An Unresponsive Woman Found Down

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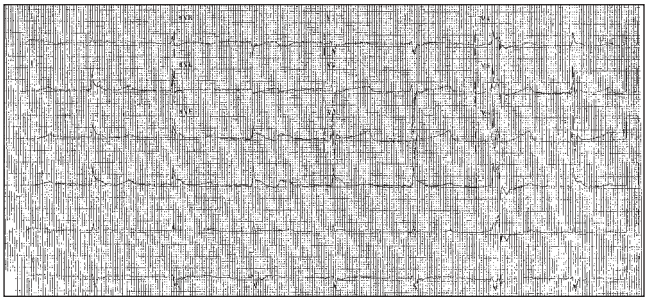
A middle-aged unidentified female presented by fire rescue to the emergency department after being found unconscious in the snow. Her identity, familiar contacts, and medical history were not known, but the patient was presumed to be homeless.

On examination, the patient was found to have a core body temperature of 75 F, an accucheck of 75, a palpable systolic blood pressure of 60, heart rate of 39, and shallow but spontaneous respirations. Pulse oximetry was not obtainable secondary to her cold peripheral extremities. In general, she was a thin disheveled female dressed in multiple layers of clothes with damp boots, and unresponsive to voice and tactile stimulus. The patient had a dry oropharynx, anicteric sclera, but equal and reactive pupils bilaterally. There was no evidence of facial droop, jugular venous distension, or thyromegaly. Her heart rate was slow but regular and there were no audible murmurs. Lung exam revealed mostly clear breath sounds, although decreased at the bases. Abdomen was nontender and soft, with mild to moderate distention and no organomegaly. Extremities were cold, with severe sloughing of the skin over both feet and ankles (trench foot and wet gangrene). Dorsalis pedis pulses were intact by Dopplers. Neurological exam revealed symmetric reflexes throughout with downgoing plantar reflexes. She was immediately intubated for airway protection and started on warmed intravenous fluids, with warming blankets and warmed oxygen. She was taken to the CCU for further monitoring and care.

Laboratory data revealed a WBC 6,000/ml3, hemoglobin 9.5 grams/dL, and 154,000 platelets. Coagulation profile was abnormal with a PTT of 60, PT of 27.7, and INR of 2.42. Urinalysis was significant for 4+ blood and 20 RBCs. A post-intubation arterial blood gas revealed a serum pH of 7.45, pCO2 of 34, PO2 of 150 and 100 % oxygen saturation. BUN was elevated at 36 with a normal creatinine, and both blood alcohol and urine drug screens were negative. Amylase, lipase, and chemistries were normal. Chest x-ray revealed a normal cardiomediastinal silhouette and no consolidation. Head CT on admission showed no acute intracranial abnormalities. Echocardiogram showed normal right and left ventricular function, no pericardial effusion and trace mitral regurgitation, tricuspid regurgitation and mild



pulmonary regurgitation. ECG showed sinus bradycardia with a heart rate of 44, borderline intraventricular conduction delay, prolonged QT interval, and Osborne waves (J waves).

The patient was later identified as a 55 year-old homeless female with past medical history significant for schizophrenia, HIV, alcohol and tobacco abuse. The patient was treated for severe hypothermia compounded by MSSA bacteremia, sepsis, frostbite and wet gangrene of the lower extremities, eventually requiring bilateral below the knee amputations.

Brief Discussion

Approximately 700 people die in the United States each year as a result of hypothermia. Risk factors include homelessness, mental illness, older age, and alcohol and drug addiction. Hypothermia is a core body temperature less than 35 C (95 F). It can be additionally categorized into mild hypothermia (32 C to 35 C or 90 F to 95 F), moderate hypothermia (28 C to 32 C or 82 F to 90 F), and severe hypothermia (below 28 C or 82 F). At these temperatures, the systems responsible for thermoregulation begin to fail. Temperature regulation includes the balance between heat production and loss. Heat is generated through various cellular mechanism and is lost through evaporation, radiation, conduction (transfer of heat to another object) or convection (transfer of heat to air currents). Temperature is regulated in the nuclei of the preoptic anterior hypothalamus. Activation of thermostats in the nuclei and peripheral cold receptors initiate compensatory mechanisms, which eventually leads to progressive depression of metabolism in each organ system. In cold temperatures, the hypothalamus stimulates heat production through shivering and

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increases catecholamine, thyroid and adrenal activity. Other compensatory mechanisms include vasoconstriction, which reduces heat loss by decreasing flow to peripheral tissues. As metabolism in each organ system slows, complications such as cardiac arrhythmias, confusion, lethargy, hypoventilation, pulmonary edema, muscle rigidity, metabolic derangements, and bleeding diatheses, including DIC, can ensue.

Cold exposure is the most obvious cause of hypothermia, but there are also many other conditions that can exacerbate or precipitate it. Altered pituitary/adrenal/thyroid axes can predispose, along with hypoglycemia, sepsis, pancreatitis, uremia, and neurological diseases. Illicit drugs or iatrogenic medications can also alter body temperature fairly rapidly.

Medical approach for hypothermic patients primarily focuses on airway protection, volume resuscitation, careful monitoring of core body temperature, rewarming techniques, and management of complications. Endotracheal intubation is important in these patients for airway protection, particularly in those with altered mental status or a diminished cough reflex. Many develop bronchorrhea with copious secretions, progressing to frank pulmonary edema in some patients. Volume resuscitation and pressor support are also important elements in the management of hypothermic patients. As core temperature rises, peripheral vasodilatation occurs, leading to profound hypotension. Aggressive intravenous hydration with warmed saline is crucial.

Hypotension can contribute to cardiac arrhythmias, which can be refractory to conventional therapies. Specifically, the bradyarrhythmias are typically unresponsive to atropine and pacing is not indicated, unless they persist after adequate rewarming. Classic ECG findings show a sinus bradycardia with elevation of the J-point (J or Osborne wave), representing distortion of membrane repolarization. Ventricular arrhythmias occur when core temperature is less than 82 to 90 F (28 to 32 C). At these temperatures, the myocardium becomes more susceptible to hypoxia, movement, and hypovolemia. Caution must be taken not to jostle the patient, as sudden movement can predispose them to instantaneous arrhythmias. Often, ventricular arrhythmias are treatment-refractory until core body temperature has returned to normal. Standard ACLS protocol is quickly initiated (defibrillation and appropriate pharmacological agents); if initially unsuccessful, rewarming techniques with CPR must be instituted first, and after core body temperature has reached 86 F to 90 F (30 C to 32 C), defibrillation should be reattempted. In some studies, ventricular arrhythmias in animal models have shown a response to bretylium, which has even been used prophylactically at some institutions. Life-supporting measures are continued, unless the body is completely frozen and chest compressions cannot be performed adequately, or there is ice in the airways. There have been case reports describing resuscitation efforts lasting several hours, until the patient has been sufficiently rewarmed.

Monitoring core body temperature is a crucial element in the management of hypothermic patients. The goal is to increase core body temperature 1 to 2 degrees per hour, and temperature should be monitored at more than one site. Standard thermometers can give readings as low as 93 F, and in these situations cannot reliably be used. It should also be noted that rectal and bladder probes have temperature readings that tend to lag behind true core body temperature, while esophageal temperature probes can be falsely elevated due to inhalation of warmed air. Rewarming techniques include intrinsic heat production (shivering), removal of wet clothing, and the use of heating blankets and pads/insulation. Active internal warming techniques, including pleural, peritoneal, and bladder irrigation with warm saline, continuous veno-veno or arterio-veno hemodialysis, and cardiopulmonary bypass with warmed oxygen are more invasive options. Pleural and peritoneal lavage is recommended only in patients with a normal cardiac rhythm and a stable blood pressure. During the rewarming process, a unique phenomenon known as afterdrops can occur. Afterdrop occurs when the extremities and trunk are warmed simultaneously and cold acidotic blood from the periphery returns to core circulation, causing acute temperature drops.

Complications of hypothermia are of particular concern. Skin damage from both cold exposure (i.e. frostbite and trenchfoot) or from heating pads are common. Care must

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be taken not to rub or massage the skin and induce further friction-related injuries. Compartment syndrome and gangrene are frequently seen, which can lead to limb or digit amputation. Metabolic derangements such as rhabdomylosis, acidosis, hyperglycemia, hypoglycemia, and adrenal suppression can also occur. In severe progression, renal failure, DIC, pancreatitis, sepsis and shock liver can lead to death. Initial work up should always consist of chest x-rays, ECG and complete laboratory studies including TSH, cortisol, electrolytes, CBC, LFTs, pancreatic enzymes, serial arterial blood gases, and coagulation studies. Head CT, urine drug screen and blood alcohol level in the unresponsive patient can help rule out other etiologies of syncope. Empiric antibiotics, thiamine, dextrose, and naloxone for patients with altered mental status should also be considered.

In summary, the management of the hypothermic patients focuses on combined resuscitative and rewarming techniques. Resuscitation efforts should be continued until the patient is adequately rewarmed, as arrhythmias can be refractory to standard therapies until the core body temperature is reached. The low temperatures do have a protective effect on neurological function, allowing for patients to recover after a prolonged arrest. Complications can still occur long after rewarming, and careful monitoring of the patient in a critical care setting is essential.

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