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Heat Emergencies:
A Guide to Assessment and Management

Introduction. It is at this time of the year that the EMT and paramedic must review and prepare himself for the onslaught of medical emergencies that are peculiar to summer. I am referring to heat-related disorders: the 65-year-old woman with heart disease who is found in her hot, humid apartment, unconscious with hot, dry, reddish skin; and the 16-year-old aspiring National Football League linebacker who, during strenuous exercise in the scorching July sun, complains of severe cramps in his legs and abdomen. This article will review the basic pathophysiology, diagnosis, and treatment for heat-related disorders.

Pathophysiology. There are a number of different mechanisms that the human body utilizes to decrease body temperature. The most common method of heat reduction is radiation of body heat to the external environment. However, this method only reduces body heat when the ambient or external environment temperature is lower than that of the internal body temperature. When the ambient temperature increases above body temperature, heat radiation becomes a heat producer rather than a reducer.

Convection is another means of reducing body heat. Cooler air passes over the body, drawing heat from the body. The most important method of cooling is evaporation of sweat. Sweating is an active process which produces heat, but when sweat evaporates, heat is drawn from the body, reducing body temperature most effectively.

As mentioned earlier, radiation only works when the ambient temperature is below that of internal body temperature. Evaporation, on the other hand, can continue to work even when the external temperature surpasses the internal temperature. The external factor that most affects the process of evaporation is relative humidity. As the relative humidity increases, evaporation decreases; when relative humidity reaches 100%, evaporation ceases.

Overall, the body regulates temperature through two processes: biochemical and biophysical changes; and acclimatization. The biochemical and biophysical changes are mediated through the hypothalamus which puts into effect a number of physiological responses. An increase in the respiratory rate, which will increase body heat lost through exhaled air, increases cardiac output and skin vasodilation, thereby bringing more blood to the skin and periphery. This will decrease body temperature through radiation, convection, and evaporation. A rapid increase in the amount of sweat production is very inefficient because there is a large resultant loss of sodium chloride, and a concurrent rise in levels of antidiuretic hormone and aldosterone to offset the large loss of salt and fluids.

The second process is acclimatization, which is by far the most efficient method of handling increased external temperatures. Unfortunately, this method of handling increased external temperatures requires a period of several weeks to take effect. The body responds to acclimatization by increasing cardiac output, decreasing peripheral vascular resistance, and increasing circulating blood volume with the net result being an increase in sweat production, but a marked decrease in sodium chloride excretion.

Diagnosis. Now that we have reviewed the basic physiology behind summer heat-related emergencies, how do you, as the EMT or paramedic, diagnose these conditions? It is important to know that there are three clinically identifiable entities involved with derangement of the body's temperature regulation mechanism.

Heat Cramps. The first and most innocuous disorder is usually a result of profuse sweating over a short period of time with adequate replacement of H_2O, but inadequate replacement of sodium chloride. Since sodium plays a large part in muscular activity, the result of sodium depletion will usually be severe, painful muscular cramps. Usually, this patient will present as a young, well-conditioned person who has been engaged in strenuous physical activity in a hot environment. The most frequent and bothersome complaint is severe muscular cramping of leg and abdominal muscles. The patient will be somewhat agitated due to the muscular cramping. Skin will be wet and warm due to excessive sweating; temperature should be normal or slightly elevated, but under 100°F (37.7° C). Blood pressure will be slightly elevated due to exertion as will be the pulse and respiratory rate. Mental state should be clear and oriented in all spheres. Skin color should be normal.

Heat Exhaustion. This entity occurs over a somewhat longer period of time usually as a result of both fluid and salt depletion and will present as a form of

peripheral vascular collapse. This occurs due to the vascular system having tremendous demands placed upon it. These demands come from two different areas, the muscular mass and the brain, which due to increased work loads require increased blood flow. The skin also requires increased blood flow to facilitate radiation, convection, and evaporation, thereby aiding the reduction of body temperature. These patients present as being much more acutely ill than patients with heat cramps, but in reality they are not. These patients will complain of generalized weakness, vertigo, headache, nausea, and loss of appetite. Most of these symptoms are directly attributable to the increased vascular demands, which place the patient in a functional low flow state. It must be stated that patients with preexisting medical conditions, such as arteriosclerotic heart disease (ASHD), protracted vomiting or diarrhea states, will be predisposed to this clinical entity. These patients classically present with ash-gray color or clammy skin. Their body temperatures will usually be normal or possibly slightly below normal due to low flow state. Their blood pressure will be normal or slightly depressed. Pulse may be normal or slightly increased. Respiratory rate should be normal. Their mental state should be awake and oriented x three with expected complaints of headache.

Heat Stroke. Heat stroke (hyperpyrexia) is seen less frequently than other heat-related conditions, but if not recognized early and treated aggressively, has a mortality rate of up to 70%. Since these patients are generally comatose by the time the paramedics arrive, the diagnosis must depend very heavily on environmental and apparent past history factors as well as the physical examination. As mentioned earlier, radiation, convection and evaporation all cease when humidity is high and ambient air temperature reaches body temperature; therefore, we can conclude that patients will be in the most danger on extremely hot, humid, and windless days. Usually, it will take more than one day for most patients to develop symptoms; it is after two to three days of extreme temperatures (usually above 90°F with humidity above 70%) that patients become symptomatic. Patients who reside in older buildings without air conditioning or adequate ventilation are also at a higher risk. Another factor that may predispose a patient to heat stroke is chronic alcohol abuse. The mechanism is unclear, but over 35% of all patients treated for heat stroke are alcoholics. Medication plays an important part in this; patients who are currently taking tricyclic antidepressants, phenothiazines, thiazides, and sulfonylureas, i.e., tolbutamide (Orinase) chlorpropamide (Diabinese), are at increased risk. Any patient with preexisting ASHD or long-standing debilitating disease process automatically becomes a prime candidate; this risk also increases if the patient is nutritionally deficient. Many of these patients will present with two to five days' complaint of headache, nausea, dizziness and loss of appetite. Since most of these symptoms will be ignored by the patient, he will usually be in the latter stages of heat stroke syndrome when he presents. The patient in latter stage heat stroke will most likely be in coma, with all the resultant management problems that a coma patient presents.

The best indication for heat stroke is body temperature. Body temperature is most accurately recorded by rectal temperature. It is imperative that the paramedic take accurate continuous rectal temperatures throughout his contact with the patient. A rectal temper-

ature of 106°F (41°C) is to be expected in patients with heat stroke. The other sign that is most usually associated with heat stroke is the absence of sweating (red, dry skin). The direct reason for the absence of sweating is unclear, but it is felt by many that excessive exposure to heat of an elderly, debilitated patient over a number of days causes "sweat gland fatigue," thereby reducing the patient's ability to produce sweat with the result being a rapid uncontrolled rise in body temperature. These patients, due to increased cerebral temperature, will usually have neurological irritability; therefore, seizures are to be expected, as well as other neurological manifestations. Other vital signs will vary from patient to patient depending on the time frame. The most important diagnostic signs to look for are hot, dry, red skin and a rectal temperature of 106°F (41°C) or greater.

Treatment. *Heat Cramps.* All treatment for heat-related disorders should be vectored toward removing the offending agent—heat—so removal to a cool, shaded area is the first priority. Assessment of vital signs is the second priority. Rarely will patients with heat cramps suffer vital sign derangement. Ingestion of p.o. fluids with NaCl added may be helpful in restoring both lost volume and salt. Since the job of the paramedic is to evaluate in the prehospital setting, not make final determinations, the patient should be encouraged to seek further medical attention to rule out the possibility of underlying pathology.

Heat Exhaustion. Treatment for heat exhaustion is basically the same as for heat cramps: removal to a cool, shaded area, and removal of all nonessential clothing and assessment of vital signs. Treatment with p.o. fluids containing NaCl is in order. As mentioned previously, a sign of heat exhaustion is peripheral vascular collapse so the patient may, in fact, be hypotensive. Treatment for this is the shock position. If the patient is severely hypotensive, or due to a depressed mental state is unable to ingest p.o. fluids, an IV of 0.9% NaCl 500 cc should be considered after consultation with the base station physician. Cool, moist soaks may be of value to the patient. It is important for any patient in the prehospital phase of emergency care to have constant monitoring of all vital signs, including accurate cataloguing of temperature and mental state for in-hospital evaluation. A final note on heat exhaustion and heat cramps: many systems use enteric-coated salt tablets in both the prehospital and in-

hospital phases. Recent evidence has demonstrated these to be of questionable value due to the fact that they pass through the gastrointestinal tract basically undissolved. It is interesting to note that both these processes could be prevented by increasing salt intake and fluid intake during high-risk periods. An effective public education campaign could significantly reduce morbidity.

Heat Stroke. When the diagnosis of heat stroke has been established, the treatment must be aggressive and complete—complete in the sense that heat stimulates changes in every body system; therefore, every system must be carefully evaluated. The first priority is, as in other heat emergencies, to remove the offending agent. All windows should be opened, any available fans turned on, and the patient placed on the floor due to the fact that it is 2°F cooler on the floor. The patient's clothing should be removed and vigorous attempts at cooling the patient should be started. The process most frequently used consists of wrapping the patient in bed sheets soaked with cold water and placing all available ice inside the sheets. Immersing the patient in the bathtub with cold water may also be employed, as well as rubbing the patient with alcohol. Patient cooling must be as rapid as possible. Keep in mind that because there is already a preexisting problem with the body's temperature-regulating mechanism, *the patient must not be cooled any lower than 102°F* (38.8° C). If the patient's temperature is lowered below 102°F, a real possibility exists of his body temperature dropping quickly to below 95°F (35°C). At this point, I am sure it can clearly be seen why the need exists for constant monitoring of all vital signs, but in particular, rectal temperature.

During the cooling process, particular attention must be paid to the patient's respiratory integrity. Since heat stimulates catabolic metabolism, the patient's oxygen demands are greatly increased. If no history of chronic obstructive pulmonary disease (COPD) is elicited, high-concentration oxygen is in order. Should there be a history of COPD, then low-concentration oxygen therapy should be initiated, via venti mask. If the patient appears in imminent danger of respiratory arrest, then endotracheal intubation should be performed.

All heat stroke patients should be placed on a cardiac monitor. An IV with D_5W should be initiated, at a TKO rate. Since coma can occur as a result of many conditions, the patient should receive 25 g 50% dextrose intravenously after an SMA-6 has been drawn. Naloxone, 0.8 mg intravenously, should also be administered. Many hyperpyrexic patients will experience grand mal seizures. These may be treated in the conventional manner with diazepam, diphenylhydantoin, or phenobarbital. All of these maneuvers should be carried out while focusing on spending as little time at the scene as possible. If any of these maneuvers can be carried out during transport, then all the better. The base station should be contacted to discuss with the physician any other maneuvers or procedures he may find appropriate.

Summary. It is of major importance for the reader to understand that each of the aforementioned heat emergencies was presented in a separate and isolated fashion. These may be occurring simultaneously. Not all patients will present with vital signs within the limits given; patients may present differently under different circumstances. It is with this in mind that I sincerely hope all emergency medicine clinicians who read this will be stimulated to go back and review what they have learned about this subject and to read and explore further. With this attitude, not only will you profit, but more importantly, your patients will benefit. •