UK HEMS

Clinical Excellence in Helicopter Medicine

HEAT-RELATED ILLNESS

Background

Heat illness is a major cause of preventable morbidity worldwide, especially in regions characterized by high ambient temperatures. Minor forms of heat illness are namely heat cramps and heat syncope. The major heat-related illnesses - heat exhaustion and heatstroke - exist along a continuum of severity caused by dehydration, electrolyte losses and eventually failure of the body's thermoregulatory mechanisms.

Heat exhaustion is an acute heat injury with hyperthermia caused by dehydration. It occurs when the body no longer can dissipate heat adequately because of extreme environmental conditions or increased endogenous heat production. It may progress to heatstroke when the body's thermoregulatory mechanisms become overwhelmed and fail. Heatstroke is extreme hyperthermia with thermoregulatory failure. The condition is characterized by serious multi-organ damage and universal involvement of the CNS.

Heatstroke traditionally is divided into exertional and classic varieties, which are defined by the underlying etiology but are clinically indistinguishable. Exertional heatstroke typically occurs in younger athletic patients who exercise vigorously in elevated ambient temperatures until the body's normal thermoregulatory mechanisms are overwhelmed. Classic heatstroke more commonly occurs in older patients or in patients with underlying illnesses who are exposed to extreme environmental conditions.

Risk of death is directly related to peak temperature and duration of exposure. The male-to-female ratio is 1:1. Elderly persons are at increased risk for heat-related illness because of underlying illness, medication use, declining adaptive thermoregulatory mechanisms, and limited social support networks. Neonates have an increased risk of heat-related illness because of poorly developed thermoregulatory mechanisms.

The mortality rate in patients with heatstroke has been reported to be 10-70%, with the highest number of deaths occurring when treatment is delayed for more than 2 hours.

Pathophysiology

Heat transfer to and from the body occurs via the following 4 mechanisms:

- Conduction is the transfer of heat via direct physical contact; it accounts for 2% of the body's heat loss.
- Convection is the transfer of heat from the body to the air and water vapor surrounding the body; it accounts for 10% of the body's heat loss. When air temperature exceeds body temperature, the body gains heat energy.
- Radiation is the transfer of heat via electromagnetic waves; it accounts for most heat dissipation. As long as air temperature is less than body temperature, 65% of the body's heat is lost by radiation.
- Evaporation is the transfer of heat by transformation of a liquid into a vapor; it accounts for 30% of the body's heat loss.

The body's dominant forms of heat loss in a hot environment are radiation and evaporation. However, when air temperature exceeds 35°C, radiation of heat from the body ceases and evaporation becomes the only means of heat loss. An individual exercising in the heat easily can sweat 1-2 L/h. If humidity reaches 100%, evaporation of sweat is no longer possible and the body loses its ability to dissipate heat.

Initially, the body attempts to lower the core temperature via renal and splanchnic vasoconstriction with concomitant peripheral vasodilatation, thereby shunting blood to the periphery. Eventually, the vasoconstriction needed to keep the blood in the periphery fails; cutaneous (peripheral) blood flow decreases, less heat is carried away from the core, and hyperthermia results. This hyperthermia causes cerebral edema and cerebrovascular congestion, which culminate in increased intracranial pressure (ICP). This increased ICP combined with a decreased mean arterial pressure (from the failure of renal and splanchnic vasoconstriction and decreased peripheral resistance) causes cerebral blood flow to decrease. This is manifested clinically as CNS dysfunction.

Tissue damage during heatstroke is believed to result from uncoupling during oxidative phosphorylation, which occurs when the temperature exceeds 42°C. As energy stores are depleted because of the uncoupling, cell membranes become more permeable and sodium influx into cells is increased. The declining energy reserves impair thermoregulatory mechanisms, the body loses its ability to dissipate heat, and clinical signs of heatstroke appear. Proteins begin to denature at higher temperatures, with resultant widespread tissue necrosis, organ dysfunction, and organ failure.

Risk factors

Patients suffering from one or more of the following conditions are more susceptible to developing major heat-related illnesses:

- Heart disease
- Skin diseases (scleroderma, ectodermal hyperplasia)
- Extensive burns
- Dehydration (vomiting, diarrhea)
- Endocrine disorders (hyperthyroidism, diabetes, pheochromocytoma)
- Neurologic diseases (autonomic neuropathies, parkinsonism, dystonias)
- Delirium tremens
- Fever

Likewise, any of the below listed external factors increase the risk of developing hyperthermia:

- Exercise in a hot environment
- Lack of air conditioning or proper ventilation
- Inappropriate clothing (occlusive, heavy, vapor-impermeable)
- Lack of acclimatization
- Decreased fluid intake
- Hot environments (inside of tents or autos in the sun, hot tubs, saunas)

Finally, patients taking one or more of the following medications are at increased risk of developing major heat illness:

- Beta-blockers
- Anticholinergics
- Diuretics
- Ethanol
- Antihistamines
- Cyclic antidepressants
- Sympathomimetics (eg, cocaine, amphetamines)
- Phenothiazines
- Lithium
- Salicylates

Minor heat-related illness

Heat cramps:

Heat cramps are classically intermittent, of short duration and often excruciating. They are most commonly associated with prolonged exercise over several hours or days. They are thought do be due to relative or absolute sodium deficiency following excessive water (but not electrolyte) replacement after a period of dehydration. The treatment consists of fluid and electrolyte replacement, i.e. with isotonic drinks or in severe cases with intravenous fluids (Ringer's lactate, Hartmann's solution). Care must be taken to correct hyponatremia gradually, in order to avoid the development of cerebral oedema.

Heat syncope:

Heat syncope is a common problem and is classically seen in runners after completion of a run in the resting phase. Venous pooling of the circulating blood volume occurs due to extreme vasodilatation (exercise and heat) and is no longer counteracted by the calf muscle pump action during running. The subsequent reduction of venous return causes a fall in cardiac output and cerebral perfusion, resulting in syncope. The patient should be placed in the Trendelenburg position with about 30º of leg elevation in order to aid venous return to the heart and improve cerebral perfusion. Oral fluid intake should be encouraged, but intravenous fluid replacement is usually not necessary. Active cooling may or may not be necessary, depending on the core temperature.

Major heat-related illness

Heat exhaustion:

Symptoms are nonspecific and may be insidious in onset; these symptoms often resemble a viral illness and include:

- Fatigue and weakness
- Nausea and vomiting
- Headache and myalgias
- Dizziness
- Muscle cramps and myalgias
- Irritability

Physical signs and findings include:

- Muscle weakness
- Orthostatic pulse and blood pressure changes
- Sweating (absent or present)
- Piloerection
- Tachycardia
- Temperature usually less than 41°C (106°F).

Heatstroke:

This condition may be characterized by any or all of the symptoms and findings of heat exhaustion. The critical feature of heatstroke is CNS dysfunction, which has a sudden onset in 80% of cases.

The patient's temperature is usually higher than 41°C (106°F). Initial temperature readings may be normal or only marginally elevated if pre-hospital cooling measures were used.

Symptoms include bizarre behavior, hallucinations, altered mental status, confusion, disorientation, and coma. CNS dysfunction includes seizure, coma, delirium, opisthotonus, decerebrate rigidity, cerebellar dysfunction, oculogyric crisis, and fixed and dilated pupils.

The patient may exhibit signs of a hyperdynamic cardiovascular system, including tachycardia, increased pulse pressure, decreased cardiac output, decreased diastolic blood pressure. Tachyarrhythmias are not uncommon and may not be amenable to cardioversion.

Respiratory symptoms include tachypnea and respiratory decompensation secondary to acute respiratory distress syndrome (ARDS).

Coagulation disorders include disseminated intravascular coagulation (DIC) and result in signs such as purpura, conjunctival hemorrhage, melena, bloody diarrhea, hemoptysis, hematuria, myocardial bleeding, and CNS hemorrhage.

Skin findings may range from warm and dry to diaphoretic. Many individuals with temperatures higher than 41°C (106ºF) are sweating diffusely. Anhydrosis typically is a late finding in heatstroke and is more common in classic than in exertional heatstroke.

Genitourinary symptoms include hematuria (myoglobinuria), oliguria, or anuria that may occur as signs of acute renal failure.

Unlike malignant hyperthermia and neuroleptic malignant syndrome, heatstroke is not characterized by muscular rigidity. Muscle cramps or flaccidity may be noted.

Pre-hospital management:

Heat exhaustion:

- Treat heat exhaustion with rest, removal from hot environment, and correction of dehydration and electrolyte abnormalities.
- Patients may be cooled gently with cool packs applied to the neck, groin, and axilae.
- For mild cases, oral rehydration with 0.1% isotonic sodium chloride solution usually is adequate.
- For more severe cases characterized by orthostatic hypotension IV fluids may be required. The water deficit is best corrected slowly (one half of the total water depletion replaced in the first 3-6 h, with the remainder replaced over the next 6-9 h).
- Monitor vital signs, including pulse rate, blood pressure and urine output to guide fluid replacement.

Heatstroke:

- Rapidly conduct initial stabilization of airway, breathing and circulation. Administer supplemental oxygen and up to 1L of IV crystalloid while core temperature is determined and clothing is removed.
- Institute aggressive cooling measures as rapidly as possible to minimize end-organ damage. An ideal goal is to drop the patient's core temperature by 1°C (0.5ºF) every 15 minutes.
- Evaporative cooling is the preferred cooling method because it is safe, effective, easily accomplished and well tolerated. Undress the patient, wet the patient with any suitable fluid (0.9% saline would be ideal, dispensed from an IV bag with several small holes pierced through using a green or white needle and squeezing, providing several fine jets of fluid: note do not use glucose as this will make the patient sticky).
- Try to induce airflow over the patient by fanning, exposing to any breeze or even flying with the doors open if this is considered safe by the pilot and other crew members and adjusting the ventilators in the aircraft to blow onto the wet skin surface of the patient.
- Adjunctive measures include cool packs if available applied to the patient's neck, axillae, and groin as well as wet cooling blankets.
- Monitor vital signs, including pulse rate, blood pressure .

Appendix

System	Heat exhaustion	Heat stroke
CNS	Fatigue, headaches, dizziness, irritability	**Confusion**, reduced LOC, delirium, coma, cerebellar signs, seizures
CVS	Tachycardia, orthostatic hypotension	Hyperdynamic **shock**, increased pulse pressure
RS	Tachypnea, increased respiratory effort	Respiratory distress, pulmonary oedema
GUS	Oliguria, concentrated urine	Anuria, myoglobinuria, acute tubular necrosis
MS	Muscle weakness, flaccidity	Muscle cramps, flaccidity, rhabdomyolysis
Thermoregulation	**Temperature <41ºC** (106ºF), sweating and piloerection	**Temperature >41ºC** (106ºF), profuse sweating followed by anhidrosis

Table 1: Differential clinical findings for heat exhaustion versus heatstroke