A Case of Hypothermia

LAURA RUHLAND, MD; JONATHAN AMELI, MD; WILLIAM BINDER, MD

From the Case Records of the Alpert Medical School of Brown University Residency in Emergency Medicine

DR. LAURA RUHLAND: The patient is a 48-year-old woman who was brought to the hospital by EMS this past February. By report, the patient had expressed suicidal thoughts to her family and then went missing at noon today. Police were notified at around 3 pm, and search and rescue became involved shortly thereafter. The patient was found with the lower half of her body submerged in brackish water and with an empty bottle of vodka cradled next to her. Per EMS, the patient had recently been diagnosed with leukemia and was despondent. At the scene, the patient was noted to be cold and obtunded. Access was difficult to obtain and an intraosseous catheter was placed in the left proximal tibia and the patient received .9 normal saline (NS) fluid. She was given 2 mg of naloxone with no effect and was brought to the emergency department.

Upon arrival, vital signs revealed her blood pressure to be 84/52 mm Hg, pulse of 72, irregular rhythm and oxygen saturation of 92% on 100% oxygen face mask. A rectal probe revealed a temperature of 83.6 degrees F (28.7 C). The patient was unresponsive to verbal or noxious stimuli, and was moving very little air with minimal chest rise. She had weak pulses in the radial, femoral, and carotid locations. There was no outward evidence of any deformity or trauma to her body. Her pupils were 5 mm and minimally reactive, her heart exam revealed an irregular S1S2 with no murmurs. Her abdomen was soft, and her upper extremities were initially found to be rigid in flexion. Her Glasgow Coma Score (GCS) was 3.

DR. BRUCE BECKER: What were your initial concerns and next steps?

DR. RUHLAND: Our initial concern was for the patient’s airway, as she was comatose. Intravenous access was obtained and simultaneously the patient was placed on a monitor. An ECG revealed atrial fibrillation at a rate in the 70s, without peaked T waves, suggesting that the patient was not significantly hyperkalemic, a complication often noted in hypothermia. One caveat is that hypothermia can obscure classic ECG changes in a range of conditions, including hyperkalemia. (1) The patient was intubated, without incident, using etomidate and succinylcholine, a depolarizing agent that can exacerbate hyperkalemia. (2) Laboratory studies are noted in Table 1.

DR. NATHAN HUDEPOHL: Can you discuss the severity and implications of this patient’s decreased temperature.

DR. RUHLAND: Accidental hypothermia is defined as an involuntary drop in core body temperature to below 35 degrees C (95 F). (3) Up to 1500 patients in the United States are estimated to succumb annually to hypothermia, with about 50% of these noted to be accidental, but the true incidence and morbidity and mortality is unknown. (3, 4) The Swiss hypothermia (HT) classification system provides an estimate of the severity of the patient’s hypothermia in comparison to clinical signs. (Table 2) (3, 5) Previous systems described hypothermia as either mild, moderate, or profound (<28 C). (5, 6) One of the limitations of both the Swiss model, as well as other systems, is that clinical findings may not correspond to classification. (7)

Our patient was unconscious, did not demonstrate any shivering, and had a core temperature hovering near 28 C, and was consequently HT2/HT3. Her altered consciousness may have also been affected by ingestion of alcohol.

Table 1.

<table>
<thead>
<tr>
<th></th>
<th>Initial</th>
<th>Repeat 2 hours later</th>
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<tbody>
<tr>
<td>Venous pH</td>
<td>7.21</td>
<td>7.29</td>
</tr>
<tr>
<td>WBC</td>
<td>7.2</td>
<td>15.0</td>
</tr>
<tr>
<td>Hb</td>
<td>16.3 g/dl</td>
<td>13.6 g/dl</td>
</tr>
<tr>
<td>Potassium</td>
<td>4.9 meq/L</td>
<td>3.8 meq/L</td>
</tr>
<tr>
<td>creatinine</td>
<td>.49 mg/dl</td>
<td>.53 mg/dl</td>
</tr>
<tr>
<td>CPK</td>
<td>634 mg/dl</td>
<td></td>
</tr>
<tr>
<td>troponin</td>
<td>&lt;.006 ng/ml</td>
<td></td>
</tr>
<tr>
<td>alcohol</td>
<td>369 mg/dl</td>
<td></td>
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Table 2.

<table>
<thead>
<tr>
<th>Stage</th>
<th>Clinical Signs</th>
<th>Temperature</th>
</tr>
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<tbody>
<tr>
<td>HT1</td>
<td>Unimpaired consciousness; shivering</td>
<td>35 C to 32 C</td>
</tr>
<tr>
<td>HT2</td>
<td>Altered consciousness; no shivering</td>
<td>&lt;32 C to 28 C</td>
</tr>
<tr>
<td>HT3</td>
<td>Unconscious</td>
<td>&lt;28 C to 24 C</td>
</tr>
<tr>
<td>HT4</td>
<td>No vital signs</td>
<td>&lt; 24 to 13.7 C</td>
</tr>
<tr>
<td>HT5</td>
<td>Death</td>
<td>&lt;13.7 C (may be lower)</td>
</tr>
</tbody>
</table>
**DR. MARK GREVE:** What is the physiologic response to hypothermia?

**DR. WILLIAM BINDER:** The initial response to cooling is peripheral vasoconstriction and increased metabolism due to thermogenesis from shivering, which can increase metabolic heat production 2-5 times above resting levels. [8] Respiratory effort, oxygen consumption, cardiac output, and mean arterial pressure initially increase. However, when core temperature drops to approximately 32°C, metabolism begins to decrease. Bradycardia and diminished myocardial contractility occur, and hypoventilation with concomitant carbon dioxide retention, hypoxia, and respiratory acidosis are noted in hypothermia below 32°C. [8] As temperature drops below 30°C, dysrhythmias are frequently observed. Central nervous system physiology is altered as well, with abnormalities on electroencephalogram (EEG) noted below 34°C, and decreased EEG activity seen below 28°C. [9] Correspondingly, mental status is affected and patients develop irritability, confusion, apathy, lethargy, and proceed to somnolence and coma. (5) Other systems are impacted as well. Coagulopathy, renal dysfunction and cold diuresis occur, and endocrine and immunologic changes are seen in hypothermic patients. [8,9]

**DR. JESSICA SMITH:** What are the usual mechanisms for warming a patient and what steps were taken for your patient?

**DR. RUHLAND:** In mildly hypothermic patients, passive rewarming using a forced air warming blanket can raise the core temperature 1-2 degrees C/hour. [9] Heating pads, warm water bottles, and warmed intravenous fluids in conjunction with a warming blanket can increase the core temperature up to 3°C/hour. [3] Additional modalities used in more severe hypothermia include body cavity lavage. Warm fluids exchanged through a Foley catheter and nasogastric tube can transfer heat and in patients with profound hypothermia, pleural and peritoneal lavage can warm patients up to 3 degrees C/hour. [3] The use of warm, humidified air during mechanical ventilation can contribute to rewarming, as well. In profound hypothermia, extracorporeal assisted rewarming (ECAR) techniques are currently standard of care in trauma centers.

ECAR techniques were first successfully utilized in the 1960s. Data suggests that in patients who have not had a cardiac arrest, ECAR can be used in the setting of arrhythmias, hypotension, respiratory distress and failure, refractory acidosis, core temperatures < 28°C, and failure to respond to non-invasive warming methods. [10] ECAR is less successful in patients with hypoxic cardiac arrest (suffocated avalanche victims, prolonged submerged drowning victims) as neurologic outcomes are poor. [10]

Our patient did not require extra corporeal membrane oxygenation (ECMO). We were able to perform both passive and active rewarming using a forced air warming blanket, a heated room, warm intravenous fluids, warm humidified air in the ventilation circuit, as well as gastric and bladder lavage. The patient’s core temperature rose approximately 1.5–2 degrees C during the first hour of resuscitation, and her temperature increased 2.5 degrees C hourly for the next two hours. At 32°C her risk for arrhythmia was diminished and a CT of her brain was performed and was normal and without hemorrhage.

**DR. BECKER:** This patient was rather fortunate in that she was found early enough and did not suffer profound hypothermia. We recently encountered a patient who was frozen solid and succumbed to hypothermia. How does one determine whether a patient is truly dead from hypothermia?

**DR. JONATHAN AMELI:** Stage 4 hypothermia is noted when the core temperature is below 24°C. Vital signs are frequently absent in such a profound state. However, while some people may be cold and without vital signs, numerous case reports describe prolonged cardiopulmonary resuscitation (CPR) in severely hypothermic patients who make partial and full recoveries with active core rewarming. [10–14]

Standard advanced cardiac life support may not be effective below 30°C. Body rigidity and fixed dilated pupils can occur with hypothermia, and may be reversible. Consequently, these exam findings should not be used to guide resuscitation prior to warming. Core temperature should be > 32°C prior to terminating CPR in patients without vital signs – no one should be pronounced dead until warm and dead. [3] However, patients who suffer cardiac arrest prior to hypothermia or who are frozen solid and have a non-compressible chest, have very poor outcomes and can be considered dead if they are without vital signs. In patient’s with vital signs, a core temperature of > 36°C should be maintained prior to initiating brain death evaluation.

While core temperature is not necessarily predictive of outcome, poor prognostic signs include potassium levels greater than 10 mmol/L in adults, and > 12 mmol/L in children, severe acidosis (pH < 6.5), coagulopathy, as well as severe traumatic injury. [10, 15]

**DR. SETH GEMME:** What was the patient’s outcome?

**DR. RUHLAND:** The patient was transferred to the medical intensive care unit, and she was extubated on hospital day 1. On hospital day 2 she divulged that she had gotten into an argument with her best friend, and decided to drink and hurt herself. She also revealed that she had been diagnosed with chronic myelogenous leukemia, and was being treated with imatinib. She was evaluated by the psychiatry service and agreed to a transfer to an in-patient psychiatric facility on hospital day 3.
References


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