Severe Hyperthermia in a 65-year-old Man Following Sustained Environmental Heat Exposure

On a day when the outdoor temperature was 38.3°C (101°F), a 65-year-old man was brought by paramedics to the emergency department. Neighbors placed a 911 call when they found the unkempt and malnourished gentleman lying on the floor of his trash-filled residence. There was no fan or air conditioner in the home. No one knew when the man had last been seen. Paramedics reported the apartment temperature was approximately 46°C (115°F). Daytime temperatures in the area had not been below 32.2°C (89.9°F) for more than a week.

At the scene, the patient was mildly hypotensive, tachycardic, and tachypneic with a tympanic membrane temperature of 41.7°C (107°F). Paramedics placed 2 large-bore intravenous catheters, a nasal trumpet, and an oxygen nonrebreather mask. Upon arrival at the emergency department, the patient had sonorous respirations and dried vomitus around his mouth. He was completely unresponsive to all stimuli. Initial vital signs in the emergency department were as follows: blood pressure, 135/98; heart rate, 145 per minute (narrow complex rhythm); and respiratory rate, 24 per minute. Rectal temperature was 42.2°C (108°F).

Within minutes of arrival, the patient was intubated for airway protection and ventilatory management. A thermistor catheter was inserted to provide continuous bladder temperature readings. To rehydrate the patient and reduce body temperature, 3 L of normal saline solution were rapidly infused.

Active cooling measures were instituted immediately. The patient was sprayed with cool water, and air was blown on him with an electric fan. Ice bags were placed
over the axillae, groin, and back of the neck. The patient’s stomach was lavaged through an orogastric tube using a catheter tip syringe to alternately instill and drain 60 mL aliquots of chilled water. Bladder irrigation was discussed but decided against in favor of continuous temperature monitoring. A 12-lead EKG showed ST segment elevation in V2 and V3, but the patient’s CK-MB level was within normal limits. Toxicologic studies were negative for salicylates and acetaminophen, and there were no abnormalities noted on the patient’s computed tomographic brain scan.

Cooling measures continued until body temperature dropped to 38.3°C (101°F). Two and one half hours after arrival, the patient was transferred to the ICU with a bladder temperature of 37.2°C (98.9°F). During the next several days, the patient’s temperature fluctuated between 37°C and 39.7°C (98°F to 103.5°F). Throughout the course of his hospitalization, acute renal failure, heart failure, and pneumonia developed. He gradually improved and eventually was discharged to a skilled nursing facility.

Six months later he continued to exhibit signs of cerebellar dysfunction including speech difficulties, an ataxic gait, and impaired fine motor coordination.

Discussion
Heat exhaustion and heat stroke, caused by exposure to extreme environmental temperatures, are serious and potentially fatal conditions. The Centers for Disease Control and Prevention reported that between 1979 and 2002, excessive heat exposure was responsible for 8966 deaths in the United States. Heat waves—sustained periods of greater than 32°C (90°F) in a temperate climate—can trigger widespread deaths from hyperthermia. Factors contributing to heat-related illness include prolonged environmental exposure, age extremes, obesity, pre-existing cardiac or vascular disease, mental illness, and the use of alcohol or other drugs.

Heat stroke is defined as a core body temperature higher than 40°C (104°F) associated with signs of organ dysfunction. Hyperthermia causes body system failure by directly injuring the cells, producing circulatory collapse, and initiating a generalized inflammatory response. This patient exhibited neurologic abnormalities, acid-base disturbances, acute renal failure, mild hypotension, and cardiac irritability. Other common heat illness findings include dysrhythmias, coagulopathies, rhabdomyolysis, and multisystem organ failure.

There are many etiologies of elevated body temperature, including various drugs and toxins, endocrine and metabolic disorders, infectious diseases, neurologic conditions, and exposure to environmental heat sources. Although this patient clearly was hyperthermic because of environmental factors, an elevated white blood cell count (17.6/mm³; reference 4.0-11.0/mm³) implied a possible infectious process as well. This gentleman eventually was diagnosed with pneumonia. Interestingly, there appears to be a strong association between heat stroke and infection. In a series of 58 heat stroke cases, Dematte et al. identified a total of 40 infected sites in 33 of their patients.

The early identification and treatment of persons with severely elevated body temperatures dramatically improves prognosis. A core temperature should be obtained as soon as possible. Neither oral nor axillary measures are a reliable reflection of core temperature. Tympanic membrane thermometers also are not recommended; studies have shown that their accuracy can be influenced by many variables. Once hyperthermia has been confirmed, continuous temperature monitoring should be initiated using a bladder, rectal, or esophageal probe. Alternatively, a pulmonary artery catheter can be placed.

Patient management is aimed at decreasing body temperature as quickly as possible without causing hypothermia. Body heat is lost by means of evaporation, radiation, convection, and conduction. Various cooling procedures take advantage of each of these modes of heat loss. External cooling methods include cool water spraying with fanning, ice pack application, cold water immersion, and the use of cooling blankets. This patient was sprayed with cool water and an electric fan was applied. This “spritz and fan” technique quickly reduces body temperature because it simultaneously facilitates evaporative, conductive, and convective heat loss. Studies have shown that the spritz and fan process achieves a remarkable cooling rate of 0.07°C to 0.14°C (0.13°F-0.25°F) per minute.

This patient had ice packs applied over major vessels to promote conductive heat loss. Although this common
practice can serve as an adjunct to care, research indicates it provides inadequate results when used as a primary means of patient cooling. Additionally, conductive cooling measures (ice packs, cooling blankets, and immersion) all lower skin temperature and cause cutaneous vasoconstriction. This leads to shivering, which produces a rise in the core temperature.

Internal cooling measures also rely on conductive heat loss. Methods include iced lavage of the stomach, peritoneum, or bladder. The newest mode of cooling involves placement of specialized intravascular catheters designed to rapidly and directly reduce blood temperature. Iced gastric lavage was used to cool this patient. This minimally invasive, inexpensive, and technically simple procedure can be instituted rapidly. Iced gastric lavage has been reported to reduce body temperature by 0.15°C (0.27°F) per hour, but evidence of efficacy is limited. Knowing when to stop cooling is crucial in order to prevent hypothermia. There are no data that define an exact temperature at which active cooling should cease. However, a rectal temperature of 37.8°C to 39.4°C (100.0°F-102.9°F) has been used as a therapeutic endpoint in multiple studies.

The ABCs of care are important components of hypothermia management. Like this patient, persons with heat stroke have both an impaired level of consciousness and huge metabolic demands. Therefore, airway and breathing are best controlled with intubation and mechanical ventilation. Using a nonheated ventilator circuit will aid cooling. Circulation should be supported with aggressive fluid resuscitation. Persons with heat stroke may lose up to 2 L of fluid per hour through sweating. This patient arrived severely volume depleted. He received 3 L of isotonic fluids in the emergency department and additional volume in the critical care unit. The goal of fluid therapy is to replace half of the water deficit over the first 3 to 6 hours. Complications of hyperthermia may be evident in the emergency department or may develop later. Although this patient’s initial laboratory studies were remarkable only for hypokalemia and elevated lactate, creatine kinase, white blood cell, and creatinine levels, he experienced significant damage to his brain and kidneys. Neurologic injury is common following heat-related insults. In fact, a follow-up study of survivors of the 1995 heat wave in Chicago, Ill., noted ongoing neurologic compromise in 76% of patients.

Gradual rehydration, normalization of serum electrolytes, rapid cooling, and supportive care are currently the most effective means of limiting further organ damage.

REFERENCES