

Heat Stroke

T. K. Dutta, R. Sahoo

Introduction

Heatstroke is the most severe form of the heat-related illnesses and is caused by an excessive rise in deep body temperature due to thermoregulatory failure. It is characterized primarily by hyperthermia usually with core temperature above 40.6° C (105° F), central nervous system dysfunction, metabolic derangement and coma¹. Heat stroke is the least common but most serious form of heat disorder. It needs to be distinguished from heat exhaustion, a benign condition. Unlike heat exhaustion, it carries a high mortality if effective treatment is not given immediately (Table 1).

Table 1 : Differentiating features between heat exhaustion and heat stroke

	Heat exhaustion	Heat stroke
Body temperature	37-40° C	> 40.6° C
Skin	Moist	Dry
Sweating	Profuse	Absent
Hydration	Dehydrated	Normal
Pulse	Weak	Full-bound and strong
CNS symptoms	Absent	Delirium, Convulsions, Coma
Prognosis	Good	May be life threatening

Two forms of heatstroke exist, 'classical' non-exertional heatstroke (NEHS) and Exertional heatstroke (EHS)².

Epidemiology

With the influence of global warming, it is predicted that the incidence of heatstroke cases and fatalities will become more prevalent. The exact data on incidence of heat stroke is not precise. Exertional heatstroke (EHS) generally occurs in young and fit individuals, usually military recruits, athletes who engage in strenuous physical activity for a prolonged period of time in a hot environment. Approximately 40-50 cases of heatstroke occur per annum in Indian Armed forces³. Lack of acclimatization, wearing of heavy / inappropriate clothings and dehydration are important predisposing factors for exertional heatstroke. Deployment of troop in desert terrain causes an understandable increase in number of such cases³.

Classical non-exertional heatstroke (NEHS) more commonly affects sedentary elderly individuals, persons who are chronically ill, and very young persons. Classic NEHS occurs during environmental heat waves and is more common in areas that have not experienced a heat wave in many years. Both types of heatstroke are associated with a high morbidity and mortality, especially when therapy is delayed. Risk factors that increase

the likelihood of heat-related illnesses include a preceding viral infection, dehydration, fatigue, obesity, lack of sleep, poor physical fitness and unacclimatization². While non-acclimatization is a risk factor for heatstroke, EHS also can occur in acclimatized individuals who are subjected to moderately intense exercise.

Causes²

Increased heat production

- Increased metabolism
- Infections, Sepsis
- Encephalitis
- Stimulant drugs
- Thyroid storm

Increased muscular activity

- Exercise
- Convulsions
- Tetanus
- Sympathomimetics
- Thyroid storm
- Moderate physical exercise, convulsions, and shivering can double heat production and result in temperature elevations that generally are self-limited and resolve with discontinuation of the activity.
 - Strenuous exercise and status epilepticus can increase heat production 10-fold and, when uninterrupted, can overwhelm the body's heat-dissipating mechanisms, leading to dangerous rises in body temperature.
 - Stimulant drugs, including cocaine and amphetamines, can generate excessive amounts of heat by increasing metabolism and motor activity through the stimulatory effects of dopamine, serotonin and norepinephrine. The development of heatstroke in individuals intoxicated with stimulants is multifactorial and may involve a complex interaction between dopamine

and serotonin in the hypothalamus and the brain stem.

Decreased heat loss

- Reduced sweating
 - Dermatologic diseases
 - Drugs
 - Burns
- Reduced CNS responses
 - Advanced age
 - Toddlers and infants
 - Alcohol
 - Barbiturates
 - Other sedatives
- Reduced cardiovascular reserve
 - Elderly persons
 - Beta-blockers
 - Calcium channel blockers
 - Diuretics
 - Cardiovascular drugs - Interfere with the cardiovascular responses to heat and, therefore, can interfere with heat loss
- Drugs
 - Anticholinergics
 - Neuroleptics
 - Antihistamines
- Exogenous factors
 - High ambient temperatures
 - High ambient humidity

Reduced ability to acclimatize

- Children and toddlers
- Elderly persons
- Diuretic use
- Hypokalemia

Table 2 : Predisposing Factors For Heat Stroke⁴

- Ambient temperature $\geq 35^{\circ}\text{C}$ (e.g. during heat wave)
- Humidity $> 75\%$
- Extremes of age
- Obesity
- Alcohol intake
- Febrile illness
- Sleep deprivation
- Non-acclimatization
- Inappropriate clothing - which prevents dissipation of heat
- On Drugs – Diuretics, phenothiazines, antiparkinsonians, tricyclic antidepressants

Reduced behavioral responsiveness

Infants, patients who are bedridden, and patients who are chronically ill are at risk for heatstroke, because they are unable to control their environment and water intake.

Pathophysiology

Despite wide variations in ambient temperatures, humans and other mammals can maintain a constant body temperature by balancing heat gain with heat loss. When heat gain overwhelms the body's mechanisms of heat loss, the body temperature rises, and a major heat illness ensues. Excessive heat (usually temperature $> 42.2^{\circ}\text{C}$ [108°F]) denatures proteins, destabilizes phospholipids and lipoproteins, and liquefies membrane lipids, leading to cardiovascular collapse, multi-organ failure due to cellular death, and, ultimately, death. The exact temperature at which cardiovascular collapse occurs varies among individuals because coexisting disease, drugs, and other factors may contribute to or delay organ dysfunction. Full recovery has been observed in patients with temperatures as high as 46°C , and death has occurred in patients with much lower temperatures. Temperatures exceeding 41.1°C (106°F) generally are catastrophic and require immediate aggressive therapy.

Under normal physiologic conditions, heat gain is counteracted by a commensurate heat loss. This is orchestrated by the hypothalamus, which

functions as a thermostat, guiding the body through mechanisms of heat production or heat dissipation, thereby maintaining the body temperature at a constant physiologic range. In a simplified model, thermo-sensors located in the skin, muscles, and spinal cord send information regarding the core body temperature to the anterior hypothalamus, where the information is processed and appropriate physiologic and behavioral responses are generated. Physiologic responses to heat include an increase in the blood flow to the skin (as much as 8 L/min), which is the major heat-dissipating organ; dilatation of the peripheral venous system; and stimulation of the eccrine sweat glands to produce more sweat.

As the major heat-dissipating organ, the skin can transfer heat to the environment through conduction, convection, radiation, and evaporation. Radiation is the most important mechanism of heat transfer at rest in temperate climates, accounting for 65% of heat dissipation, and it can be modulated by clothing. At high ambient temperatures, conduction becomes the least important of the four mechanisms, while evaporation, which refers to the conversion of a liquid to a gaseous phase, becomes the most effective mechanism of heat loss.

The efficacy of evaporation as a mechanism of heat loss depends on the condition of the skin and sweat glands, the function of the lung, ambient temperature, humidity, air movement, and whether or not the person is acclimated to the high temperatures. For example, evaporation does not occur when the ambient humidity exceeds 75% and is less effective in individuals who are not acclimatized. Nonacclimatized individuals can only produce 1 L of sweat per hour, which only dispels 580 kcal of heat per hour, whereas acclimatized individuals can produce 2-3 L of sweat per hour and can dissipate as much as 1740 kcal of heat per hour through evaporation. Acclimatization to hot environments usually occurs over 7-10 days and enables individuals to reduce the threshold at which sweating begins, increase sweat production, and increase the capacity of the sweat glands to reabsorb sweat sodium, thereby increasing the efficiency of heat dissipation.

Table 3 : Presentation of 'classical (non-exertional)' and 'exertional' heat stroke

	Classical (NEHS)	Exertional (EHS)
Age group	Infants, elderly	15-65 yrs
Health status	Acute/chronic illness	Usually healthy
Activity	Sedentary	Usually highly active e.g. athletes, army personnel
Drug use	Diuretics, Antiparkinsonians, Anticholinergics, Tricyclic antidepressants	-

When heat gain exceeds heat loss, the body temperature rises. Classic heatstroke occurs in individuals who lack the capacity to modulate the environment (e.g. infants, elderly individuals, individuals who are chronically ill). Furthermore, elderly persons and patients with diminished cardiovascular reserves are unable to generate and cope with the physiologic responses to heat stress and, therefore, are at risk of heatstroke. Patients with skin diseases and those taking medications that interfere with sweating also are at increased risk for heatstroke because they are unable to dissipate heat adequately. Additionally, the redistribution of blood flow to the periphery, coupled with the loss of fluids and electrolytes in sweat, place a tremendous burden on the heart, which ultimately may fail to maintain an adequate cardiac output, leading to additional morbidity and mortality.²

Factors that interfere with heat dissipation include an inadequate intravascular volume, cardiovascular dysfunction, and abnormal skin. Additionally, high ambient temperatures, high ambient humidity, and many drugs can interfere with heat dissipation, resulting in a major heat illness. Similarly, hypothalamic dysfunction may alter temperature regulation and may result in an unchecked rise in temperature and heat illness.

On a cellular level, many theories have been hypothesized and clinically scrutinized. Generally speaking, heat directly influences the body on a cellular level by interfering with cellular processes along with denaturing proteins and cellular membranes. In turn, an array of inflammatory cytokines and heat shock proteins (HSPs), HSP-70 in particular, which allows the cell to endure the stress of its environment), are produced. If

the stress continues, the cell will succumb to the stress (apoptosis) and die. Certain preexisting factors, such as age, genetic makeup, and the nonacclimatized individual, may allow progression from heat stress to heatstroke, multiorgan-dysfunction syndrome (MODS), and ultimately death. Progression to heatstroke may occur through thermoregulatory failure, an amplified acute-phase response, and alterations in the expression of HSPs. Thermoregulatory failure switches off vasodilatation and sweating which leads to anhidrosis and subsequent rapid rise of body temperature causing cellular death of brain, liver, kidney and muscle. In brain, there occurs petechial hemorrhage and cerebral edema.

Clinical features

There are essential differences between presentation of 'classical (non-exertional)' and 'exertional' heat stroke¹ (Table 3).

Signs and Symptoms of Heatstroke^{5,6,7,8}

Onset - Sudden

Core temperature (rectal): > 40.6° C or >105° F (occasionally upto 45° C [113° F])

Signs	Symptoms
Anhidrosis	Prodrome (only in 25% cases)
Rapid and shallow breathing	- Nausea
Full bound pulse	- Vomiting
Mild BP	- Irritability
Confusion	- Dizziness
Slurred speech	Irritability
Coma	Seizures
Ataxia	

Table 4 : Management of heat stroke**Management***External cooling –*

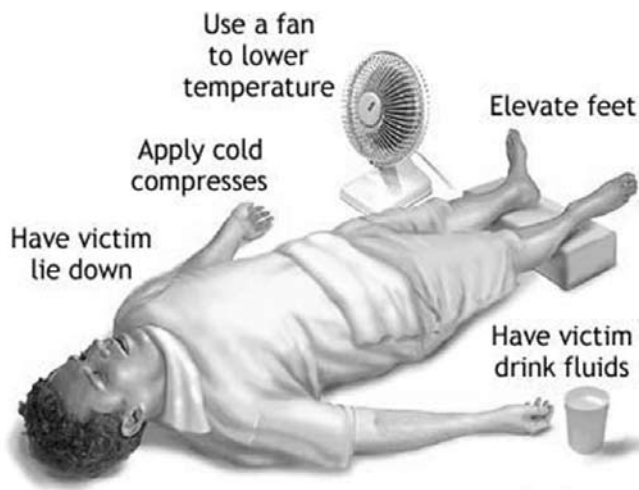
Heat loss by:

- Evaporation – sprinkling water
- Convection – fanning
- Conduction –
 - Immersion in ice water (not preferred since limits heat loss due to intense peripheral vasoconstriction)
 - Putting ice packs in axilla, neck and groin

*Internal cooling -*

- Refrigerated intravenous saline infusion
- Cold water lavage - gastric, peritoneal
- Extra-corporeal cooling – cardiac bypass, hemodialysis

Most preferred practice – sprinkling water at 20° C over the body combined with fanning/blowing of warm air. Body cooling unit (BCU) equipment may also be used for this purpose (Fig.1)¹.

**Complications:**

Ventricular fibrillation
 Disseminated intravascular coagulation
 Hepatic failure
 Pulmonary edema
 Renal failure
 Rhabdomyolysis

Figure 1 : Body cooling unit for treatment of heatstroke.**Shock**

Lactic acidosis

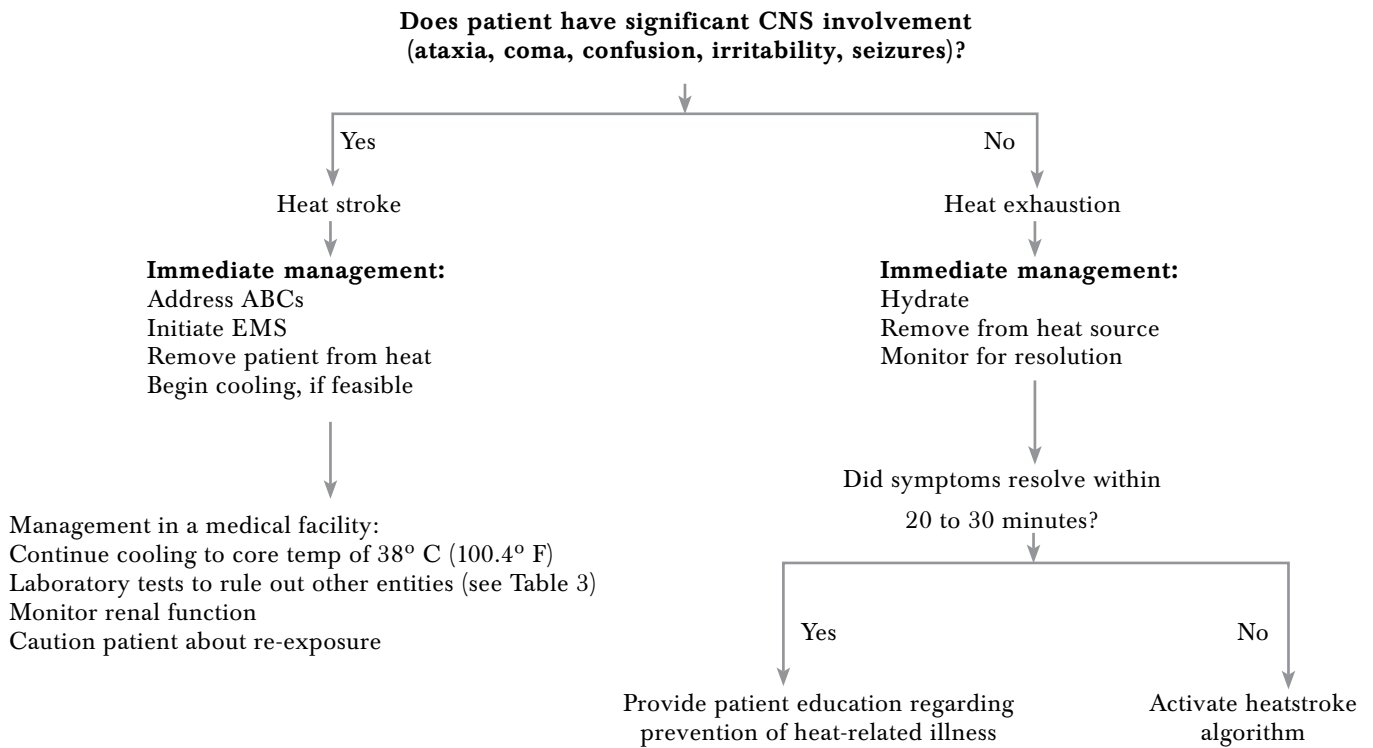
Multiorgan failure

Management**Heat stroke first aid**

- Move the victim to a cool place. Remove heavy clothing; light clothing can be left in place.
- Immediately cool the victim by any available means, such as placing ice packs at areas with abundant blood supply (neck, armpits, and groin where major blood vessels pass). Wet towels or sheets are also effective. The clothes should be kept wet with cool water.
- To prevent hypothermia continue cooling the victim until their temperature drops to 102 degrees Fahrenheit.
- Keep the victim's head and shoulders slightly elevated.
- Seek medical attention immediately. All heat stroke victims need hospitalization.
- Care for seizures, if they occur.
- Do not use aspirin or acetaminophen.

Heat stroke in hospital

In the hospital, following line of treatment (as shown in Table 5) can be adopted.^{1,9,10}

Algorithm for a hyperpyrexia patient¹¹**How to Beat the Heat**

- Wear light, loose clothes.
- Drink plenty of fluids.
- Stay in the shade or indoors.
- Avoid foods that are high in protein which increase metabolic heat.
- Check on elderly friends and neighbors each day.
- Keep strenuous activities to a minimum or do them in the coolest part of the day.
- Never leave children and pets in automobiles.
- Bring pets indoors and give them plenty of water.

Prognosis

Recovery is rapid in exertional heat stroke (EHS); patient recovers fully within half an hour of rectal temperature being brought down to 38° C (100.4° F) i.e. approximately 1-2 hours after starting treatment. However, if initial temperature is > 42.2° C (108° F) (temperature at which cellular death occurs), there is 80% mortality. In classic heat stroke (NEHS), there may be a lucid interval of 12-24 hours after which the patient may again deteriorate.

Summary

Heat stroke (HS) is a life threatening emergency characterized by high fever (core temperature > 40.6° C [105° F]), which leads to absence of sweating, dry hot skin and sudden loss of consciousness, because of failure of thermostat mechanism of hypothalamus. Primarily of two types, 'classical' or non-exertional heat stroke (NEHS) and exertional heat stroke (EHS), EHS has better prognosis since it attacks an otherwise healthy individual like an athlete or a soldier. Observed at an ambient temperature of ≥ 35° C, classical HS (NEHS) attacks individuals at extremes of age, who may be having an underlying illness. The condition is complicated by hepatocellular failure, acute renal failure, disseminated intravascular coagulopathy (DIC) and multiorgan failure (MOF), if continues. Treated promptly, almost all EHS patients recover within one to two hours of starting treatment, which is in form of sprinkling water (20° C) all over the body followed by fierce fanning over the same area. Putting ice pack in axilla, neck and

groin is also practiced to bring down temperature rapidly. In a sophisticated setting, patient is placed in a body-cooling unit (BCU); however, if initial temperature is $> 42.2^{\circ}\text{C}$ (108°F), there is usually 80% mortality.

References

1. Cook GC, Zumla A. Heatstroke, Text Book of Manson's Tropical Disease. 21st Edition 2003:550-554.
2. Robert SH, Rania H. Heatstroke. www.emedicine.com/med/topic956.htm, Updated on June 6, 2005.
3. Anand KP, Sashindran VK. Heatstroke (Newer Insight into Old disease). *Medicine Update* 2003;13:983-986.
4. Killborne G. Risk factors of heat stroke- a case control study. *JAMA* 1982; 247: 24-33.
5. Bouchama A, Knochel JP. Heatstroke. *N Engl J Med* 2002; 346:1978-88.
6. Lee-Chiong TL Jr, Stitt JT. Heatstroke and other heat-related illnesses. The maladies of summer. *Postgrad Med* 1995; 98:26-8,31-3,36.
7. Hassanein T, Razack A, Gavalier JS, Van Thiel DH. Heatstroke: its clinical and pathological presentation, with particular attention to the liver. *Am J Gastroenterol* 1992; 87:1382-9.
8. Parsons LR. Surviving the hot zone. *Emerg Med Serv* 1993; 22:42-6.
9. Hanlon P, Byers M, Walker BR, Summerton C. Environmental and nutritional factors in disease. In: Boon NA, Colledge NR, Walker BR eds. Davidson's Principles and Practice of Medicine, 20th edn., Churchill Livingstone, Edinburgh; 2006: 93-127.
10. Cohen R, Moelleken RW. Disorders due to physical agents. In: Tierney LM, McPhee SJ, Papadakis MA eds. Current Medical diagnosis & Treatment. 45th Edn. Lange Medical Book/McGraw Hill, New York, 2006; 1589-1606
11. www.aafp.org/afp/20050601/2133.html, Management of Heatstroke and Heat Exhaustion - June 1, 2005 - American Family Physician.htm