AMERICAN COLLEGE OF EMERGENCY PHYSICIANS
SCIENTIFIC ASSEMBLY
1999

Temperature-Related Illness: Advances in the Management of Heat- and Cold-Related Injuries

This course will review the pathophysiology of temperature-related illness, the spectrum of clinical presentations, and the acute management required. Environmental hypothermia and heat illness, as well as malignant hyperthermia and the neuroleptic malignant syndrome, will be highlighted.

- Recognize the pathophysiology and presentation of illnesses related to cold and heat exposure, and how to manage each condition appropriately.
- Recognize risk factors for injury or illness related to extremes of temperature.
- Describe how to manage each condition appropriately.

TH-111
Thursday, October 14, 1999
11:00 AM - 11:55 AM
Room # N212
Las Vegas Convention Center

**Part Owner: Adventure Medical Kits*

FACULTY

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HEAT EMERGENCIES

MECHANISMS FOR ACCUMULATING HEAT

1. Basal metabolism alone can create a heat load of 65 to 85 kcal/hr, which would raise the body temperature 1.1 degrees centigrade/hr if mechanisms for dissipating heat did not exist.

2. Moderate work could increase this temperature rise by 500% (300-600 kcal/hr).

3. An individual in bright sun may gain 150 kcal/hr.

4. Prolonged exposure in hot tubs, saunas, and steam rooms can raise body temperature.

5. Elevated body temperature in turn impose its own intrinsic additional heat load since cellular metabolism will increase by 13% for every l-degree centigrade rise in body temperature.

6. Studies done in runners have shown that dehydration alone is capable of elevating body temperatures. This is probably due to an increased activity of the cellular sodium pump, which accounts for 20-45% of the basal metabolic rate (BMR).

7. When the air temperature is greater than the body temperature, radiant heat gain is possible.

MECHANISMS FOR DISSIPATING HEAT

1. Radiation, the transferring of heat from the body to a cooler environment, accounts for 65% of cooling as long as the air temuerature is lower than the body temuerature.

2. Normally, 30% of cooling results from evaporation of sweat. For each 1.7 ml of sweat evaporated, the body loses 1 kcal of heat. When the ambient temperature approaches 95 degrees Fahrenheit, evaporation prevails as the only mechanism that the body has to dissipate heat. If the humidity level should exceed 75%, evaporative heat loss potential will decrease. Sweat that drips from the skin only exacerbates dehydration without providing any cooling benefit.

*****TAKE HOME MESSAGE*****
--The combination of high temperature and high humidity blocks the two main mechanisms that the body has to dissipate heat. The conditions are right for “heat stroke.”

PHYSIOLOGICAL RESPONSES TO HEAT

1. Cutaneous blood vessels dilate to increase the surface cooling area.

2. To maintain blood pressure, in the face of this greatly decreased peripheral vascular resistance, cardiac output may double or quadruple, placing a strenuous load on the heart.

3. There is a compensatory vasoconstriction in the splanchnic vessels.

4. Sweat volume will increase.

5. Acclimatization to heat.

   a. In contrast to hypothermia, physiologic acclimatization to heat is possible. It usually takes 8-11 days to reach maximum benefit and requires some degree of exercise (at least 1 1/2 to 2 hours) each day.

   b. The mechanisms, although poorly understood, seem to be mediated through activation of the Renin-Angiotensin system with increased production of aldosterone. This results in sodium conservation in both urine and sweat with concomitant losses of potassium.

   c. Sweating is initiated at lower core temperatures and the amount of sweating may more than double.

   d. Three cardiovascular adaptations which result in enhanced delivery of heated blood from the core to the surface have been shown to occur: (1) An increased cardiac output; (2) A decreased peak heart rate; and, (3) An increased stroke volume.

   e. There occurs a marked increase in the density of mitochondria per unit muscle mass. This allows increased potential for oxygen utilization.

INDIVIDUAL CHARACTERISTICS WHICH PREDISPOSE SOMEONE TO HEAT

1. The elderly are less able to increase cardiac output for heat dissipation and are often dehydrated. Intrinsic diseases of the heart such as CAD, CHF, or previous MI would limit the ability to compensate for peripheral vasodilatation.

2. Neonates lack thermoregulatory and sweating capabilities.

3. Obese individuals have more insulation and less surface area-to-volume ratio with which to dissipate heat.

4. Hyperthyroidism can markedly increase metabolic rate with a rise in endogenous heat production.

5. Dermatologic disorders, as well as burns affecting large surface areas, may limit heat dissipation by sweating.

6. Various medications and drugs may predispose one to environmental heat illnesses.

   a. Beta-blockers will inhibit compensatory increases in cardiac output.

   b. Amphetamines, PCP, cocaine, and other stimulants can increase muscular activity with a resultant increased endogenous heat load.

   c. Anticholinergics such as phenothiazines, lithium, tricyclic antidepressants, antihistamines, and antispasmodics reduce sweating and can disrupt hypothalamic function.

   d. Diuretics may produce dehydration.

HEAT EXHAUSTION VS. HEAT STROKE

(See Table I)

Heat exhaustion and heat stroke are often discussed separately, implying that they are two different and distinct pathophysiologic entities. This is misleading because, with few exceptions, they define a continuum of one disease process. Heat exhaustion often presents with flu-like symptoms, including malaise, headache, anorexia, nausea, vomiting, and muscle cramps. Core temperatures are usually less than 41 degrees centigrade (106 degrees fahrenheit) and often are normal. Dehydration is almost always manifest.

Clinical signs may include orthostatic hypotension, tachycardia, diaphoresis, and moderate pyrexia Heat stroke represents the late stages of heat exhaustion as compensatory mechanisms for dissipating heat are failing. Decreased sweating due to dehydration and high-output cardiac failure contribute to a decompensating system for heat dissipation. Core temperatures may rise rapidly, producing cellular damage. A key point in differentiating heat exhaustion from heat stroke is that CNS function remains essentially intact in heat exhaustion. CNS dysfunction, such as delirium, ataxia, seizures and coma, would suggest heat stroke and mandate aggressive cooling measures. The exact temperatures at which cellular damage starts to occur is not clear, but oxidative phosphorylation becomes uncoupled at temperatures above 42 degrees centigrade (107.6 degrees fahrenheit). The resultant damage is both a function of the higher temperatures as well as the exposure time. Patients with higher temperatures for shorter periods may do better than those individuals who maintain more moderate temperatures for longer periods. Dry, hot skin is not mandatory to make the diagnosis of heat stroke. In one study of military recruits, 50% of the patients with heat stroke maintained their ability to sweat. Anhidrosis may be a late manifestation as a result of profound dehydration and necrotic plugging of sweat gland ductules. Patients with heat stroke almost always manifest signs of tachypnea, hypotension and sinus tachycardia. The cerebellum is most sensitive to heat and ataxia may be an early clue.

*****TAKE HOME MESSAGE*****

---Heat exhaustion and heat stroke are probably a continuum of heat disorders rather than distinct pathophysiologic entities. In the setting of heat illness, patients with CNS dysfunction should be treated for heat stroke.

-Sweating may still be present in heat stroke.

COMPLICATIONS OF HEAT STROKE

1. Decreased renal perfusion can lead to acute tubular necrosis and renal failure.

2. Damage to muscle and rhabdomyolysis can produce myoglobinuria and exacerbate the nephropathy.

3. Hypoglycemia and hypocalcemia may occur.

4. Although hyperkalemia may be seen initially, total body potassium is usually decreased.

5. Markedly elevated liver enzymes are often seen. Values may be in the tens of thousands after 24 hours. Elevations to several thousand are often found even in some patients with heat exhaustion.

6. Bleeding, secondary to a consumptive coagulopathy, may occur. The precipitating factor is thermal damage to vessel endothelial cells with exposure Type III basement membrane collagen. A syndrome of DIC may occur at l-3 days after onset of heat stroke.

7. The only organ not reported to be directly damaged in patients suffering from heat stroke is the pancreas.

TREATMENT OF HEAT EXHAUSTION

1. Fluids, rest in a cool environment, glucose and conservative measures of cooling are essential treatment modalities. Urine should be monitored for rhabdomyolysis and a thorough neurological exam should be performed.

TREATMENT OF HEAT STROKE

Pre-Hosuital Care:

1. Begin cooling immediately at the scene. Remove clothing, spray any available liquid on the patient and fan to promote evaporative cooling. If nothing else is available, even urine can be used as a liquid medium. The fluid does not need to produce evaporative cooling.

2. Ice bags, chemical ice packs, or cold compresses should be placed in areas where large blood vessels come near the surface such as the neck, axilla, groin, and scalp. Studies using this technique have documented cooling rates of 0.1 degree centigrade / minute (5 times that of controls). This technique avoids generalized cutaneous vasoconstriction and shivering.

3. Nothing should be given orally.

4. Aspirin and Tylenol are not effective, and aspirin is contraindicated because of its effect on platelets and clotting. The hypothalamic set-point is not elevated as it is in fever.

5. Patients should be transported as quickly as possible to a medical facility.

***'*TAKE HOME MESSAGE**“”

---Heat stroke should be considered a life threatening emergency. Several studies have shown mortalitv rates from 30% to 80%.

Emeraencv Deuartment Care: The optimum method for cooling is controversial. Some large centers still use ice baths while others rely more on evaporative techniques. The different methods, along with experimental data on cooling rates, have been listed in Table II.

If the emergency facility is air-conditioned or in a non-humid environment, I prefer the evaporative techniques for cooling. The patient is fully undressed and sprayed with warm water to keep the skin temperature at approximately 32 degrees centigrade. Fans are used to maximiie evaporation. Ice packs can be simultaneously placed along the neck, axilla, and groin. This technique is safe and effective, and requires a minimum of preparation. It is also more practical for managing monitors, I.V.'s, endotracheal tubes and complications that might occur. In addition, it minimizes avoidance behavior and shivering. Most patients can be cooled to temperatures of 101 to 102 degrees fahrenheit in under 40 minutes using either the ice bath or evaporative techniques. Cooling should be discontinued when the temperature falls below 102 degrees fahrenheit in order to prevent hypothermia from occurring.

Administration of I.V. fluids, consisting of D51/2 normal saline or normal saline, should be guided by urine output, central venous or wedge pressures, and blood pressure. Initial laboratory work should include a blood gas, CBC, electrolytes, BUN, creatinine, liver enzymes, CPK, calcium, platelet count, PT, PTT, and FDP.

*****TAKE HOME MESSAGE*****

---Heat stroke victims should be cooled as rapidly as possible. The more rapid the cooling, the lower the mortality.

TABLE I

	HEAT EXHAUSTION	HEAT STROKE
YMPTOMS	Flu-like symptoms (Headache, vomiting, muscle cramps, anorexia)	Same
WEATING	Present	May be present or absent
NS SIGNS	CNS function unimpaired	CNS Dysfunction: 1. Bizarre behavior 2. Confusion, delirium 3. Ataxia 4. Seizure 5. Coma
'EMPERATURE	Usually less than 41 degrees Centigrade (106 degrees Fahrenheit), often normal	Usually 41 degrees Centigrade or greater

TABLE II

EXPERIMENTAL DATA ON COOLING

Method			
PERITONEAL LAVAGE (Dialysate at 6-10^0 C)	0.56^0 C/Min	FAST!	1. Time consuming to set up. 2. Inherent complications
EVAPORATIVE (15^0 H_2O spray 0.4 M/Sec Fan) 45^0 C Air	0.31^0 C/Min	1. Less shivering 2. Less vasoconstriction 3. Easier to monitor	1. Requires non-humid environment.
ICE WATER BATH	0.1-0	1. Effective in humid environment.	1. Tub not available 2. Management difficult 3. Shivering 4. Vasoconstriction
ICED GASTRIC LAVAGE (Iced H_2O) at 200 cc/min)	0.2^0 C/Min	1. Easy and rapid	1. Aspiration
ICE PACKS TO (Neck, Axilla, Groin)	0.1^0 C/Min	1. Easy and rapid	-----
COLD INHALED AIR BY IPPB	0.02^0 C/Min		

Hypothermia

Hypothermia is an abnormally low body temperature due to exposure to a cold environment. Core (rectal) temperature down to 90 degrees Fahrenheit (32 degrees centigrade) is considered mild to moderate hypothermia, while temperatures below this indicates profound or severe hypothermia. When the body's temperature falls below 83 degrees, the heart becomes very irritable and is prone to lethal irregularities such as ventricular fibrillation. Death from hypothermia is likely to occur at around 75-80 degrees. The lowest recorded core temperature in a surviving adult is 60.8°F. For a child it is 57°F.

Your thermostat is in your skin!

Your perception of whether you are cold or warm depends more on yours skin temperature than on your core temperature. Even when your core temperature is above normal, if your skin is cold you will "feel" cold and begin shivering (an involuntary condition in which your muscles contract rapidly to generate additional body heat). Conversely, if your core temperature is low but your skin is warm, you "feel" warm, and do not shiver despite being hypothermic.

If you warm a hypothermic individuals skin without providing any heat to the core (putting a chemical heat pad on the skin for example), you can extinguish the drive to shiver, and cause the blood vessels on the skin to dilate which will make him more hypothermic.

It is known that profoundly hypothermic victims sometimes rip off their clothes, prior to death. This phenomenon, known as "paradoxical undressing", occurs because the constricted blood vessels near the body's surface suddenly dilate when the core temperature reaches a certain level and produce a sensation of warmth at the skin.

How to Recognize and Treat Hypothermia

Hypothermia is often divided into mild and profound based on a victim's temperature and behavior. The distinction is important because the treatment and the worry factor are different. It can be hard to tell where one level starts and the other stops without a special low-reading thermometer. Certain signs and symptoms can often be used to gauge a victim's level of hypothermia.

Mild Hypothermia

At 95 **degrees,** a victim enters the zone of mild hypothermia.

Signs and symptoms

1) The victim feels cold and shivering reaches its maximum level.
2) The victim maintains a normal level of consciousness, is alert, and has normal or only slightly impaired coordination
3) At 93 degrees the victim develops apathy, amnesia, slurred speech, and poor judgment.

Treatment

1) Get the victim into shelter and insulate him from the cold.
2) Replace any wet clothing with dry insulated garments.
3) Give the victim warm food and lots of sugar containing fluids to drink. Elevating the core temperature of an average-size individual one degree requires consuming about 60 kilocalories worth of a hot beverage. Since a quart of hot soup at 140 degrees provides about 30 kilocalories, a victim would have to consume 2 quarts to raise his temperature one degree. The sugar content of the fluid, however, will provide added fuel for the victim's furnace so that he can generate his own internal heat.
4) Heat loss may be slowed by wrapping the victim in plastic bags or tarps as well as sleeping bags. Huddling together will reduce heat loss.
5) Resist the urge to use hot-water bottles or heat packs as they can turn off the shivering mechanism, and by themselves, add very little heat to the core. Instead, bring water to a boil and have the victim inhale the steam, or build a fire.

Profound hypothermia

At 90 degrees, a victim is profoundly hypothermia.

Signs and symptoms

1) The victim becomes weak and lethargic and has an altered mental state (disorientation, confusion, combative or irrational behavior, or coma).
2) The victim is uncoordinated (unable to walk a straight line, heel to toe, without stumbling)
3) At 88 degrees, the victim will stop shivering.
4) At 86 degrees, the victim's heart pumps less than two-thirds the normal amount of blood. Pulse and respirations will be half of normal.
6) At 83 degrees, the heart is very irritable and unstable and prone to developing irregularities, like ventricular fibrillation. The victim is in danger of sudden cardiac arrest. Rough handling of the victim increases the potential for this to happen.

Treatment

First-aid treatment is aimed at preventing any further cooling and at stabilizing the victim.

1) Handle the victim very gently. Rough handling may cause the victim's heart to fail.
2) Place the victim in a sleeping bag, or place blankets or clothing underneath and on top of him. Any heat that you can provide will probably not rewarm the victim, but will help prevent further cooling.
3) A victim with a significantly altered mental state should not be allowed to eat or drink because of the potential for choking and vomiting.
4) Rewarming is best done in a hospital, because of the potential complications associated with profound hypothermia. Professional assistance is usually needed to evacuate a profoundly hypothermic victim.

When to perform CPR

If the victim is breathing or has any pulse, no matter how slow, do not initiate CPR. If there is no sign of a pulse or breathing after one minute, what to do next depends on your situation.

1) If you're alone or with only one other person, cover the victim and place him in a protected shelter. (place insulation on top and underneath him). Both individuals should go for help and stay together for safety.
2) If there are multiple rescuers, and it is safe to stay with the victim, begin CPR, while at least two people go for help. Chest compressions should be done at one-half the normal rate for a profoundly hypothermic individual.

*Temperature related findings**

Core temperature (degrees F)	Characteristics
99.	Normal rectal temperature
98.6	Normal oral temperature
95	Maximum shivering
93	Poor judgment; slower movements
91.5	clumsy movements; apathy
88	Shivering stops; stupor; altered level of consciousness
83-86	Heart is irritable and prone to arrhythmias;
80	Voluntary motion ceases; pupils not reactive to light
72-77	Maximum risk of cardiac arrest

Hospital Management

Volume Expansion (500 ml. of 5% dextrose in warmed normal saline) may be the most important treatment after rewarming. It stabilizes the conduction system of the heart and decreases the incidence of ventricular fibrillation.

Warmed IV solutions or heated humidified oxygen can provide only a small, but important amount of heat.

If Vfib, Defibrillate once with 2wsec/kg. Defibrillation is rarely successful below 30 degrees C.

Passive external rewarming can be expected to raise the temp. 1.5 degrees C/hr.

Heated humidified oxygen raises the temp. 1 degree C. by mask and 1.5-2 degrees C. by ETT.

Closed thoracic lavage with two large bore thoracostomy tubes (36-40 Fr.) can produce rewarming rates of 2-3 degrees C/hr. One is placed anteriorly in the second to third ICS MCL. One is placed in the posterior axillary line at the fifth ICS.

Peritoneal Lavage: Normal Saline (10-20 ml/kg) heated to 40-45 degrees C. is infused and retained for 10-15 minutes and then aspirated. The usual exchange rate of 6-7 liters/hr. yields a rewarming rate of 2-3 degrees C/hr.

Gold standard remains extracorporal blood rewarming. Cardiopulmonary bypass is three to four times faster at rewarming hypothermic victims than any other techniques (9-10 degrees C./hr.)

Microwave Rewarming: low-frequency (13.56-40.68 MHz) microwave radiation. Average rewarming rate was 1 degree C./minute!!

Ventricular arrythmias: Lidocaine has never been proven to be effective either for prophylaxis or for treatment of ventricular fibrillation. The class III agent Bretylium Tosylate has been effective in several animal studies. In studies in dogs, Bretylium at doses of 15 mg/kg was found to prevent VF when administered before the dogs were made hypothermic.

Mg Sulfate at doses of 100mg/kg IV has converted hypothermic patients out of VF.

Negative Pressure Rewarming:

Negative pressure rewarming is a new technique that is based on the principle that thermoregulatory blood flow to descrete skin areas is regulated by arteriovenous anastamoses. With this technique, negative pressure is used to manipulate regional blood flow and open up constricted arteriovenous anastamoses. This technique thus overcomes peripheral vasoconstriction which limits the transfer of cutaneously applied heat.

The Thermo-STAT has been developed as non-invasive means of creating a direct thermal pipeline between the skin and body core. To accomplish this, the patient's forearm is fitted through an acrylic sleeve with a air tight seal around the arm. Vacuum pressure of -40 mm Hg is then established and the thermal load is applied via a chemical heating pad. Data will be presented from recent experiments with this device.