Severe Hypothermia Associated With Prolonged Cardiorespiratory Arrest And Full Recovery

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Recovery from prolonged cardiopulmonary resuscitation after cardiac arrest during a state of accidental hypothermia has been documented many times, but currently there is debate about how to treat the condition. Some physicians advocate aggressive, rapid core rewarming by open thoracotomy and cardiac bypass. Other physicians have had success with conservative methods of core rewarming. This case illustrates success with the latter approach and reminds physicians that hypothermic patients in full cardiopulmonary arrest are protected by their low metabolic rate and can survive intact after long periods of cardiopulmonary resuscitation and slow rewarming.

Case Report
A 38-year-old man was brought to the emergency department of a Level II 230-bed community hospital in Philadelphia on Christmas Eve by paramedics. Ambient temperatures that day were recorded as a low of 5°C (41°F) and a high of 18°C (64°F). The patient had been found lying in wet clothes, awake but confused, in an unheated apartment. He had no medical problems other than a history of alcohol dependency with withdrawal seizures. He had the odor of alcohol on his breath. As he was being undressed, he went into cardiopulmonary arrest. Cardiopulmonary resuscitation was begun, and he was ventilated by means of an endotracheal tube. A cardiac monitor showed ventricular fibrillation. A rectal temperature of 23°C (73°F) was recorded with a thermistor probe.

The patient had received dextrose and naloxone when he was found. In the emergency department he received epinephrine, atropine, lidocaine, sodium bicarbonate, and thiamine. Attempts at electrocardioversion with 360 J were unsuccessful. Using a subclavian route, the patient was given a 300-mL bolus of normal saline that had been warmed in the microwave until the temperature of the bag was near neutral to touch. After the fluid warmer was set up, nasogastric lavage was begun with saline warmed to 40°C (104°F), and oxygen was delivered by warm mist at 40°C. A venous blood gas reading provided the following findings: pH 7.19, pCO₂ 59 mmHg, and pO₂ 31 mmHg, uncorrected for temperature. The patient remained in ventricular fibrillation as confirmed by different leads, so external chest compressions at a rate of 50 per minute and adjunct ventilation with warmed 100 percent oxygen were continued. Repeat attempts at cardioversion were made. One hour and 22 minutes after arrest, the patient was electrically converted to sinus rhythm at a rate of 53 beats per minute. His pulse became palpable, and his systolic pressure was 86 mmHg. His rectal temperature at this time was 24°C (75°F). The patient’s temperature rose at a rate of 1.3°C per hour to 36.7°C (97°F).

The patient’s alcohol level on admission was 192 mg/dL; his leukocyte count was 4,000/mm³; hemoglobin, 15 mg/dL; platelet count, 6 x 10⁴/mm³; and magnesium level, 1.2 mg/dL. He was given magnesium sulfate intravenously. An initial electrocardiogram done within 1 hour after the patient converted to sinus rhythm showed elevated ST segments in anterior leads and Osborne waves, but these abnormalities resolved within 24 hours. The following day, the patient was tremulous and experienced a grand mal seizure and fever. A chest radiograph was unremarkable, and his blood, sputum, and urine cultures grew no organisms. He was treated with diazepam, phenytoin, and cefazolin. The patient’s leukocyte count rose to 10,000/mm³, hemoglobin dropped with fluid replacement to 10.6 mg/dL, and platelet count was 31,000/mm³. The platelet count gradually rose during the next few days. Prothrombin time was normal. There was no evidence of bleeding. Blood glucose dropped from 190 to 44 mg/dL. The total creatine kinase rose from 2857 U/L on the day of admission to 47,006 U/L (1 percent MB fraction only) 2 days later. His peak aspartate

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aminotransferase was 1454 U/L and lactate dehydrogenase 1883 U/L. The patient did not develop either acute tubular necrosis or pulmonary edema, two frequent sequelae of severe hypothermia. He did have frostbite of his toes, which resolved without any surgery or hyperbaric treatment. The patient was in the hospital for 10 days. His neurologic abnormalities and mental status alterations resolved completely within 1 week.

Discussion
Hypothermia occurs with exposure if the hypothalamic center of thermoregulation is damaged or if the body cannot retain heat by vasoconstriction or cannot generate warmth by shivering. Various disease states thus can contribute to the development of hypothermia (Table 1). The most common associated condition is alcoholism. Mortality depends primarily on the presence or absence of underlying disease. No relation has been established between survival and temperature at admission, age, or rewarming rate.  

Under controlled conditions, patients have survived with no sequelae after experiencing body temperature as low as 9°C (48°F). In accidental hypothermia patients have recovered fully after having temperatures as low as 18°C (64°F).  

Severe hypothermia has been defined as a temperature of less than 30°C (86°F). At this level there are usually no purposeful movements, no reflexes, and pupils are dilated and unreactive. Dysrhythmias can be precipitated and cardiac arrest can occur, so patients should be handled gently, with as little movement as possible. Procedures such as intubation and central line placements should not be withheld, however, when indicated.

Hypothermic patients are fluid depleted as a result of vasoconstriction and diuresis, secondary to a cold-induced concentrating defect in the renal tubule. It is important, therefore, to give the patients normal saline at a rate of 100–200 mL/h, especially because acute tubular necrosis is a concern. Rehydration of the hypothermic patient can be compicated by noncardiac pulmonary edema, the cause of which is unknown. This condition needs to be distinguished from aspiration pneumonia, which can also occur as a result of a diminished gag reflex.

Ileus and gastrointestinal bleeding caused by ischemic effects of vasoconstriction have been seen in hypothermic patients. Platelet counts are frequently low because of sequestration of platelets in the spleen and liver, but the count rises quickly after rewarming.

Bradycardia is a physiologic response to hypothermia and does not warrant treatment unless it persists after normal temperature is restored. Osborne waves, which are J-point elevations, are commonly seen on the electrocardiogram. Once thought to be pathognomonic for hypothermia, they have been reported in normal states, as well as with central nervous system disease. Elevations in lactate dehydrogenase and creatine kinase are frequently seen after hypothermic insult. It is postulated that the cold alters cell membrane integrity allowing the enzymes to leak out. In our patient, rhabdomyolysis and tissue necrosis from prolonged cardiopulmonary resuscitation and frostbite probably contributed to the hugely elevated creatine kinase. Most of the creatine kinase was noncardiac in origin. The severely hypothermic heart is generally unresponsive to drugs and defibrillation, and drug metabolism is reduced. Thus, it is important to be conservative with the use of medications and defibrillation attempts and to focus efforts on core rewarming.

Table 1. Factors Associated with Hypothermia.

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<tr>
<td>Central nervous system disease</td>
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<td>Hypoglycemia</td>
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<td>Thiamine deficiency and malnutrition</td>
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<td>Uremia</td>
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<td>Hepatic failure</td>
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<td>Adrenal insufficieny</td>
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Hypothermia and Cardiorespiratory Arrest
The treatment of severe hypothermia has been divided into external rewarming and core rewarming methods. Active external rewarming has a higher mortality rate,13 because of hypotension that results from peripheral vasodilatation and possibly an after-drop of core temperature when cold, acidic peripheral blood recirculates.18 Conservative methods of core rewarming include inhaled oxygen, intravenous fluids, and gastric and colonic lavage fluids all warmed to approximately 44°C (111°F).10 More aggressive methods of core rewarming include peritoneal, pleural, and mediastinal lavage, as well as hemodialysis and cardiopulmonary bypass.2-4 There is currently debate about how aggressively the body should be rewarmed.1,5

Hypothermia protects tissues by lowering the metabolic rate,19 so highly invasive, quick rewarming might not be necessary. Rewarming at a rate of 1°C/h has been considered optimal.20 Cardiopulmonary resuscitation has been carried out for up to 4 hours without neurologic sequelae.21 Unless cardiac function does not return within a few hours or body temperature fails to rise at least 1°C/h, conservative core rewarming procedures, with continuous cardiopulmonary resuscitation, might be appropriate. These measures were adequate for our patient, who recovered neurologically intact after 1 hour and 22 minutes of ventricular fibrillation. It will require many more case reports of severely hypothermic patients in cardiopulmonary arrest to establish how long conservative treatment should continue before highly invasive techniques are started.

Summary
A patient with severe hypothermia was in ventricular fibrillation for more than 1 hour. Conservative core rewarming methods were used with complete success. In dealing with severely hypothermic patients, it is important to continue treatment despite prolonged cardiopulmonary arrest. If conservative core rewarming methods are not successful, then aggressive invasive techniques might be effective.

References
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