

Management of Heat Emergencies

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Objectives

At the end of the CE module, the EMS provider will be able to:

1. Understand the basic physiology of thermoregulation.
2. Understand the pathology of heat-related illnesses.
3. Formulate a treatment plan for the management of heat cramps, heat exhaustion and heat stroke.

Introduction

Heat-related illnesses exist on a continuum and range from mild discomfort to life-threatening neurological and cardiovascular dysfunction. Heat stroke, the most severe of the heat-related illnesses is often fatal but those who do survive frequently suffer permanent neurological injury. This article will familiarize EMS professionals with the pathophysiology and progression of heat-related illness while providing useful information for developing treatment plans for the various conditions.

Excessive heat is a known public health hazard (Kovats & Hajat, 2008). Many researchers have demonstrated an association between heat and a variety of health-related outcomes, including death (Anderson & Bell, 2009; Baccini et al., 2008; Basu, 2009; Basu & Samet, 2002). Mortality is more greatly affected by the temperature on any given day and to a much lesser degree, the duration of excessive heat days (Gasparrini & Armstrong, 2011).

During the summer of 1996, nearly 700 people in Chicago died as the result of heat-related emergencies (Semenza et al., 1996). More than half of the dead were 75 years of age or older. Seven years later, heat waves across Western Europe killed between 22 and 50 thousand people (Brucker, 2005) with almost 15 thousand deaths in France alone during a 14-day period (Dhainaut, Claessens, Ginsburg, & Riou, 2004). The elderly, again,

accounted for the most of the deaths (Brucker, 2005).

Introduction to thermoregulation

The human body attempts to keep its internal temperature within the very narrow range of 97.7°F (36.5°C) to 99.5°F (37.5°C) (Lewis, 2007). This process is called thermoregulation. If the internal temperature falls below the threshold, cells within the body convert stored energy into heat. On the other hand, if the temperature is too high, thermoregulatory mechanisms must rid the body of excess heat before cellular injury occurs. There are four ways for the body to do this.

Infrared radiation is a form of heat energy emitted by all objects. Humans cannot see infrared energy because it is below the spectrum of visible light. Human skin absorbs infrared radiation emitted by nearby objects causing the body temperature to rise. On the other hand, heat created within the body through cellular metabolism radiates away from our skin, heating the objects around us. Although half of our daily heat loss occurs through radiation (Martini, Bartholomew, & Bledsoe, 2002), this method of cooling is ineffective in a very warm environment since the body absorbs more heat than it emits.

Conduction occurs when the body transmits heat directly to another object through touch. Conduction requires physical contact between two objects, such as when a hyperthermic patient sits

on a cool, plastic bench. The heat from the body transfers to the bench.

Convection describes the movement of warmed objects and begins when the body conducts heat to the air molecules in contact with the surface of the skin. Since heat rises, the warmed air molecules float away carrying the heat with them. Cooler air molecules rush in to replace the warmer molecules near the surface of the skin. These molecules contact the skin, are heated and float away only to be replaced by cooler molecules starting the process again.

Evaporation involves the conversion of a liquid to a gas. Conduction heats the small beads of sweat that accumulate on the skin during warm weather. Some of the water molecules in the sweat absorb enough energy to break free from the surface of the droplet and float away or evaporate, carrying the energy away from the body.

Although radiation accounts for most of the heat loss from the body, evaporation is the most efficient cooling process and the primary method of cooling on warm days (Glazer, 2005). However, as the humidity rises, the efficiency of evaporation as a cooling mechanism decreases.

The body can adjust its internal physiology in an effort to maximize the efficiency of these primary methods of temperature reduction. For example, in warm weather, the brain sends vasodilatory signals to the blood vessels, especially those near the outside surfaces of the body. Upon receiving the signals, the vessels enlarge, thereby allowing more blood and consequently more heat to flow toward the surface of the skin, which increases the efficiency of all heat transfer mechanisms. In addition, the heat increases sweat production, which places more moisture on skin surfaces and maximizes evaporative efficiency.

The normal physiological response to hyperthermia is to increase cardiac output by as much as 20 liters per minute in an effort to divert the heated blood to the surface of the skin (Bouchama & Knochel, 2002). Patients with preexisting cardiovascular problems, such as the elderly, may not be able to increase cardiac output sufficiently to cool the body, making them more susceptible to the effects of hyperthermia.

Heat illnesses

It is important to recognize the difference between fever and heat illness. Fever is a normal body response that remains under control of the thermoregulatory centers of the brain (Jardine, 2007). Circulating proteins in the bloodstream called pyrogens are capable of resetting the body's internal thermostat, thereby raising the temperature. Within limits, this temperature elevation helps the body fight disease.

Heat illness, on the other hand, occurs

because the body's heat transfer mechanisms, which normally help keep the body cool, become ineffective. Without the heat transfer mechanisms, the thermoregulatory centers in the brain are quickly overwhelmed and the internal temperature rapidly rises to dangerous levels.

Prior to developing life threatening heat illnesses, patients will usually experience lesser degrees of discomfort. The least serious form of heat illness is heat stress, which is the psychological discomfort and physical stress felt by individuals in a hot environment (Jardine, 2007). Although the internal body temperature remains within normal limits, patients experiencing heat stress are often uncomfortable (Bouchama & Knochel, 2002). A slight decrease in physical performance may be the only symptom (Jardine, 2007).

The next step along the continuum is the development of heat cramps. Heat cramps are painful involuntary spasms that usually develop in the muscles of the calves, thighs and shoulders within several hours of vigorous exercise (Lugo-Amador, Rothenhaus, & Moyer, 2004). Although the exact nature of the spasms is unclear, researchers believe that as sweat loss is replaced by water, the concentration of sodium in body fluids falls, resulting in dilutional hyponatremia (Hubbard, Gaffin, & Squire, 1995; Olson & Benowitz, 1984; Wexler, 2002). The internal temperature of a patient experiencing heat cramps is often within normal limits, although slight elevations are not uncommon.

Once the body temperature starts to rise, the patient progresses to heat exhaustion (Jardine, 2007). Heat exhaustion produces a mild degree of dehydration with or without accompanying sodium and other electrolyte abnormalities (Jardine, 2007). Temperatures are above normal but do not generally rise above 104° F (40° C). Patients are sweating, often profusely, with complaints of thirst, nausea, headache and dizziness. There may be associated confusion and vomiting. Normally, however, patients experiencing heat exhaustion do not exhibit any major neurological deficits (Jardine, 2007).

The most serious of the heat-related illnesses is heat stroke. Heat stroke occurs when direct cellular injury results from an increased body temperature (Jardine, 2007). Prolonged internal temperatures of 105.8° F (41° C) or greater will cause a change in the structure of cellular proteins in a process known as denaturing. Once these proteins denature, they no longer function and the cell dies. If enough proteins within an organ denature, the organ is at risk for failure. Approximately 12 to 60 percent of people who suffer a heat stroke will die (Jardine, 2007; Bouchama, Dehbi, et al., 2007).

EMS professionals can distinguish between heat stroke and heat exhaustion by three characteristics. Heat stroke will have a history

of exposure to elevated ambient heat, an internal temperature greater than 104° F (40° C) and neurological dysfunction (Jardine, 2007). Patients suffering from heat stroke will be severely dehydrated and present with hot, dry and flushed skin (Bouchama & Knochel, 2002), although one should not consider the absence of sweat a diagnostic criterion (Lugo-Amador, Rothenhaus, & Moyer, 2004). In fact, loss of sweating is a rare clinical finding in exertional heat stroke (Epstein & Roberts, 2011).

Heat stroke is a multi-organ problem and the signs and symptoms are a reflection of the degree to which the organ systems are injured. One study demonstrated that one-third of heat stroke patients have failure in more than one organ system (Bouchama & De Vol, 2001). A hallmark of heat stroke is the presence of neurological dysfunction, which may range from confusion and delirium to coma and seizures. Generally, severe neurological dysfunction does not occur with rectal temperatures under 105.8° F (41° C) (Jardine, 2007). Patients with severe neurological dysfunction have a poor prognosis (Jardine, 2007).

Patients suffering from heat stroke often present in shock, especially when internal temperatures exceed 107.6° F (42° C). As many as 65 percent of heat stroke victims experience circulatory failure (Sprung, 1979). Early in the heat illness continuum, hypotension results from the vasodilation produced by the body in an attempt to cool the system. Continued fluid losses through incessant sweating, which can be greater than one to two liters per hour (Epstein & Roberts, 2011) produce dehydration that contributes significantly to the low blood pressure (Jardine, 2007). Although dehydration is not the direct cause of the heat stroke, its presence may impair cooling mechanisms and promote cardiovascular collapse (Epstein & Roberts, 2011).

Hyperthermia causes bleeding within the gastrointestinal tract, along with a release of harmful endotoxins. These toxins, along with other harmful substances, place the liver at risk for damage. A significant percentage of patients suffering from heat stroke develop renal insufficiency (Jardine, 2007). Severe hyperthermia shortens red blood cell half-lives, reduces the platelet count and produces disseminated intravascular coagulation.

Researchers classify heat stroke into two categories: exertional heat stroke and classic heat stroke (Epstein & Roberts, 2011). Exertional heat stroke occurs during physical activity and primarily affects young athletes and individuals engaged in strenuous work. Classic heat stroke occurs in individuals who physically lack the ability to escape the heat and may appear despite the absence of strenuous physical activity by the patient. This

type of heat stroke is more common during the summer months and especially during heat waves (Jardine, 2007).

Two age groups have an increased risk of developing classic heat stroke, the elderly and the sleeping infant. With the elderly, medical conditions such as diabetes and cardiovascular diseases may impair thermoregulatory efficiency while vasoconstrictors and diuretics may alter fluid balance, making the elderly more vulnerable to heat exhaustion with more rapid progression to heat stroke (Lewis, 2007). Bed-confined patients or those with mobility deficits may be incapable of self-hydration or movement to cooler locations. City dwellers are susceptible due to the “heat island effect” created by the concrete infrastructure, which produces environmental temperatures as much as 22° F higher than the surrounding countryside (U.S. Environmental Protection Agency, 2007). Other factors that appear to influence the susceptibility of this age group to heat include pre-existing illnesses, social isolation and lack of adequate air-conditioning (Centers for Disease Control and Prevention, 1995).

Infants lack the physical mobility skills to escape a warm environment. In addition, the sleeping infant may retain excessive heat if covered with a blanket during naptime. In that situation, the infant may not possess the physical maturity to shed the blanket with movement during sleep.

Both infants and children are at an increased risk for classic heat stroke if left unattended in a vehicle directly exposed to the sun or during warm weather. Studies of parked cars in the summer sunlight demonstrate an ambient temperature increase inside the vehicle from 97° F (36° C) to 153° F (67° C) in as little as 15 minutes (King, Negus, & Vance, 1981).

Exertional heat stroke results from the inability to dissipate excess heat produced during physical activity. While high ambient temperatures may contribute to the development of exertional heat stroke, the condition may develop during cooler months if the activity is strenuous. Exertional heat stroke is more common in adolescents and adults. In fact, among high school athletes, it is the third leading cause of death (Coris, Ramirez, & Van Durme, 2004). Exertional heat stroke is less common in children, which is likely the result of disengagement from physical activity due to the heat stress discomfort that precedes heat stroke. Adolescents and adults, on the other hand, may be sufficiently motivated to ignore those early warning signs and push their bodies to dangerous levels.

One common devastating systemic emergency that often accompanies heat illness is rhabdomyolysis (Dr. Gil Salazar, personal communication, August 4, 2011). During rhabdomyolysis, muscle cells disintegrate and

release massive quantities of toxic cell components and electrolytes into the bloodstream (Poels & Gabreels, 1993; Vanholder, Sever, Ereik, & Lameire, 2000). These substances damage the kidneys (Prendergast & George, 1993), contribute to dangerous electrolyte imbalances (Coco & Klasner, 2004) and significantly alter the acid-base balance of the bloodstream (Dayer-Berenson, 1994; Poels & Gabreels, 1993). In addition, massive amounts of fluid can accumulate in the damaged muscle (Curry, Chang, & Connor, 1989; Vanholder, Sever, Ereik, & Lameire, 2000) which can contribute to a profound cardiovascular collapse.

The most immediately life threatening complication of rhabdomyolysis is the cellular release of potassium (Bagley, Yang, & Shah, 2007). Rising potassium levels in the extracellular space can produce respiratory failure, life-threatening ventricular rhythms and asystolic cardiac arrest (American Heart Association, 2005).

Assessment and management

As with all emergency responses, responders must remain safe. The environmental factors that produce the emergency for the patient can create a threat to the responding medical team. Dr. Marshall Isaacs (personal communication, July 26, 2011), Assistant Medical Director for the BioTel system, stresses that medics must remain hydrated throughout the day and not wait for thirst or other symptoms before consuming fluids.

EMS professionals must carefully monitor and aggressively treat any patient suffering from a heat-related illness to prevent the development of heat stroke. If hot or warm patients display any alteration in mental status, medics must assume that heat stroke is present and manage that patient accordingly. Removing the patient from the overheated environment is often enough to begin the cooling process, although rescuers should also remove the victim's clothing while protecting the patient's modesty. If bystanders removed the patient from the offending environment prior to EMS arrival, the internal body temperature may appear normal but the cellular and organ damage has already occurred.

For patients experiencing heat cramps, treatment is supportive and consists of patient rest and oral rehydration with an electrolyte solution (Lugo-Amador, Rothenhaus, & Moyer, 2004). Intravenous administration of a saline solution is an effective alternative to oral rehydration and usually relieves the spasms and accompanying pain rapidly. It is usually not necessary to provide analgesics or antispasmodics, as administration of electrolytes will lessen the frequency and severity of the muscle spasms. The spasms often occur in isolation, however they may also present during heat exhaustion.

Treatment for patients suffering from heat exhaustion will vary depending upon the presentation. In general, medics should move heat exhaustion patients into a cool environment and remove the patient's clothing. If the patient is alert, has normal vital signs and no clinical evidence of dehydration or vomiting, oral rehydration with an electrolyte solution is usually sufficient.

If the patient fails to respond to conservative treatment, presents with vital sign abnormalities or vomits, intravenous fluid replacement is indicated. Begin with fluid boluses of 250-500 mL and reassess frequently. Medics should assess blood glucose levels and correct any deficits that might be present. Transport to the hospital is essential in order to prevent any complications from electrolyte or blood chemistry imbalances. The vast majority of patients suffering from heat exhaustion recover with no long-term problems (Lugo-Amador, Rothenhaus, & Moyer, 2004), however, if the symptoms do not resolve within 20 or 30 minutes or if significant neurological symptoms begin, the rescuer must strongly consider the possibility of heat stroke as a diagnosis.

Since heat stroke is considered a life-threatening condition, much more aggressive therapy is warranted. The single most important intervention for EMS personnel to perform for patients suffering from heat stroke is aggressive cooling. Morbidity and mortality are directly related to the duration of the elevated temperature (Chou, Lai, Lee, & Lin, 2003; Costrini, 1990; Costrini, Pitt, Gustafson, & Uddin, 1979; Shapiro & Seidman, 1990). Clinical studies have shown that death most often occurs shortly after the onset of the hyperthermia (Ferris, Blankenhorn, Robinson, & Cullen, 1938; Levine, 1969).

Similar to heat exhaustion, patients suffering from heat stroke must be moved out of the heat and into a cooler environment. However, medics must act much more aggressively to reduce the patient's temperature below 104° F (40° C). Historically, immersing the patient in ice water was considered the most effective method of reducing the patient's temperature quickly (Smith, 2005) with one case series demonstrating a mean temperature reduction to under 102.2° F (39° C) in less than 20 minutes (Costrini, 1990). However, for obvious reasons, this method may not be practical in the prehospital environment.

Many EMS treatment protocols and guidelines recommend the application of cold packs to the patient's neck, axillae and groin, however, a much more effective technique involves evaporative cooling. With this technique, EMS providers remove the patient's clothing and spray the patient with cool or tepid water. The medics then set a battery-operated fan to blow across the patient thereby enhancing evaporation and convection



(Smith, 2005). If a portable fan is not available, medics can manually fan the patient.

Another frequently used method of cooling the patient is the intravenous infusion of chilled saline. This method of cooling has the added advantage of rehydrating the patient who may also be suffering from fluid and electrolyte imbalance (Jardine, 2007). Fluid losses through sweating may approach two liters per hour or more in some patients (Bouchama & Knochel, 2002), although most will not need large volumes of saline replacement (Lugo-Amador, Rothenhaus, & Moyer, 2004).

However, infusion of cold saline is not without risk. The temperature difference between chilled saline and the patient's blood may be as high as 70° F. Dr. Ray Fowler (personal communication, August 3, 2011), Deputy Medical Director for the BioTel system warns that slamming these patients with chilled saline places them at risk for the development of life threatening arrhythmia. Dr. Fowler recommends that medics use room temperature fluids, as long as the saline bags are not as hot as the ambient temperature. Room temperature fluid cools the body efficiently and reduces the risk of arrhythmia.

Heat exhaustion and heat stroke treatment does not end with core temperature reduction. If the patient is exhibiting any signs that suggest rhabdomyolysis, such as altered mental status, arrhythmia or myocardial infarction, Dr. Salazar (personal communication, August 4, 2011) suggests that medics assume the condition is present and begin corrective action as soon as possible. This includes airway management with supplemental oxygen administration, IV fluid resuscitation of at least 1000 ml, continuous cardiac monitoring, blood glucose measurement and early transport to a facility capable of managing the condition.

The neurological disability associated with heat stroke may manifest as seizure activity. Fortunately, anticonvulsants such as the benzodiazepines are effective for controlling these seizures. Any observed respiratory failure is likely the result of neurological impairment instead of pulmonary pathology (Jardine, 2007).

Patients with mild heat stroke usually recover intact, especially if managed early and aggressively. The risk of complication increases with moderate heat stroke, and patients with an internal temperature greater than 107.6°F (42°C) have the poorest prognosis.

Summary

Although EMS personnel cannot prevent summer heat, morbidity and mortality resulting from its effects can be improved through prevention, proper assessment, and prompt and efficacious management. Fortunately, the most common heat-related illnesses are easily managed

with rest, relocation and oral rehydration, and they do not result in long-term consequences. However, for those patients who suffer from the most severe form of heat illness, the degree of disability is directly related to the amount of time the patient remains hyperthermic. For those patients aggressive cooling techniques are indicated and may mean the difference between full recovery and death.

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