while waiting for appropriate laboratory data. Formerly healthy patients should be asymptomatic within 12 hours and can be discharged. If the patient remains symptomatic or vital signs remain abnormal after 2-3 hours, the patient should be admitted to the hospital. In younger children without a clear history of heat exposure, a septic workup is always indicated in the face of a high temperature.

It is my firm opinion that all patients with heat exhaustion should be treated in the field as if they have early and potentially progressive heatstroke. Since the current definition is retrospective, and it is quite difficult to ascertain tissue damage in the field, medical providers should presume the worst and treat accordingly.

Heatstroke

Heatstroke occurs when the body heat loss mechanisms are either overwhelmed or are insufficient to meet environmental demands and generalized tissue damage results.

Heatstroke Variants. There are two distinct, major variants of heatstroke: classic heatstroke and exertional heatstroke. Unfortunately, symptoms, signs, and clinical milieu may overlap.

Problems in Diagnosis. For years, it was taught that heatstroke had a classic triad—coma, hot, dry skin, and temperature higher than 41°C. This classic triad leads to substantial underdiagnosis. The diagnosis of heatstroke calls for a high index of suspicion before all of the facts may be available.

It is important to note that an elevated body temperature should not be the only test that determines the disease nor is it necessary for a diagnosis. As noted earlier, there are several studies in the literature of body temperatures as high as 42°C in young athletes engaged in vigorous activity. Likewise, bystanders may have cooled a patient, and the patient now has a temperature lower than 41°C.

It is also important to note that patients with heatstroke may have either dry skin or be moist and sweaty. Older texts emphasized the hot and dry skin of the dehydrated classic heatstroke victim. As multiple coaches, athletic trainers, and drill sergeants will attest, this simply isn't true for exertional heatstroke. Hot and dry skin does not rule out heatstroke, especially in young, healthy adults.[19-23]

The diagnosis of heatstroke must be suspected whenever alteration or loss of consciousness (including bizarre behavior) occurs under any possible condition of heat load. Any patient with an alteration of consciousness and with exposure to heat stress should be considered to have heatstroke and evaluated accordingly. Core temperature should be taken rectally, preferably with a continuously reading thermometer. Any hyperthermia should be immediately treated and the patient investigated for multiple system involvement.

Classic Heatstroke

Classic heatstroke presents with coma, elevated temperature, and hot, dry skin. Clinical descriptions of classical heatstroke are based on observations made about clusters of cases during heat waves.[24] The population at most risk includes the chronically ill and those at the extremes of age. A smaller subset of cases may be found in those taking major tranquilizers and/or alcohol. A point to be strongly emphasized in the etiology of this form of heatstroke is that the ambient temperature and humidity are often high and have been at sustained highs, night and day, for several days. Because the heat wave will affect an entire city or area, classic heatstroke frequently occurs in epidemics.

The typical pediatric victim, after being subjected to this heat stress for several days, may become dehydrated and then start to show symptoms of confusion, delirium, or lethargy. Initially, young children may not be recognized as ill. After 2-3 days of unrelieved heat stress, the child may lapse into a coma. If still unattended, he or she may die in bed. This is not an uncommon presentation for excess deaths during a heat wave and may cause underreporting of actual heatstroke mortalities.

Alternatively, the child may be rapidly overwhelmed by the heat load and succumb. This occurs when the child is left in an unventilated car or a non air-conditioned, top-level apartment, for example.

Exertional Heatstroke

Exertional heatstroke is found in those who are exercising in the heat. It usually presents as an acute derangement of mental function. Incoherent speech, confusion, delirium, seizures, decerebrate posturing, transient hemiplegia, and frank coma may all be presenting symptoms. Initial symptoms may go unnoticed, with the victim feeling increasingly hot and noting dizziness or a decrease in concentration. Many athletes are familiar with these symptoms but think that they are the normal result of exertion. Other, early neurologic warning signs include irritability, confusion, agitation, or incoherent speech. These previously healthy patients also may have no prodrome or warning symptoms before collapse. Since sporting events frequently are sites of intense competition and consequent exertion, many simultaneous cases of exertional heatstroke may be seen in summer runs, football practices, and similar sporting events.

Complications. The clinical and metabolic consequences of heatstroke, which include hypotension, disseminated intravascular coagulopathies, lactic acidosis, lung injury, and hyperglycemia are very similar to the syndrome of septic shock. This similarity is furthered by the finding of circulating endotoxins in heatstroke patients and in primate models of heatstroke.

Hypotension. Patients with heatstroke of either variety will have tachycardia due to the shunting of blood through dilated skin vessels. Hypotension and shock are also early manifestations of heatstroke. The mechanism of the hypotension is complex and probably due to three or four factors that simultaneously combine to present a multifactorial but ubiquitous picture of hypotension and shock.

Hypovolemia. Hypovolemia is often present. With the rapid loss of fluid (2-3 L per hour) found in the unconditioned, unacclimatized person, large volume losses may occur more rapidly than commonly appreciated.

Peripheral Vasodilation. Peripheral vasodilation with the subsequent peripheral pooling of blood is both a mechanism of heat reduction and a source of hypotension. The shunting of blood first leads to a tachycardia as noted above. After the heart is unable to further compensate for the apparent decrease in blood volume, a high output failure intervenes with subsequent hypotension. This hyperdynamic state resembles that seen with sepsis. At least one study has noted that older patients who have had symptoms for less than six hours have had a hyperdynamic state (high output cardiac failure), while the patients with symptoms for more than 12 hours have had a hypodynamic state (low output cardiac failure).[25] The author of that article feels that the spectrum of circulatory responses to heatstroke is more a function of the patient's cardiac reserves than age alone. Younger patients with exertional heatstroke do not usually have low output heart failure.

Cardiac Dysfunction. Cardiac dysfunction is a major component of the hypotension of heatstroke. Myocardial infarction (MI) and heart failure are repeatedly reported from heatstroke, even in the very young. Vascular obstruction is usually not the cause of the MI. Though the mechanisms involved in the genesis of myocardial infarctions with heatstroke are not completely understood, hypoxia caused by the increased metabolic demands is a likely cause. Circulating endotoxins may well contribute to the cardiac dysfunction.

Disseminated Intravascular Coagulation. Clotting studies are routinely abnormal during heatstroke. Clotting disturbances may be found with decreased platelets, decreased prothrombin (PT), and decreased fibrinogen.

Disseminated intravascular coagulation (DIC) is often found

in the patient with heatstroke, and subsequent bleeding can contribute to the hypotension.[26,27] Proposed mechanisms of the bleeding disorders found with heatstroke include:

- Hepatic necrosis with subsequent impairment of the clotting factors;
- Tissue damage with subsequent release of thromboplastic substances that cause intravascular thrombosis (consumption coagulopathy);
- Vascular damage with subsequent increased capillary permeability;
- Thrombocytopenia; and
- Circulating endotoxins.

It should be emphasized that any or all of the mechanisms proposed may be operative in each case of heatstroke that is complicated by bleeding disorders. Patients with bleeding disorders differ from nonbleeders in that they appear to have higher rectal temperatures, more significant hypotension, higher incidence of shock, and a higher mortality rate.[28]

Respiratory. Hyperventilation likewise is universal with rates up to 60 per minute. This hyperventilation may cause a respiratory alkalosis that leads to hypercapnia and frank tetany.

Pulmonary edema, caused by both cardiac dysfunction and intravascular coagulopathy is common in heatstroke. Circulatory failure has also been attributed to an increase in the pulmonary vascular resistance.[29] Because of these findings, cautions about fluid administration are often noted in the literature.[30,31] Again, these studies were in older patients and may not apply to pediatric and adolescent patients. Other, more recent, investigators feel that there is no evidence for left ventricular failure. These authors administer all fluid replacements and challenges (mean fluid administration of 5719 ± 568 mL) with careful monitoring of serial pulmonary artery wedge pressures.

Renal. Renal function abnormalities are quite common in heatstroke and probably reflect the hypovolemia and hypoperfusion associated with the syndrome.[32]

Both red and white blood cells and protein are found in the urine. The urine may contain ketones and casts of white blood cells, hyaline, and granular nature. The specific gravity is usually low because of the kidney's decreased ability to concentrate the urine. The urine is sometimes "machine oil" in nature, concentrated and colored with blood and myoglobin from muscle destruction if rhabdomyolysis is present.

Unless it is transient, renal failure may require dialysis for treatment. The necessity for dialysis does not carry the ominous prognosis of chronic renal failure. Most patients will eventually recover normal or near normal renal function.

Hepatic Function. The liver is also commonly injured in heatstroke. Hepatocellular necrosis can produce marked liver enzyme and bilirubin elevations and impair the synthesis of clotting factors. These hepatocellular enzymes reflect liver cell death, which may be corroborated on biopsy or post-mortem examination. The liver is frequently enlarged and tender on examination. Frank jaundice may occur from the cell destruction and concomitant liver failure. These abnormalities, especially common in the exertional form of heatstroke, usually peak about 48-72 hours after the insult. A SGOT (AST) level above 1000 IU is predictive of a poor outcome.[33]

Musculoskeletal. Rhabdomyolysis leading to myoglobinuria is often reported and may be a significant factor in producing renal failure. Both degeneration of muscle and the increased metabolic requirements in the face of cardiovascular collapse combine to produce a profound lactic acidosis. Although conventionally taught that rhabdomyolysis commonly is seen only in exertional heatstroke, patients with classic heatstroke and rhabdomyolysis have been commonly reported in recent literature.[34]

Neurologic. As has been mentioned before, a disturbance of the central nervous system is necessary for the diagnosis of heatstroke. Confusion, irrational behavior, ataxia, or a sudden alteration or loss of consciousness are characteristic disturbances. Convulsions are common during the course of heatstroke and may be expected to occur in as many as one-third of cases.[35] Seizures that occur early, before, or during cooling, carry little prognostic import, but can increase metabolic oxygen consumption.

Seizures that occur after cooling carry an ominous prognosis. They may reflect either the effects of damage to the brain parenchyma, thrombosis, or hemorrhage. Ischemic cord lesions, diffuse brain damage, hemiplegia, and long-term ataxia have all been described as late complications of heatstroke. Prolonged coma after restoration of a normal body temperature is indicative of a poor prognosis.

Laboratory Findings

Cooling of the heatstroke victim takes precedence over all lab, x-ray, and ECG procedures. Critical treatment must not be delayed in those who have heatstroke.

Electrocardiogram Findings. Electrocardiograms (ECGs) may show some conduction changes or nonspecific ST segment changes. Frequently, changes of myocardial ischemia are reported. Although ischemic changes may be noted on an ECG, structural lesions that would compromise cardiac flow are only rarely found.

Electrolytes. Hypokalemia, hypophosphatemia, and hypocalcemia are found in heatstroke, with the specific etiology in each case not yet fully understood. Certainly, a deficit in total body potassium due to obligatory sweating losses is expected. Muscle breakdown may also cause the rapid rise of serum potassium. Potassium-containing solutions should not be employed in these patients until accurate labs are available.

If hyperkalemia is found, it must be managed urgently as it is a serious complication. Early dialysis is probably the treatment of choice, although some authors recommend Kayexalate enemas, insulin, and glucose infusions.

Hypocalcemia is most common on the second or third day following heatstroke. It is thought to be due to deposition of calcium on areas where muscle breakdown has occurred. Tetany is unusual unless bicarbonate excess has been iatrogenically induced.

Blood Sugar. Khogali noted that 70% of his patients had an abnormally high blood sugar. In the majority of these patients, there was no prior history of diabetes.[2] Prior investigators have also noted that high serum glucose measurements were common in nondiabetic heatstroke patients.[36] The etiology of this hyperglycemia is unclear. Only 30% of these patients will have sugar in the urine.

Arterial Blood Gases. Metabolic abnormalities in classic heatstroke patients commonly include a moderate hypokalemia and a mild respiratory alkalosis. Lactic acidosis is relatively uncommon and is an ominous finding.

In exertional heatstroke, metabolic acidosis is common and often severe enough to need treatment. The metabolic acidosis appears to be increased lactic acid levels due to a combination of acute hypoxemia, shock, and increased metabolic demand. Respi-

ratory alkalosis is also commonly found in conjunction with the metabolic acidosis either compensating or as part of a complex combination of metabolic acidosis and respiratory alkalosis.[36]

A possible explanation for the metabolic changes that have been seen is that the patient exposed to high heat stresses will first start to hyperventilate and develop respiratory alkalosis. If the heat exposure outstrips the ability to compensate, increased tissue oxygen consumption and relative hypoxia will follow. Lactic acid will then be produced and metabolic acidosis ensues.

Hepatic Function Tests. With the liver and muscle damage noted, the SGOT, lactate dehydrogenase (LDH), and creatinine phosphokinase (CPK) are markedly elevated on admission. These laboratory tests will often be the most sensitive indicators that the patient has had heatstroke rather than heat exhaustion.

Therapy

The treatment of heat illness is cooling. Good emergency practice dictates that life support ABCs take precedence over all other types of therapy. This axiom is particularly true because of the hypermetabolic state of heatstroke, with accelerated oxygen consumption for all tissues. The margin of safety in airway management is reduced by this increased oxygen consumption. Intubation should be considered early in the course of treatment since coma, convulsions, and vomiting are all common.

It is important to rapidly administer oxygen to the patient with very elevated temperatures. Oxygen should always be administered at the highest available concentrations through an endotracheal tube or a non-rebreathing mask.

An intravenous line must be started. Normal saline should be started initially and changed to one-half normal saline to prevent hyperosmolar changes.

Rhythm monitoring is routinely indicated since these patients have myocardial ischemia and dysrhythmias and may have frank infarction. A urinary catheter may allow monitoring of urine output and osmolarity.

A temperature probe should be inserted in the ear, the rectum, or the esophagus. Glass thermometers are to be avoided in seizing, delirious, or obtunded patients. Ordinary clinical electronic thermometers may be unable to measure temperatures above 42°C. Reflective tympanic temperatures may be used for screening.

Cooling. The primary therapy of heatstroke is cooling of the patient. The patients should be removed from the heat. Clothing should be removed, and core body cooling should be instituted. The temperature should be reduced to 39°C as quickly as possible. The more rapid the cooling, the lower the mortality that has been observed, particularly in exertional heatstroke.

Traditional Methods. Ice water soak, immersion in iced bath, ice to groin, neck, and axilla, and cooling blankets have been used by multiple centers as cooling modalities. Sheets or towels that have been soaked in ice water are an easy method to use. The patient should have all clothes removed and then be covered with towels or sheets. The towels should then be soaked in ice water and ice chips. The sheets or towels keep the ice water in contact with the skin and prevent the ice from running on the floor. A large fan will accelerate the cooling rate. The sheets or towels should be changed frequently and kept cold. Body massage may aid circulation and reduce the reflex vasoconstriction. This method can be managed by any ED, requires no special equipment, and provides rapid cooling with little risk.

Immersing the victim in cold water tubs has not been proven to be more effective and may make monitoring more difficult, although it has been enthusiastically recommended by some experts. Management of a seizing, incontinent, vomiting, obtunded patient with an endotracheal tube, multiple intravenous lines, catheter, and rhythm monitoring in a tub of ice water is clearly difficult. If a dysrhythmia supervenes, defibrillation can be hazardous or fruitless until precious minutes are wasted drying the patient.

Evaporative Method. Most investigators have abandoned ice water cooling in favor of the more theoretically sound method of evaporative cooling.[37,38] Evaporation of water involves the removal of 540 kcal per kilogram of water evaporated, while melting of ice to water involves the removal of only 80 kcal per kilogram. This means that evaporation of water from the skin will cool the patient up to four times more quickly than an ice water bath. The skin temperature is kept at 30-32°C to enhance vasodilation and increase heat flow. Shivering is much less prevalent.

Current methods of evaporative cooling include the use of a body-cooling unit (BCU) that sprays 15°C water about the patient suspended in a net hammock. High-speed fans ensure the most rapid evaporation possible. Although not found in most U.S. EDs, the effects of the BCU can be emulated with fans and mist sprayers. Evaporative cooling will work only if the patient has been completely undressed and is not covered with sheets or towels.

This method of cooling presupposes a dry microclimate to allow evaporation to take place. If the patient is not in an air-conditioned area or an area of low relative humidity, the needed evaporation will not take place. Evaporative cooling also presupposes an intact circulation to take cooled blood from the skin to the core. If the patient is in shock, evaporative cooling will most likely not be as effective as ice water cooling.

Other Cooling Methods. Although other methods are available, they require special equipment, special training, or have large risks. There are no studies available that have tested the efficacy of these methods against the more commonly employed methods. Numerous anecdotal reports describe the use of one or more of these methods in groups of one or two patients.

Peritoneal lavage with iced Ringer's lactate or dialysate solution provides faster core cooling but requires both special training and equipment. It has the theoretical advantage of cooling the liver preferentially, which may decrease the hepatic complications often associated with heatstroke.

Cardiopulmonary bypass provides rapid core cooling but requires preparation and a specially trained team. It should be reserved for patients in whom standard therapy proves ineffective. Cardiopulmonary bypass carries with it the risk of bleeding in the face of the frequent bleeding disorders associated with heatstroke.

Ice water enemas, bladder lavage, and ice water gastric lavage may be associated with water intoxication. If appropriate solutions are available, there is no reason not to employ these methods. They also contribute little to cooling in comparison to the vast skin surface areas.

Chilled air or oxygen administration is frequently difficult to arrange as most hospitals do not have this equipment readily available. It likewise contributes little to cooling in comparison to other more common methods.

Hypothermia. After reaching 39°C, current literature dic-

tates that the cooling measures should be modified or slowed to avoid overshoot into the hypothermic range. There is some controversy that carefully monitored cooling of the patient into the hypothermic range may allow for better recovery of stressed and hypoxic neurons. This idea awaits a well-controlled study.

Adjunctive Therapy

Shivering. When we start to vigorously cool a patient, the shivering and vasoconstriction reflexes may be activated. The shivering will cause an increase in oxygen consumption and will also increase the metabolic heat production.

Diazepam should be considered as the drug of choice to decrease shivering. Valium (diazepam) intravenously (0.1-0.3 mg/kg) will abolish the shivering reflexes. Diazepam also has significant anticonvulsant properties and does not significantly potentiate hypotension. It can cause respiratory depression, and appropriate airway maintenance equipment should be ready if the child has not already been intubated.

Thorazine (chlorpromazine), given very slowly intravenously, has also been recommended if the child starts to shiver during cooling. Although chlorpromazine can affect the normal thermoregulatory mechanisms, and has been implicated in the cause of heatstroke, after the onset of the heatstroke, there is no contraindication to its use on this basis. Chlorpromazine has the additional beneficial effect of increasing peripheral vasodilation and, thus, promoting heat exchange. Unfortunately, the phenothiazines can cause hypotension and arrhythmias and increase the chances of a seizure. Diazepam appears to be a better choice in this regard.

Dantrolene. Dantrolene, a hydantoin derivative, is the current treatment of choice for malignant hypothermia.[39,40] Dantrolene (1 mg/kg) was proposed as a treatment for heatstroke. Its mechanism was touted to be a calcium-release inhibition in the skeletal muscle. The drug does not seem to have significant cardiovascular effects when given in doses of up to 10 mg/kg by intravenous infusion. The major side effects appear to be muscle weakness and nausea.

Preliminary studies of dantrolene note that it will increase the rapidity of cooling by any means. Mortality and morbidity appear to be unchanged. Further investigation is needed before it can be routinely used in heatstroke.

Ineffective Therapy

Acetaminophen, Ibuprofen, and Aspirin. Ibuprofen, aspirin, and acetaminophen are often used to control fever. Children, of course, should rarely receive aspirin, and never when there is a possibility that the fever is due to influenza or varicella. There is a difference between the elevation of temperature due to a fever and the overwhelmed heat control mechanisms found in heatstroke. Ibuprophen or acetaminophen will not control the temperature of the heatstroke victim. In addition, ibuprophen may aggravate the clotting disorders found in heatstoke.

Alcohol. Since alcohol will evaporate faster than water, alcohol sponge baths have often been proposed as a cooling device. Alcohol may be absorbed by inhalation and through the skin and should not be used for cooling. With their thinner skin, children are particularly susceptible to toxic absorption.

Specialized Aspects of Therapy

Hypotension. Hypotension is often an early manifestation of heatstroke. Particularly in exertional heatstroke, large intravascular and interstitial fluid deficits are common and should be rapidly corrected. The Israeli army advocates the immediate use of at least 2L of Ringer's lactate in all suspected cases of heatstroke, even on the battlefield. The amount of fluids and rate of administration should be based upon hemodynamic parameter, level of consciousness, and urine output to determine the adequacy of resuscitation. Major fluid shifts can occur in those patients with acute rhabdomyolysis.

Many authors note that the next step in the treatment of hypotension associated with heatstroke should be continuing the cooling process. Certainly with the classic form of cooling with ice or iced cloths, a degree of peripheral vasoconstriction is induced, and the increased peripheral resistance may relieve the hypotension. This would be expected to be most effective in the patients with a hyperdynamic state and high-output cardiac failure, rather than those with hypotension from pump failure.

Alpha sympathomimetic drugs, such as epinephrine or norepinephrine should be avoided as they impair heat dissipation by induction of peripheral vasoconstriction. Either dopamine or dobutamine make better physiologic sense. The splanchnic vasodilation of small doses of dopamine may aid cooling. There are no special clinical trials that address dopamine use in patients with heatstroke.

Acidosis. Bicarbonate may be administered for significant acidosis, but metabolic acidosis is often found in conjunction with a significant respiratory alkalosis. The dose of bicarbonate given should be based on the arterial blood gases and should be initiated to particularly correct the base deficits by one-quarter to one-half at most.

Central Nervous System (CNS) Abnormalities

It is important to evaluate the patient carefully for the presence of cerebral edema. If cerebral edema is found, mannitol 0.5-1.0 g/kg and dexamethasone 10 mg IV may both be indicated, although the efficacy remains unclear.

Seizures should be treated with intravenous diazepam (Valium) or other benzodiazepines in conventional doses. Some authors advocate early treatment of patients with prophylactic doses of diazepam to prevent seizures prior to their occurrence. Phenytoin or Phosphenytoin has been recommended but may also precipitate dysrhythmias and hypotension.

Renal Function

The kidneys are severely affected by heatstroke, but these changes usually are reversible. Victims of heatstroke routinely will have elevated BUN and creatinine levels and almost always have abnormalities of the urine. The management most frequently reported to decrease the effects of the heatstroke is the early and vigorous use of mannitol in 0.25 g/kg dosages. Urine flow should be maintained at rates higher than 1 mL/kg/hr or 50-75 mL/hr. Alkalinization of the urine should also be considered.

Gastrointestinal Function

Nasogastric drainage is essential to prevent regurgitation of stomach contents. Ileus is a frequent component of the heatstroke syndrome. Gastric bleeding is infrequently reported and will be recognized earlier with the nasogastric tube in place.

Table 2. Precautions to Prevent Heatstroke in Hot Climates

WET BULB GLOBE TEMPERATURE INDEX	PRECAUTIONS
Cooler than 18.3°C	No precautions are needed. Observe those athletes who lose more than 3% of body weight.
18.8-23.3°C	Insist that unlimited amounts of water be given. Flavored or iced water is preferable. Coaches or race officials should ensure that 0.5-1.0 L of water per hour is consumed. Moderate risk of heat injury exists.
23.8-27.7°C	Any person who is susceptible to heat or humidity probably should not run in races. Coaches must ensure adequate water consumption of 1-2 L per hour. Water should be cooled and flavored. High risk of heat injury exists.
Higer than 27.7°C	Work/rest cycles of 30 min/30 min for all work. Alter practice schedule and conduct practice in shorts. Practice at 6 a.m. is preferable. Races and competitive athletic events should probably be rescheduled. Severe risk of heat injury exists.

Table 3. Safe Heat Acclimation for Athletes

- Complete heat adaptation takes as long as three weeks.
- Train either early morning or late evening for the first week's workouts.
- Gradually schedule workouts closer to midday.
- On particularly hot days, resume interval training in early morning or later in the evening. *(See WBGT temperature guidelines, in Table 2.)*
- Ensure appropriate fluids are taken all times.
- Drink cold fluids if at all possible.
- Drink 6-8 ounces of fluids every 10-15 minutes. (Remember that thirst is a poor guide to acute dehydration.)
- Electrolyte replacement solutions are optional, but should have no more than 120 mmol/L concentration of all electrolytes.

Prognosis

The high morbidity and mortality of heatstroke stands in stark contrast to that of profound hypothermia, where the prognosis is not generally related to the depth of temperature but rather to underlying diseases. The overall prognosis of heatstroke depends upon the body habitus, the functional reserve capacities, intercurrent chronic diseases, and delay in institution of cooling.

In general, there are three major prognostic groups found in heatstroke:

The first group consists of those who have had a prolonged period of coma, delayed seizures, coagulation abnormalities, acute tubular necrosis, and profound hypotension. Few of these patients awaken from coma and mortality is quite high. Hypotension that does not respond to volume resuscitation and cooling is particularly ominous. The levels to which the SGOT, SGPT, and LDH are elevated are of prognostic significance, with early, high elevations being poor prognostic indicators.

The second group consists of those patients who have regained consciousness within 4-10 hours. The hepatic and renal pathology reaches a peak within 3-5 days and then subsides. Some of these patients have transient or permanent neurologic sequelae. Ataxia and cerebellar pathology is particularly common. These patients often have long-lasting deficits in thermo regulation.

The third group has only transient unconsciousness, rarely lasting greater than three hours. Hepatic and renal pathology is limited in scope. There is usually rapid recovery.

Poor prognostic signs for all patients with heatstroke include a core temperature greater than 41.1°C, particularly if prolonged; SGOT greater than 1000 in the first 24 hours; prolonged duration of coma; hypotension that does not respond to cooling and fluid repletion; and either renal failure or hyperkalemia. Common causes of death in these patients include irreversible CNS damage, irreversible bleeding diathesis, hyperkalemia, renal failure, hepatic failure, and late sepsis.

Prevention—General Principles

The most important therapy in the treatment of heatstroke is to prevent it.

Wet Bulb Globe Temperature (WBGT) Index. Heatstroke becomes more likely when humidity and temperature are both high. This may be predicted by the use of the WBGT index. *(See Table 2.)* The effects of humidity and radiant heat are added to the dry air temperature to predict the heat stress. The dry air temperature is measured with a shaded regular thermometer. A thermometer enclosed in a wet wick (wet-bulb) measures the effects of humidity on the temperature and accounts for 70% of the reading. The radiant effects of the sun are estimated by the use of a thermometer enclosed in a 6-inch hollow black sphere.

The WBGT may be calculated by:

WBGT = 0.7 (wet bulb temp) + 0.2 (black globe temp) + 0.1 (dry bulb temp).

The dry bulb temperature alone is generally considered to be a poor indicator of heat stress since it neglects the effects of radiant heat, air movement, and humidity. Of these, humidity appears to have the greatest effect, since high humidity precludes effective evaporation.

If a means for assessing the WBGT is not readily available, an alternative equation may be employed using the dry air temperature and the water vapor pressure:

$$WBGT = (0.567\ T_{db}) + (0.393\ P_a) + 3.94$$

Where T_{db} = dry bulb temperature

P_a = water vapor pressure.

These environmental variables should be readily available from local weather or radio stations. In many cases, the WBGT is calculated by these sources.

If the WBGT index is above 32°C in the hottest two-hour part of the day, physical training and strenuous exercise should be suspended. The American College of Sports Medicine recommends that if the WBGT is above 28°C, consideration should be given to postponing or rescheduling races and similar activities.

Acclimation. Acclimation to the heat can be achieved in about 2-3 weeks for those who must exercise in hot and humid ambient

temperatures. *(See Table 3.)* Prior physical conditioning is an important part of acclimation. In practical terms, the more fit the young athlete and the less effort that it takes to maintain a given pace, the fewer problems the athlete will have with the heat.

The principle of acclimation is gradual increase to tolerance of physical activity during periods of heat and humidity. The runner should start with 15-20 minutes of training during the warmest hours of the day. At the end of two days with no ill effects, the runner should increase the duration to 35 or 40 minutes for 2-4 more days. If this remains satisfactory, then the duration can be increased to one hour.

Fluid Intake—General

It is especially important to maintain adequate fluid intake. Thirst is a poor indicator of dehydration, and there is no signal for salt depletion. Cool, mildly flavored water is tolerated much better than warm water. A good indication of adequate fluid intake is maintenance of clear or unconcentrated urine. Salt intake with meals may be increased, but salt tablets and salted beverages are of questionable benefit.

Athletes doing moderate to heavy work in the heat must sweat, and may lose from 3-5 L of water per hour. Depending upon environmental and work conditions, he or she may require up to 16 L of water to drink per day. The loss of 3 L of water is equal to about 2% of the body weight. Between 2-6% dehydration, the athlete's performance is impaired, both physically and mentally. Beyond 7% dehydration, the body will no longer tolerate sweating or cutaneous vasodilation and these mechanisms of heat control are lost.

During a training session, run, or race, the young competitor should drink 6-8 ounces of water or some kind of fluid every 20 minutes. Athletes who drink anything—water, sugar water, sports drinks—show fewer symptoms of heat stress. Cooled drinks may or may not lower core temperature, but they are more palatable and make the athlete feel better. After training, have the athletes end the training session with a long celebratory drink of at least 10-20 ounces of water.

The major advantage of the sports drinks is the flavoring and the subsequent increased consumption of fluids. There is no good evidence that one sport drink is better than another or even better than water for short training sessions. Orange juice concentrate diluted to twice usual concentration will provide similar electrolytes, taste, and will be much cheaper than the sports drinks.

Infants and Children

Children should not be bundled and should not be left in cars or hot buildings. Children with a febrile illness, vomiting, diarrhea, or an upper respiratory tract infection should be promptly treated for these illnesses. For a given level of dehydration, children have a greater increase in core temperature than do adults.[41] If possible, these patients should be provided with air conditioning. Abundant fluids, fans, and antipyretics are alternatives, but the child should be carefully monitored.

Other High-Risk Persons

During high ambient temperatures, those children at high risk should avoid the heat as much as possible and should reduce activity as much as possible. No child should sleep in direct sunlight during a heat wave.

Those parents who do not have home air conditioners should be advised to take very young children to an air conditioned library or to window shop in air conditioned malls for at least 1-2 hours during the hottest part of the day. Certainly, those who live on the top floors of multilevel apartment buildings should realize that increased heat stress is associated with this location.

Physicians should evaluate children on diuretics, anticholinergics (to include tricyclics), calcium blockers, beta-blockers, phenothiazines, and cardiac medications on a more frequent basis during the summer months. Medications should be carefully titrated, with a decrease of doses during hot weather as indicated for individual patients.

Training and Competition in the Heat

Organizers of athletic events should be cautious of regional weather variations in scheduling for competition. Races should be organized to avoid the hottest parts of the day and, preferably, the hottest months of the year. Summer events should be scheduled before 8:00 a.m. (ideally) or in the evening after 6:00 p.m. to avoid the additional stresses of solar radiation.

For those who need to increase fitness and are not currently in good physical condition, fitness training should be done when the air is cooler, before 9:00 a.m. and after 6:00 p.m. Even then, humidity may mandate a reduction in the pace or duration of fitness training. Proper attention to the imposed heat stresses can markedly reduce the incidence of heat illness, without compromising training objectives.

Summary

The diagnosis of heatstroke should be considered in all patients with an alteration of consciousness and an elevated temperature.

The syndrome carries a high morbidity and mortality and needs emergent treatment. Rapid cooling with iced towels or water mist and fans is the treatment of choice. Hypotension, seizures, disseminated intravascular coagulation deficits, and oliguria are all common complications of this disorder.[42]

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Physician's CME Questions

32. Self-limited swelling of the hands and feet that resolves after a patient spends a few days in an air-conditioned climate is:
    A. heat syncope.
    B. heat exhaustion.
    C. prickly heat.
    D. heat edema.
    E. heatstroke.

33. Treatment of heatstroke should include which of the following?
    A. Lasix (furosemide) 80 mg IV stat
    B. Removal of clothing
    C. Cautious fluid replacement
    D. High-flow supplemental oxygen
    E. Diazepam for muscle twitching

34. Heatstroke may be characterized by:
    A. moderate hypovolemia.
    B. nausea and vomiting.
    C. alteration in consciousness.
    D. tachycardia.
    E. All of the above.

35. Which of the following is *not* a symptom of heatstroke?
    A. Hot, dry skin.
    B. Hot, moist skin.
    C. Nausea.
    D. Headache.
    E. Insomnia.

36. Neurological damage is certain when the core body temperature rises above:
    A. 39°C.
    B. 41°C.
    C. 43°C.
    D. 45°C.
    E. 46°C..

37. Prevention of heatstroke is aided by:
    A. shopping malls.
    B. air conditioning.
    C. proper fluid management.
    D. acclimation.
    E. All of the above.

In Future Issues: Animal Bites