HEAT STROKE AND OTHER ENVIRONMENTAL HEAT ILLNESSES

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Case #1

69 year-old African-American male with a history of coronary artery disease, congestive heart failure and diabetes presented to the Parkland Emergency Department during a prolonged heat wave with hot dry skin, coma and temperature >109°F. He was intubated and treated with rapid cooling by an ice water bath. Within 45 minutes his core temperature was 102°F. He was admitted to the Intensive Care Unit and was awake within 48 hours of admission. His course was complicated by a myocardial infarction and prolonged delerium. He was discharged three weeks later with significant cognitive dysfunction.

Case #2

21 year old caucasian male college wrestler was admitted to the Parkland Emergency Department with a temperature of 108°F. He had been taking dinitrophenol (DNP) to lose weight to get to a lower wrestling weight class. He had obtained the chemical in pill form from a diet clinic in Dallas. On admission he was agitated with a temperature of 108°F. Increased muscle tone was noted. He was cooled with ice massage and fans, but his temperature continued to rise. He was then placed in an ice water bath, but his core temperature continued to climb. Severe tetany persisted despite treatment, and he developed shock refractory to fluids and vasopressors. He died four hours after admission.

Case #3

A 27 year-old professional football player was admitted with a temperature of 108°F. He collapsed just after practice where the outside temperature reached the mid 90's with high humidity and heat index of 110°. The day prior to admission, he withdrew from football practice because of heat related symptoms. He presented with shortness of breath, dizziness and weakness. He was 6'4" and weighed approximately 235 pounds. His temperature on arrival to the emergency facility was 108°F. Cooling procedures were instituted, but multi-organ failure developed. He died within 24 hours of admission (*Associated Press, 2001).*

INTRODUCTION

These cases illustrate the diverse presentation of patients with severe heat-related illness. The morbidity and mortality of heat-related illnesses, including heat stroke, remain high despite advances in our understanding and treatment of these disorders and warnings from experts in the field[1]. Each year, several cases of heat stroke involving conditioned athletes illustrate the importance of this group of illnesses. Less than one year ago, the death of Minnesota Viking Korey Stringer reminded us that even conditioned, professional athletes with supervision are susceptible to the effects of a hot environment. This review will examine various heat-related illnesses and their treatment.

The severity of heat-related illness ranges from mild (heat cramps) to severe (heat stroke). These syndromes share many pathophysiological features. However, an understanding of the distinct presentation and treatment of each syndrome is important if significant morbidity and mortality are to be avoided. Anyone involved with groups of children or adults who exercise in or are exposed to a hot environment should be familiar with the signs, symptoms and emergency treatment of these disorders.

HEAT REGULATION

The regulation of body temperature is a characteristic of warm blooded animals which allows humans and other animals to survive in different environmental conditions and maintain a constant body temperature. Temperature regulation is a complex process balancing heat production and dissipation through various mechanisms.

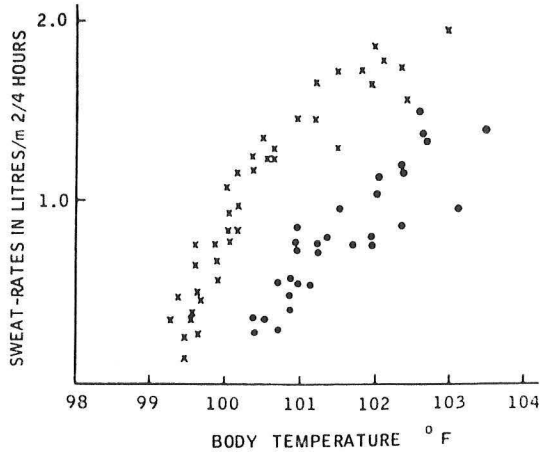
Heat production is a product of cellular metabolic activity and can be increased from its basal state of about 60-70 kcal/hour by many mechanisms that include increased metabolic activity, shivering and exercise which may bring total heat production to over 1000 kcal/hour[2]. The importance of the rate of heat production in heat-related illness is illustrated by the fact that trained athletes exercising in a hot environment can increase core body temperature by as much as 0.3°C per minute due to an increase in muscle and hepatic vein temperatures, both of which can rise to greater than 41°C during moderate to heavy exercise[3,4].

The dissipation of heat from the body involves evaporation, conduction, convection and radiation. Radiation is the transfer of heat to cooler objects in the environment by electromagnetic waves. It is most effective when the difference in temperature between the two objects is large. Convection refers to the loss of heat between the skin and currents of air or liquids. Conduction entails the transfer of heat between the body and objects to which it is in contact. Evaporation refers to the loss of heat from evaporating sweat. At rest in a cool environment, most of the heat lost from the body is by radiation of heat to cooler objects in the environment. As environmental temperature increases, the percentage of heat lost by this mechanism falls, and conduction/convection and evaporation become more important. These mechanisms cool peripheral blood in the skin which is then returned to the central circulation lowering core temperature.

Maintaining a high rate of skin blood flow to distribute this heat to the body surface is obviously critical to the transfer of adequate quantities of heat from the body in conditions of high heat production in a hot environment. Conduction and convection of heat to the environment, in most situations, is to air. This transfer of heat can be increased to some extent by increasing wind velocity. As the temperature of the body and environment increases, the importance of evaporation as a means of heat loss increases dramatically, thus the importance of adequate sweating in a hot environment. Evaporation is very dependent on air movement, humidity and sweat volume. Less heat is lost as air movement diminishes, humidity rises, and sweat rate decreases. Evaporation is an efficient means of heat loss because 1 kcal of heat is lost by the vaporization of each 1.7 ml of sweat. Consequently, the decrease or cessation of sweating in a hot environment dramatically affects the ability to lose heat.

Sweat rates can increase dramatically if necessary as illustrated in figure 1. Sweat rates rise as body temperature increases[5], and maximum sweat rate is more after acclimatization. Also sweat rate is higher at any given temperature or work load after an individual is acclimated.

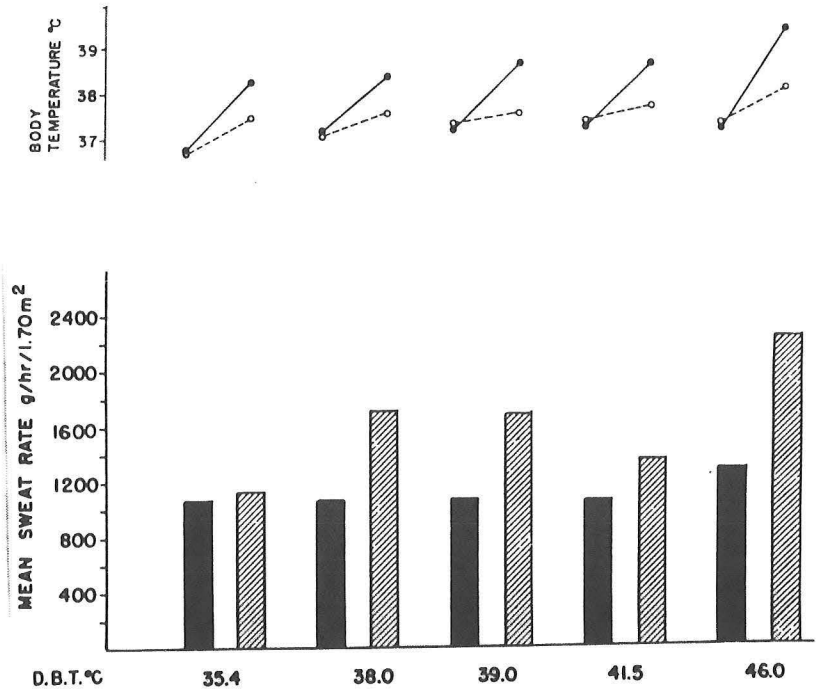
Figure 1



Dependence of sweat rate on body temperature, before (●) and after (x) acclimatization.

However, maximum heat loss efficiency can never be reached because these high rates of sweating cannot be maintained over a prolonged period and about 20% of sweat is lost by dripping. The maintenance of high sweat volumes during heat stress can be modified by fluid intake as noted by Knochel in figure 2[6].

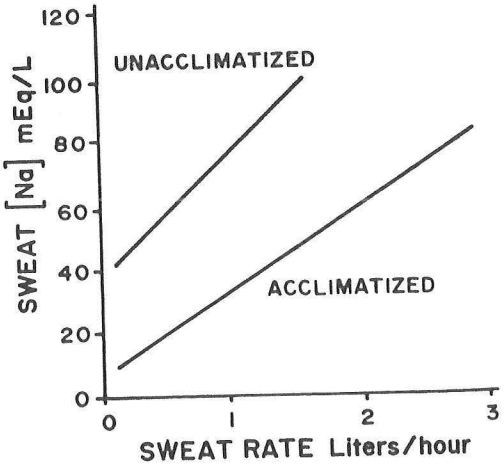
Figure 2



Effect of fluid intake on sweat rate at different ambient temperatures. Subjects were walking at 4 km/h for 2 h. Solid bars, no fluid intake; crosshatched bars, forced fluid intake (3800 ml/2 h). Top panel shows increments in rectal temperature. Increments were less when fluids were forced (filled circles) than without fluid intake (open circles).

Sweat rates were higher in individuals forced to take fluid over various heat loads induced by walking in various environmental temperatures when compared to those not taking fluids. Those forced to drink 3.8L over 2 hours had less increase in rectal temperature when compared to those taking no fluids. Normally sweat is hypotonic and contains sodium and potassium concentrations of about 40-100 mEq/L and 6 mEq/L, respectively. When a person is acclimated, the concentration of sodium in sweat falls to 20-75 mEq/L and the concentration of potassium rises to as much as 18 mEq/L. The decrease in sweat sodium is a result of increase serum levels of aldosterone during heat stress[7,8]. Despite lower concentrations of sodium in the sweat of acclimated individuals, the large volume of sweat still results in high sodium loses as noted in figure 3[6].

Figure 3



Relationship of sweat sodium ion concentration to sweat rate in acclimatized and unacclimatized humans.

Note that sweat sodium increases as sweat rate increases in both acclimated and non-acclimated individuals, probably because aldosterone has less time to act on the duct with high sweat rates. Consequently, an acclimatized individual may loose more sodium overall since the increase in maximum sweat rate is more than the decrease in sodium concentration of the sweat. This is illustrated in the calculations of table 1.

Table 1: Theoretical Total sodium excretion in sweat

	Maximum Sweat Rate (L/hr)	Sodium Conc (mEq/L)	Sodium Loss (mEq/L)
Unacclimatized	1.5	100	150
Acclimatized	2.5	70	175

Examination of these various methods of heat loss will lead to an obvious conclusion. Physical activity in a hot environment with no wind movement and 100% relative humidity is very dangerous and leads to many of the heat-related illnesses seen in the United States every year. These extreme situations of uncompensatable heat stress can be found in many everyday situations, especially in situations where heavy protective clothing is worn (firefighters, toxic cleanup crews, etc.) or heat production is great (athletes, laborers, patients during a seizure, drug users, etc.).

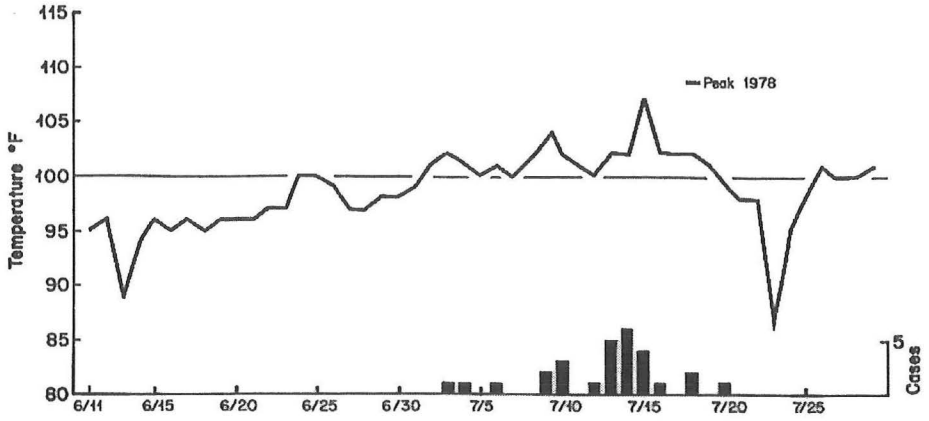
However, it is well known that many persons are able to work in a very hostile environment with high humidity and hot temperatures without suffering significant heat illness. This is because of an adaptive process called acclimatization which allows individuals to exercise or work in a very hot and humid environment without suffering serious effects from the heat. The importance of acclimatization in heat tolerance is illustrated in the 1978[9] and 1980 heat stroke epidemics in Dallas. During a normal spring and summer, the temperature in Dallas gradually increases with a peak in late July and early August. Despite many days with high heat indices and temperatures, heat strokes are relatively uncommon and occur sporadically. The daily high temperatures in the summer of 1978 are illustrated in figure 4.

Figure 4

Daytime

High temperatures

Dallas, 1978



During that year the temperature rose rapidly and did not follow the normal gradual pattern. This rapid rise in daily highs was accompanied by high night temperatures also. This rapid rise resulted in many cases of heat stroke over a short period of time illustrated in the lower part of the above graph. After this flurry of cases, very few heat strokes were seen the rest of the summer despite very hot days and prolonged periods of high humidity. The survivors in Dallas became acclimated to the heat.

Acclimatization results in changes that involve many organ systems that are illustrated in table 2[10-20].

Table 2. Physiologic Changes with Prolonged Heat Exposure

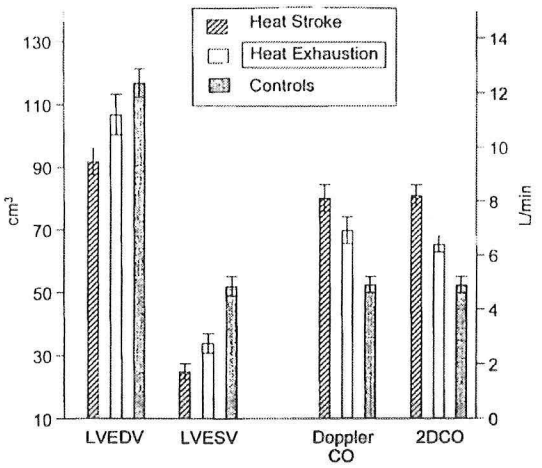
Maximum Cardiac output	Increased
Peak heart rate	Decreased
Stroke volume	Increased
Blood volume	Increased
Plasma volume	Increased
ECF	Increased
TBW	Increased
Temperature rise with given workload	Decreased
Heat production with given workload	Decreased
Aldosterone production	Increased at least acutely
ACTH	No change
Sweat production	Increased at least for a period
Sweat sodium concentration	Decreased
Sweat potassium concentration	Increased
GFR	Increased

Acclimatization occurs rapidly when working or exercising in a hot environment and generally takes two to three weeks. It is accelerated by physical conditioning which in itself causes similar physiologic alterations. One of the main adaptations to the heat is the ability to maintain a hyperdynamic circulation by increasing cardiac output and vasodilating vessels in the skin. This allows the rapid transfer of heat from the core to the skin where it can be dissipated to the environment by evaporation or conduction/convection. Exercising muscles generate a great deal of heat and peripheral vasodilation of muscle vessels. This

increase in heat production occurs in the muscles involved in the activity and the liver due to increased gluconeogenesis. As this heat enters the central circulation, core blood temperature rises. This change is detected by the hypothalamus which induces vasodilatation in the skin vessels and an increase in sweat secretion. The marked increase in skin blood flow results in a compensatory splanchnic vasoconstriction and an increase in cardiac output of 50-75% to prevent hypotension[21]. If exercise and heat production continue, however, effective arterial blood volume will eventually to fall because the above mechanisms are inadequate to compensate completely for the increase in skin blood flow and sweating and for the loss of extracellular fluid due to muscle activity. This decrease in effective arterial blood volume may result in decreased venous return with subsequent increase in heart rate and decrease in stroke volume leasing to a fall in cardiac output. This then causes peripheral vasoconstriction of the skin vessels and a decreased ability to dissipate heat leading to hyperthermia. These circulatory changes can be seen in the following data from Shahid[21], et al, who studied the hemodynamic changes that occur with heat exhaustion, heat stroke and heat exposure (fig 5). Note that cardiac index and output rose as expected in heat exhaustion and stroke despite hypovolemia manifested by a decrease in end diastolic and systolic volume. However, Doppler signs of vasoconstriction were seen in 10/12 patients with heat stroke.

Figure 5

Left ventricular volume at end-diastolic (LVEDV) and end-systolic (LVESV) of the three groups, along left *y* axis, and cardiac output (CO) by Doppler and two-dimensional echocardiography (2D), along the right *y* axis, demonstrating the significant difference between each study group and controls.



The reasons that some people develop heat illness while others do not in a hot, hostile environment are varied. O'Donnell[23] studied athletes who suffered heat illness in the 1976 Boston Marathon and compared them to unconditioned marine recruits and found significant differences in the two groups. The importance of acclimatization is illustrated by the fact that 7 of 8 marathoners who developed heat illness trained in a northern, cooler environment.

The hemodynamic data from the 8 marathoners is illustrated in table 3. Note that two of the participants met the diagnostic criteria for heat stroke (temperature greater than 40°C).

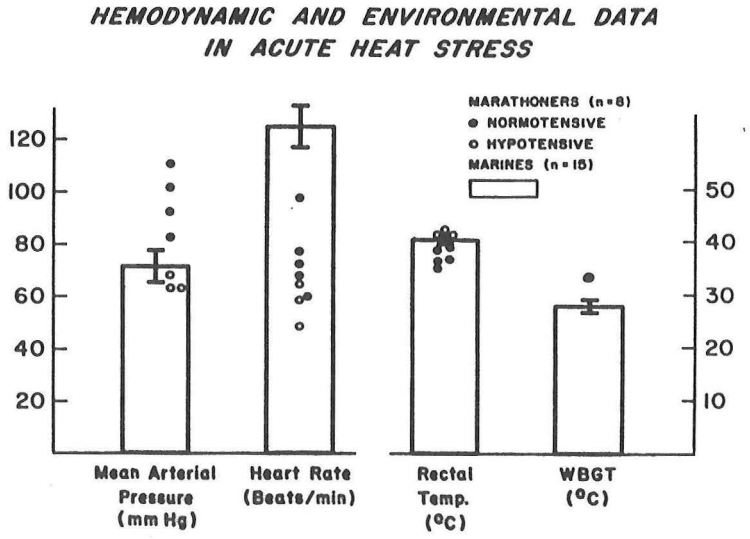
Table 3 Hemodynamic Data[23]

Subject Number	Rectal Temperature (°C)	Mean Arterial Pressure (mmHg)	Pulse Pressure (mmHg)	Heart Rate (beats/min)
1	40.5	67	20	62
2	38.4	84	36	96
3	38.3	93	40	68
4	39.0	84	34	60
5	39.2	103	40	70
6	40.0	68	25	48
7	38	111	34	74
8	40.6	68	25	58

The hemodynamic responses of the eight marathon runners are compared to the Marine recruits (figure 6) which illustrates that heart rate was inappropriately low in the marathon runners given their low blood pressure. However the blood pressure was relatively maintained despite the low heart rates indicating a higher stroke volume in the well-trained individuals when compared to the untrained ones.

Figure 6.

This figure compares hemodynamic and environmental data from 15 Marine recruits to the 8 marathoners. Although mean arterial pressure and rectal temperature are compared in the two groups, heart rate is markedly decreased in the marathoners. The environmental conditions, indicated by the wet bulb globe temperature (WBGT), were more severe for the marathoners than for the group of Marines.



Potassium seems to play an important role in the development of heat-related illness. Several mechanisms have been proposed to explain the association of hypokalemia with heat illness. Some of these can be found in table 4. Of particular importance is the lack of vasodilatation in exercising muscles in

potassium depleted animals. This contributes to the high incidence of rhabdomyolysis in exertional heat stroke[24,25].

Table 4 Hypokalemia and Heat Stroke

Impaired vascular responsiveness to catecholamines
Glucose intolerance
Hypotension
Arrhythmias
Impaired insulin release
Rhabdomyolysis
Impaired circulatory response to exercise

Heat-related illness tends to occur in two groups. First, persons with underlying diseases which affect the adaptive systems involved with heat dissipation are at risk. Almost any condition or drug affecting the central nervous system and cardiac system puts a patient at risk for heat-related illness. Obviously, conditions that affect the above systems are predisposed to heat-related illnesses. Some of these predisposing conditions illustrated in table 5.

Table 5 Conditions Associated With An Increased Risk of Heat Illness [5,26-29]

Drugs: diuretics, anticholinergics, beta blockers, antipsychotics, sympathomimetics, alpha agonists.	
Volume depletion and dehydration	Age – very young or old
Infection	Poor
Diabetes mellitus	Psychiatric illness
Sweat gland dysfunction	Cardiovascular disease
Scleroderma	Hyperthyroidism
Burns	Parkinsonism
Potassium deficiency	Pheochromocytoma
Insulating or vapor impervious clothing	Ectodermal dysplasia
Exertion	Linear skin dystrophy
Lack of acclimatization or conditioning	Fatigue
Obesity	Central nervous system disease
Alcoholism	Cystic fibrosis
Hot environment	Previous heat stroke

HEAT EDEMA

Heat edema refers to the dependent swelling that frequently occurs when unacclimatized persons are exposed to a hot environment. It usually develops within several days of initial exposure to the heat and is usually mild. The etiology is not entirely clear but is probably related to salt retention from the increase in serum aldosterone, heat induced decrease in urinary sodium excretion due to vasodilatation, and increased salt intake seen early in the process of acclimatization. This condition is self-limited and requires no treatment.

HEAT CRAMPS

Heat muscle cramps is a very common disorder that afflicts many persons exposed to heat[30]. This syndrome tends to occur in well-conditioned and acclimatized individuals. The cramps usually occur in the muscles involved in daily activity or exercise. They are very painful and tend to occur after the muscle activity is completed, frequently at night. This syndrome is not associated with any systemic symptoms, thus differentiating it from the muscle cramps that occur with heat exhaustion. Patients who suffer from muscle cramps usually drink plenty of water during and after exercise, but tend to ingest insufficient amounts of salt. These patients frequently have hyponatremia because the sweat lost is replaced with water only. The treatment for heat cramps is to liberalize the intake of salt. Usually a small increase in the amounts of dietary salt is all that is needed. Salt tablets should never be given. If patients have severe cramps and present to a medical facility for treatment, intravenous saline will relieve the cramps quickly. These patients should be encouraged to continue their liberal intake of water.

Muscle cramps can also occur with more severe forms of environmental heat illness such as heat exhaustion and heat stroke. Usually the cramps in these conditions occur while the muscle activity or exercise is ongoing and tend to involve any muscles including those of the abdomen and back. Treatment for muscle cramps in these conditions is discussed below.

HEAT SYNCOPE

Heat syncope is a condition that generally occurs in unacclimatized patients who exert themselves in a hot environment while taking insufficient salt and fluids. Heat syncope can be aggravated by assuming a position after exertion which facilitates pooling of venous blood in the lower extremities. A common scenario

for this syndrome is seen in Texas in mid-August when high school marching bands begin their practices by marching for long periods of time in the heat and then standing at attention. This syndrome is due to volume depletion combined with heat induced vasodilatation resulting in decreased cardiac output that has been exacerbated by the pooling of blood in the lower extremities. The resultant arterial vasoconstriction which can usually prevent a fall in arterial pressure in this situation is counteracted by the skin vasodilation due to the heat and muscle vasodilation due to exercise. If measured immediately these patients usually have normal body temperature and electrolyte concentrations. Occasionally, hypokalemia will be observed especially if measured a few days after the exposure to heat begins. The propensity to develop this syndrome may be increased by hypokalemia which decreases the circulatory system's ability to vasoconstrict in response to hypotension. This syndrome can be prevented by adequate intake of salt-containing solutions and intelligent management of exercise length in the heat until acclimation occurs. Treatment of heat syncope consists of lowering the patient's head, raising the lower extremities above the level of the heart, removing the patient from the heat or direct sun and the administration of salt-containing solutions by the route appropriate for the condition of the patient.

HEAT EXHAUSTION

Heat exhaustion is a common clinical syndrome occurring in warmer climates or in a situation in which a person is exposed to a hotter environment than that for which the person is acclimatized. Heat exhaustion tends to occur in two clinical settings which parallel the two types of heat stroke discussed below. Salt depletion heat exhaustion is encountered when a person is exposed to a warm environment, usually with exercise or other muscle activity without ingesting enough salt-containing solutions[31,32]. These patients do consume significant amounts of free water. As the syndrome develops, the patient develops generalized malaise, headache, giddiness, dizziness, nausea and vomiting, tachycardia, hypotensions and other signs of volume depletion as well as muscle cramps, confusion and a decreased urine output. Patients suffering from this condition usually have elevated body temperature. Because free water intake is adequate or excessive, the individuals may not develop intense thirst and will not have dehydration hyponatremia. If the exposure to heat or exertion continues, these patients frequently develop shock, rhabdomyolysis, ataxia, and other neurologic impairment. If the exertion or heat exposure continues, exertional heat stroke may develop.

Treatment for exertional heat exhaustion includes removing the patient from the hot environment, stopping the physical activity associated with the syndrome and administering salt-containing solutions. If symptoms are mild and hemodynamic

stability is documented, the solutions can safely be administered orally. Symptoms usually disappear rapidly after treatment is initiated. In more severe cases, especially when hypotension and rhabdomyolysis are present, intravenous fluid should be given a monitored environment. Complications such as rhabdomyolysis should be treated aggressively.

The second type of heat exhaustion or water depletion-type usually occurs in very young, the very old, or those who have limited acces to water during heat stress with or without exertion[5]. As dehydration develops, patients develop confusion, muscle cramps, intense thirst, paresthesias, tetany, and altered mental status. These patients then gradually lose their ability to control body temperature. Left untreated, this condition usually progresses to the syndrome of classic heat stroke discussed below. Treatment for this type of heat exhaustion includes removing the patients from the severe heat exposure and administering free water based on the deficit calculated by standard formulas. Salt should be given to these patients only to re-establish adequate extracellular fluid volume status. As with salt depletion heat exhaustion rhabdomyolysis may occur.

HEAT STROKE

Heat stroke is a devastating, frequently fatal complication of exposure to extreme heat which accounts for hundreds of deaths each summer in the United States, especially among the poor and elderly. The syndrome is defined as a temperature of greater than 105°F(40.6°C) with altered central nervous system function and usually hot, dry skin. The CNS dysfunction often takes the form of coma, delirium, lethargy, stupor, irritability or agitation. The differential diagnosis of heat stroke includes thyroid storm, pheochromocytoma crisis, CNS infection or trauma, drug intoxication, neuroleptic malignant syndrome, malignant hyperthermia, hypothalamic dysfunction, infections known to present with high temperature (malaria), brainstem hemorrhage, delirium tremens, and anticholinergic poisoning.

As with heat exhaustion, heat stroke generally occurs in two clinical situations[39]. First it occurs in young, healthy, usually highly motivated people who are required to exercise in a hot environment to which they are poorly acclimatized[35-38]. This group includes athletes, military recruits, laborers, and drug users who exert themselves in a hot environment. It is the third leading cause of death in the United States among high school athletes. This exertional heat stroke is the end result of continued exercise or exertion through the syndrome of exertional heat exhaustion and can develop quickly (usually hours). It occurs when heat generation exceeds the capability of the body to dissipate heat by normal means or by the inhibition of heat loss due to insulating, impervious clothing. Its development can also be accelerated by water or salt deprivation during

exercise. Patients with exertional heat stroke have the defining very high body temperature (greater than 40.6°C) and mental status abnormalities associated with severe metabolic derangements. Many of these patients continue to sweat (greater than 50%[5]) despite having temperatures well above 106°. However the sweating is unable to dissipate the increased heat production associated with the increased activity. Consequently, lack of sweating cannot be used as a diagnostic criteria in this type of heat stroke. Severe metabolic abnormalities occur with this syndrome including lactic acidosis from tissue hypoxia and volume depletion; rhabdomyolysis with subsequent hyperphosphatemia, renal failure with hypocalcemia and hyperkalemia; DIC and seizures. Three patients frequently have significant lactic acidosis. Mortality from this type of heat stroke is lower than with classic heat stroke if aggressive cooling and fluid-resuscitative measures are undertaken. The exact triggering mechanism of the eventual cardiovascular collapse that occurs in severe heatstroke is unknown, but may involve interleukin-1, tumor necrosis factor or endotoxin[47,48].

Most patients who survive the initial treatment of an exertional heat stroke do so without serious long-term disabilities. Occasionally patients will have sweat gland necrosis that inhibits their ability to work in a very hot environment in the future. Otherwise, the main morbidity from exertional heat stroke involves renal failure from rhabdomyolysis and DIC. The renal failure tends to resolve with appropriate treatment and dialysis.

Classic heat stroke tends to occur in elderly[8,33,34] patients who have underlying diseases that affect their ability to regulate heat in a hot environment, in persons with drug intoxication, and in persons left in an extremely hot environment, e.g., car on a very hot day. When these patients are placed in an environment that is hotter than that to which they are acclimated, their ability to regulate heat is lost if the environmental conditions are hot enough. If patients are exposed to gradually increasing amounts of heat, the normal process of acclimatization usually prevents the occurrence of heat stroke despite patients being exposed to very severe temperatures. This lack of acclimatization explains the fact that classic heat stroke tends to occur in epidemics. In Dallas for example, our two worst years of classic heat stroke (1978, 1980) occurred when daily temperatures increased significantly over a short period of time and remained elevated. When this situation occurs, patients usually present two to five days after the severe daily temperatures are encountered. Patients who do not suffer a heat stroke within a week to 10 days will become acclimated to the hotter temperatures and generally not develop a problem unless further worse heat stress is applied. Usually the environment predisposing to classic heat stroke epidemics is a sudden heat wave with high daytime and night time temperatures with high relative humidity that lasts for more than 2 or 3 days. Persons living in closed shelters with little air movement and no air conditioning are particularly susceptible for obvious reasons.

If heat exposure is prolonged, patients develop worsening symptoms of water depletion type heat exhaustion for hours to days before sweating ceases then heat stroke develops rapidly. Symptoms of incipient heat regulatory failure include goose flesh, chills, and cessation of sweating[40-42]. Whether the cessation of sweating is due to an inability to maintain high sweat rates for a long period of time (sweat fatigue)[43,44] or central nervous system failure of thermoregulation[45] is unknown. Because these patients have lost their ability to regulate temperature, anhydrosis is part of this syndrome. Unlike individuals with exertional heat stroke, patients generally do not have severe volume depletion, but can have severe dehydration and hypernatremia. Unlike exertional heatstroke, these patients usually have a respiratory alkalosis and metabolic abnormalities are not as severe. If lactic acidosis is present, prognosis is poor[9] The differences between classic and exertional heat stroke are listed in table 6. As with heat exhaustion there may be some overlap in any given patient.

Table 6 Comparison of Classical and Exertional Heat Stroke[5]

	Classical	Exertional
Age group	Very young, very old	Men 15-45
Health status	Chronic illness common	Usually healthy
History of febrile illness Or immunization	Unusual	Common
Activity	Sedentary	Common to football players, military recruits, competitive runners
Drug use	Sweat depressants Diuretics, haloperidol, Phenothiazines	amphetamines
Sweating	Usually absent	Often present
Respiratory alkalosis	Dominant	Mild
Lactic Acidosis	Absent or mild	Often marked
Acute renal failure	<5% of patients	30% of patients
Rhabdomyolysis	Seldom severe	Severe
Hyperuricemia	Modest	Severe
Creatinine: BUN Ratio	1:10	Elevated
CPK, aldolase	Mildly elevated	Markedly elevated
Hypocalcemia	Uncommon	Common
DIC	Mild	Marked
Hypoglycemia	Uncommon	Common

Survivors of classic heat stroke suffer many complications, the most common of which being severe neurologic damage that is frequently irreversible. Because classic heat stroke usually occurs in elderly or debilitated individuals, the mortality is high despite aggressive treatment measures.

The treatment of heat stroke involves aggressive supportive measures and rapid cooling. The morbidity and mortality (up to 80%) of heat stroke are directly related to the length of time the body is exposed to critical temperatures that induce tissue necrosis. Consequently, heat stroke should be considered an extreme medical emergency requiring prompt diagnosis and aggressive treatment if complications and death are to be prevented. Cooling measure should be initiated at the scene of collapse and continued during transfer to a medical facility. During heat waves, ambulances and emergency personnel should carry ice and cool intravenous fluids. Any patient with a depressed level of consciousness or inadequate ventilation should be electively intubated to protect the airway and because vomiting, aspiration, and seizures are common during cooling. All patients should be given supplemental oxygen. Rectal temperature is inadequate and tympanic temperature may lag behind core temperature for some time[50]. Core temperature should be measured and monitored with a rectal thermistor probe inserted to about 19 cm. Large bore intravenous access should be established. Crystalloid infusion with normal saline should be given while labs are being processed. The amount of intravenous fluid needed can be assessed by blood pressure, urine output, and central venous pressure after cooling is started. Before cooling, hypotension may be due to vasodilatation which may be reversed quickly with cooling. This may lead to pulmonary edema if excessive fluids are transferred quickly to the central circulation by cooling induced vasoconstriction. This is especially true in patients with congestive heart failure or coronary artery disease. In general, patient with classical heatstroke will not need more that one or two liters of saline. Patients with exertional heatstroke, however, may need massive volume repletion if the heat stroke is severe and complicated by rhabdomyolysis because of third spacing into damaged tissues and capillary leaking. Vasopressors should be used cautiously because they may worsen peripheral vasoconstriction and prevent rapid heat loss through the skin. Patient with rhabdomyolysis may develop severe electrolyte and metabolic abnormalities (hypokalemia, hypocalcemia, hyperuricemia, lactic acidosis, renal failure) which should be treated aggressively. Disseminated intravascular coagulation can be a serious consequence of heat stroke, especially of the exertional type. Seizures may occur, especially during rapid cooling so airway protection is critical. Seizures can be treated with benzodiazepines. Antipyretics are ineffective in lowering body temperature in heat stroke. Steroids theoretically can counter some of the pro inflammatory responses induced by heat stroke, but their efficacy in humans with heatstroke has not been established. Dantrolene may be considered in some patients[51].

Several methods of rapid cooling have been investigated (table 7). Since rapid cooling is critical, many have advocated immersion in an ice water bath and massaging the skin to facilitate heat transfer as the best cooling method. While it is probably the fastest method of cooling, several disadvantages of this procedure are obvious and listed in table 8.

Table 7 Cooling Methods

- Removal from hot environment
- Cooling blanket
- Ice bags to surface of body
- Iced saline gastric or colonic lavage
- Cool intravenous fluids
- Ice water immersion
- Moistened skin with fanning
- Ice-solution peritoneal lavage

Table 8 Disadvantages of ice water cooling

- Uncomfortable to patient and treating personnel
- Vasoconstriction
- Shivering
- Prevents monitoring
- CPR is difficult
- May increase vagal tone

Ice moistened skin with fanning should be used immediately when the diagnosis is suspected while a more rapid cooling method is prepared. Any method that increases evaporation can be used during transport to the hospital if ice is not available. While removing the patient from the hot environment is important, it is not adequate to cool patients with either type of heat stroke. While cooling blankets and ice applied to surface of the body are good cooling measures to be instituted during transport, their induced cooling rate is unpredictable. Consequently, more aggressive cooling measures should be used. Iced-saline gastric and colonic lavage are not very effective methods of cooling and may induce gastric or colonic dilatation.

Some have used a more natural method of cooling using evaporative heat loss. This method sprays warm atomized water on the skin while blowing warm air across the patient. This quickly evaporates the water as it makes contact with the skin drawing heat from the body. It causes no vasoconstriction if skin temperature is not significantly lowered below about 32°C. A device to generate a atomized spray of water kept at a temperature of 15°C and a draft of air at

45°C with the capability of monitoring skin temperature to prevent excessive cooling of the skin and subsequent vasoconstriction and shivering has been developed. Although effective, the equipment to do this is expensive and not readily available.

Internal methods of cooling have been used in patients with severe hyperthermia and in patients who have failed to cool with external methods. These methods include cold water lavage of the stomach and colon, peritoneal lavage, cardiopulmonary bypass. None of these methods have been shown to be more effective than ice water external cooling.

Shivering is a common result of many methods of cooling, especially those using ice. During cooling, some have advocated the use of Thorazine to inhibit shivering. It is effective but causes hypotension and should not be used. Temperature must be monitored closely to document cooling and prevent hypothermia. Cooling should be stopped when core temperature reaches about 102°C.

Complications of Heat Stroke

Rhabdomyolysis is a common complication of exertional heat stroke. If severe, it can result in acute renal failure from myoglobinuria, hypotension from extracellular fluid sequestration in muscles, hyperkalemia, and respiratory failure from diaphragmatic weakness. Normal saline should be used to maintain blood pressure. Unfortunately, patients may require massive fluid resuscitation with susequest severe edema and muscle compartment syndromes. Acute renal failure may be averted with mannitol infusion or careful bicarbonate infusion to alkalinize the urine and consequently decrease the tubular toxic effects of myoglobin. However, care must be exercised to avoid serum alkalosis which can induce tetany if hypocalcemia is present. Furosemide may also be used to maintain urine output.

Central nervous system dysfunction is common after recovery from heat stroke. In the exertional type it is usually transient and mild. However, permanent brain injury is a common result of classic heat stroke and may result in cerebellar dysfunction, stroke, cognitive dysfunction and dementia.

The hypokalemia of classic heat stroke is primarily due to the respiratory alkalosis and usually requires no treatment. Hyperkalemia from acute renal failure and muscle necrosis should be treated aggressively with dialysis or agents that lower potassium. Hyperuremia may be seen in exertional heat stroke

and it may be severe. However, uric acid usually returns to normal as renal function improves. Lactic acidosis is common in exertional heat stroke. Patients with lactic acidosis and classic heat stroke have a poor prognosis.

Some patients suffer from chronic heat intolerance after heat stroke, especially if multiple episodes have occurred[52,53]. This is probably due to alterations in hypothalamic function or sweat gland dysfunction that occur after some heat strokes; however, the precise mechanism is unknown.

SUMMARY

Heat-related illness remains a significant health problem in the United States. The very young and old, the poor, and persons exercising in the heat are at the highest risk, especially when heat exposure is acute and inadequate acclimatization occurs. The best treatment for heat-related disorders is to prevent them with adequate fluid intakes and providing a relatively cool environment with adequate wind movement and circulation.

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