Accidental hypothermia in adults

Authors:
Ken Zafren, MD, FAAEM, FACEP, FAWM
C Crawford Mechem, MD, FACEP

Section Editor:
Daniel F Danzl, MD

Deputy Editor:
Jonathan Grayzel, MD, FAAEM

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INTRODUCTION — Death from exposure and accidental hypothermia occurs throughout the world and can present significant management problems [1-5]. While typically associated with regions of the world with severe winters, hypothermia is also seen in areas with milder climates, such as the southern United States [6]. Cases of hypothermia occur during the summer months and in hospitalized patients [7]. Even with modern supportive care, the in-hospital mortality of patients with moderate or severe accidental hypothermia approaches 40 percent [8].

The definition, pathophysiology, and management of accidental hypothermia will be discussed here. A table outlining the emergency management of hypothermia in adults is provided (table 1). Drowning, which is often complicated by hypothermia, is discussed separately, as are external injuries from cold and accidental hypothermia in children. (See "Drowning (submersion injuries)" and "Frostbite" and "Hypothermia in children: Clinical manifestations and diagnosis" and "Hypothermia in children: Management".)

DEFINITIONS — Hypothermia is defined as a core temperature below 35°C (95°F). The stage of hypothermia, defined by core temperature, has a large impact on both recognition and treatment. The most commonly used definitions found in the literature are as follows [9,10]:

- Mild hypothermia – Core temperature 32 to 35°C (90 to 95°F)
- Moderate hypothermia – Core temperature 28 to 32°C (82 to 90°F)
- Severe hypothermia – Core temperature below 28°C (82°F)

In addition, some experts regard a core temperature <24°C (75°F) or <20°C (68°F) as profound hypothermia [11,12].

Because the clinical features of hypothermia differ among patients, and because core temperature measurement is imprecise, the recognition of each stage is more important than exact boundaries.

Pre-hospital personnel, while carefully measuring core temperatures, may also refer to the clinical staging scheme described by the International Commission for Mountain Emergency Medicine (table 2) [11].
● **Mild (HT I)** – Normal mental status with shivering. Estimated core temperature 32 to 35°C (90 to 95°F)

● **Moderate (HT II)** – Altered mental status without shivering. Estimated core temperature 28 to 32°C (82 to 90°F)

● **Severe (HT III)** – Unconscious. Estimated core temperature 24 to 28°C (75 to 82°F)

● **Severe (HT IV)** – Apparent death. Core temperature 13.7 to 24°C (56.7 to 75°F) (resuscitation may be possible)

● **Death (HT V)** – Death due to irreversible hypothermia. Core temperature <9 to 13.7°C (48.2 to 56.7°F) (resuscitation not possible)

This system (sometimes referred to as "the Swiss system") is intended to help prehospital rescuers estimate the severity of hypothermia by observing clinical signs. However, the response to hypothermia varies widely among patients. A potential limitation to this staging system is that shivering may not cease until around 30°C in some patients. In addition, vital signs and other evidence of life may be present at core temperatures below 24°C [13]. An analysis of published case reports found a high degree of overlap of core temperatures among the stages and many cases in which the Swiss system overestimated core temperature. The lowest observed core temperature in was 28.1°C for HT 1, 22.1°C for HT II, and 19.3°C HT III [14].

**PATHOPHYSIOLOGY** — Body temperature reflects the balance between heat production and heat loss. Heat is generated by cellular metabolism (most prominently in the heart and liver) and lost by the skin and lungs via the following processes [15]:

- **Evaporation** – Vaporization of water through both insensible losses and sweat
- **Radiation** – Emission of infrared electromagnetic energy
- **Conduction** – Direct transfer of heat to an adjacent, cooler object
- **Convection** – Direct transfer of heat to convective currents of air or water

Of these, convective heat loss to cold air and conductive heat loss to water are the most common mechanisms of accidental hypothermia [16].

The normal set point for human core temperature is 37±0.5°C. The human body maintains this temperature whenever possible, using autonomic mechanisms to regulate heat loss and gain in response to environmental conditions. Nevertheless, the human body has limited physiologic capacity to respond to cold environmental conditions. Thus, behavioral adaptations such as clothing and shelter are critical to defend against hypothermia.

The hypothalamus receives input from central and peripheral thermal receptors. In response to a cold stress, the hypothalamus attempts to stimulate heat production through shivering and increased thyroid, catecholamine, and adrenal activity. Sympathetically mediated vasoconstriction minimizes heat loss by reducing blood flow to peripheral tissues, where cooling is greatest [16,17]. Peripheral blood vessels also vasoconstrict in direct response to cold.

Cooling decreases tissue metabolism and inhibits neural activity. During the initial phase of cooling, shivering in response to skin cooling produces heat and increases metabolism,
ventilation, and cardiac output. Neurologic function begins declining even above a core temperature of 35°C. Once the core temperature reaches 32°C, metabolism, ventilation, and cardiac output begin to decline and shivering becomes less effective until it finally ceases as core temperature continues to drop.

CLINICAL PRESENTATION

**General findings and progression** — As the compensatory mechanisms preventing hypothermia are overwhelmed, the following changes typically occur (table 3) [9,18,19]:

- Patients with mild hypothermia demonstrate tachypnea, tachycardia, initial hyperventilation, ataxia, dysarthria, impaired judgment, shivering, and so-called "cold diuresis."
- Moderate hypothermia is characterized by proportionate reductions in pulse rate and cardiac output, hypoventilation, central nervous system depression, hyporeflexia, decreased renal blood flow, and loss of shivering. Paradoxical undressing may be observed. Atrial fibrillation, junctional bradycardia, and other arrhythmias can occur.
- Severe hypothermia can lead to pulmonary edema, oliguria, areflexia, coma, hypotension, bradycardia, ventricular arrhythmias (including ventricular fibrillation), and asystole [16,20].

At patient presentation, beware of vital signs inconsistent with the degree of hypothermia. Such inconsistency suggests an alternative diagnosis. (See 'Differential diagnosis' below.)

The neurologic manifestations of hypothermia vary widely, but the level of consciousness should be consistent with the core temperature. A significant discrepancy suggests an alternative diagnosis. Do not assume that areflexia or paralysis is due to hypothermia until spinal injury has been ruled out.

Risk factors associated with death from accidental hypothermia include ethanol use, homelessness, psychiatric disease, and older age [2,21].

**Geriatric population** — The elderly are at increased risk of developing hypothermia and its complications, and should be urgently assessed if found to be hypothermic [21,22]. The reasons for this increased risk include decreased physiologic reserve, chronic diseases and medications that impair compensatory responses, and social isolation. Hypothermia may go unrecognized in isolated older patients, and they may be unable to obtain assistance when the condition is recognized.

In elderly patients sepsis can manifest as hypothermia. Empiric, broad spectrum antibiotics should be administered if there is unexplained hypothermia, a probable source of infection, aspiration, failure to rewarm, or other signs suggestive of sepsis. (See "Evaluation and management of suspected sepsis and septic shock in adults".)

**ASSESSMENT**

**Physical examination and temperature measurement** — The hypothermic heart is very sensitive to movement. Rough handling of the patient may precipitate arrhythmias, including
ventricular fibrillation. Take care to avoid jostling the patient during the physical examination or the performance of essential procedures.

Effective diagnosis and management of hypothermia depend upon the use of a low-reading thermometer to determine core temperature \([23,24]\). Many standard thermometers read only to a minimum of 34°C (93°F) and are therefore unsuitable.

Use of rectal temperature is reasonable in conscious patients. In patients with severe hypothermia, particularly those requiring endotracheal intubation, an esophageal probe inserted into the lower one-third of the esophagus (about 24 cm below the larynx) provides a near approximation of cardiac temperature \([12]\). Esophageal temperature is the most accurate method to track the progress of rewarming. Rectal probe readings may rise following peritoneal lavage or fall if adjacent to cold feces; esophageal probes not inserted into the lower third of the esophagus may read falsely high if heated humidified oxygen is used. Infrared tympanic thermometers and so-called temporal artery thermometers are not accurate \([12]\).

Bladder temperatures are commonly used and are adequate in mild to moderate hypothermia. However, bladder and rectal temperatures should not be used in critical patients during rewarming. Changes in rectal and bladder temperatures significantly lag behind core temperature changes during rewarming. Core temperature may be increasing in response to rewarming while rectal and bladder temperatures are still dropping.

A total body survey should be conducted in all hypothermic patients to exclude local cold-induced injuries in areas not seen during the initial resuscitation and to assess for signs of trauma. Frostbite and local cold-related injury are discussed in detail elsewhere. (See "Frostbite".)

**Laboratory studies and monitoring** — After making the diagnosis of hypothermia, laboratory evaluation should be undertaken to identify potential complications and comorbidities, including lactic acidosis, rhabdomyolysis, bleeding diathesis, and infection. Previously healthy patients with mild accidental hypothermia may not require laboratory investigation. We obtain the following tests in cases of moderate and severe hypothermia:

- Fingerstick glucose
- Electrocardiogram (ECG)
- Basic serum electrolytes, including potassium and calcium
- BUN and creatinine
- Serum hemoglobin, white blood cell, and platelet counts
- Serum lactate
- Fibrinogen
- Creatine kinase (CK)
- Arterial blood gas, uncorrected for temperature, in ventilated patients
- Chest radiograph (take care to avoid jostling the patient)

Additional studies are obtained based on clinical circumstances. As an example, a toxicologic screen might be obtained in a patient with a depressed mental status that is not
commensurate with the core temperature. A serum cortisol and thyroid function studies might be obtained in a patient with moderate or severe hypothermia who fails to rewarm despite aggressive interventions. (See "Failure to rewarm" below and "General approach to drug poisoning in adults".)

Since the hematocrit increases 2 percent for each 1°C drop in temperature, a low normal hematocrit is abnormal in severe hypothermia [25]. Hyperglycemia that persists during rewarming suggests pancreatitis or diabetic ketoacidosis. Remember that insulin is ineffective below 30°C. Hypothermia obscures normal premonitory ECG changes commonly associated with hyperkalemia.

A list of characteristic laboratory abnormalities is provided (table 4) [16-18,20,26-28]. Rewarming can be accompanied by rapid or unpredictable changes in electrolyte concentrations, so frequent reassessment of these parameters (approximately every four hours) is prudent in moderate and severe hypothermia [15].

Inhibition of the enzymes of the coagulation cascade from hypothermia leads to a bleeding diathesis [27]. Because tests of coagulation (eg, prothrombin time, partial thromboplastin time) are always performed at 37°C, the laboratory will report deceptively "normal" results despite an obvious in vivo coagulopathy. Treatment consists of rewarming. Administration of clotting factors is ineffective.

Oxygenation should be monitored continuously; however, the response time of pulse oximeters placed on the finger is slowed by hypothermia [29]. Probes placed on the ears or forehead appear to be less influenced by decreased body temperature and the associated peripheral vasoconstriction. Assessment of oxygenation is also complicated by the fact that arterial blood gas analyzers operate at 37°C, which is (by definition) greater than the body temperature of a hypothermic patient.

The pH, PCO2, and PO2 of a blood sample all vary with the temperature, as gas tensions and hydrogen ion concentration decline as temperature drops [30,31]. Use uncorrected values to guide therapy, recognizing that these results correspond to the arterial blood gas values that would have been present if the patient's temperature were 37°C [30]. An uncorrected pH of 7.40 and a pCO2 of 40 mmHg reflect acid-base balance. (See "Arterial blood gases", section on 'Transport and analysis'.)

**Electrocardiographic changes** — Independent of the arrhythmogenic complications noted above, hypothermia causes characteristic ECG changes because of slowed impulse conduction through potassium channels. This results in prolongation of all the ECG intervals, including RR, PR, QRS, and QT [32].

There may also be elevation of the J point (only if the ST segment is unaltered), producing a characteristic J, or Osborn, wave that represents distortion of the earliest phase of membrane repolarization (waveform 1) [33]. The height of the J wave is roughly proportional to the degree of hypothermia [34,35]. These findings are most prominent in precordial leads V2 to V5; similar findings can be observed in patients with early repolarization, hypercalcemia, and Brugada syndrome. Although suggestive of hypothermia, J waves are
not pathognomonic and can be found in other conditions (eg, subarachnoid hemorrhage, brain injury).

Shivering can cause a rhythmic irregularity of the ECG baseline, and occasionally the QRS complex (waveform 2).

Available software for ECG interpretation is unable to recognize J waves, and often misinterprets them as currents of injury (ie, ischemic changes). J waves have been mistaken for ST elevations leading to unnecessary cardiac catheterization [36].

(See "Electrocardiogram in the diagnosis of myocardial ischemia and infarction", section on 'Early repolarization'.)

DIFFERENTIAL DIAGNOSIS — In addition to hypothermia from environmental exposure, many medical conditions can cause hypothermia, including hypothyroidism, adrenal insufficiency, sepsis, neuromuscular disease, malnutrition, thiamine deficiency, and hypoglycemia. Ethanol abuse and carbon monoxide intoxication have been implicated in some cases of hypothermia [37]. Hypothermia has even been misdiagnosed as psychiatric illness, including attempted suicide. The differential diagnosis of hypothermia is summarized in the following table (table 5) [16,18,23,26,38-40].

Certain medications directly or indirectly cause hypothermia, either by impairing thermoregulatory mechanisms, decreasing awareness of cold, or clouding judgment. The most common medications that impair thermoregulation are anxiolytics, antidepressants, antipsychotics, and opioids. Medications that can impair a patient's ability to compensate for a low ambient temperature include oral antihyperglycemics, beta-blockers, alpha-adrenergic agonists (eg, clonidine), and general anesthetic agents [41].

At patient presentation, beware of vital signs inconsistent with the degree of hypothermia. A relative tachycardia inconsistent with the core temperature suggests hypoglycemia, hypovolemia, or an overdose. Relative hyperventilation implies an underlying organic acidosis (eg, diabetic ketoacidosis, aspirin overdose), since CO2 production should be decreased in moderate or severe hypothermia.

The neurologic manifestations of hypothermia vary widely, but the level of consciousness should be consistent with the core temperature. If the level of consciousness is not proportional to the degree of hypothermia, suspect a head injury, central nervous system infection, or overdose.

Although the myriad secondary causes of hypothermia listed above may occur simultaneously with accidental hypothermia (eg, excessive alcohol use leading to prolonged environmental exposure), the latter can usually be distinguished on the basis of a history of exposure to cold without other complicating factors.

DIAGNOSIS — The diagnosis of accidental hypothermia is made based upon a history or other evidence of environmental exposure to cold and a core temperature below 35ºC (95ºF). Proper diagnosis depends upon the use of a low-reading thermometer to determine the core temperature accurately. (See 'Assessment' above.)
MANAGEMENT

Airway, breathing, circulation — The management of hypothermia requires evaluation and support of the airway, breathing, and circulation; prevention of further heat loss; initiation of rewarming appropriate to the degree of hypothermia; and treatment of complications [26,42,43]. A table outlining the emergency management of hypothermia in adults is provided (table 1). (See "Advanced cardiac life support (ACLS) in adults" and "Rapid sequence intubation for adults outside the operating room".)

Endotracheal intubation is performed in patients with respiratory distress or who cannot protect their airway. Early intubation can facilitate clearance of secretions produced by cold-induced bronchorrhea in patients with altered mental status or a decreased cough reflex. Standard medications can be used to perform rapid sequence intubation, if necessary; atropine is not indicated as a pretreatment agent to reduce bronchorrhea.

In the field and in the emergency department, misdiagnosis of a non-perfusing rhythm, even with a cardiac monitor, is a hazard. Peripheral pulses can be difficult to palpate in a vasoconstricted bradycardic patient. It is best to check for a central pulse for up to a full minute, using a continuous wave Doppler, if available. Alternatively, a focused bedside echocardiogram can be performed.

Cardiopulmonary resuscitation (CPR), including chest compressions, should be initiated in patients with accidental hypothermia who sustain a cardiac arrest. Contraindications to chest compressions include: verified DNR ("do not resuscitate") status, obviously lethal injuries, a frozen chest wall that is not compressible, and the presence of any signs of life. Always assume that some perfusion is occurring when any sign of life is present. Although evidence is scant, we believe that chest compressions should not be performed in patients who manifest an organized rhythm on a cardiac monitor even if they have no palpable pulses and no other signs of life. Our reasoning is that such rhythms may reflect successful perfusion that could be disrupted by chest compressions, and that any pulseless electrical activity (PEA) is likely to be transient. There is little downside to withholding CPR briefly in these hypothermic patients. Should PEA become asystole, chest compressions should be started immediately. Chest compressions should also not be started if cardiac contractions are seen on bedside echocardiogram or pulses can be detected with Doppler ultrasound.

If pulses are not found after checking for one minute, CPR should be started immediately and provided continuously if possible. Continuous CPR may not be possible due to safety considerations or during evacuation of the patient. Mechanical chest-compression devices should be used, if available, to avoid interruption of CPR during transport. CPR can be delayed up to 10 minutes while rescuers move the patient to a safer location. In a patient with core temperature of 20 to 28°C, or if the core temperature is not known, administer CPR continuously for periods of at least 5 minutes, alternating with periods of no longer than 5 minutes without CPR. In patients with a core temperature below 20°C, administer CPR continuously for periods of at least 5 minutes, alternating with periods of no longer than 10 minutes without CPR [10,44]. (See "Therapies of uncertain benefit in basic and advanced cardiac life support", section on 'Automatic compression devices'.)
Patients with moderate or severe hypothermia frequently become disproportionately hypotensive during rewarming from severe dehydration and fluid shifts [9, 16, 18]. Two large (14 or 16 gauge) peripheral IVs should be placed. Blood pressure is supported with warmed (40 to 42°C) infusions of isotonic crystalloid. Large infusions may be necessary. Use of warmed crystalloid is critical. Infusion of room-temperature fluids can worsen hypothermia. Intraosseous (IO) access may be easier to obtain than intravenous access in cold, vasoconstricted patients. IO lines should be primed with a 10 mL bolus of isotonic crystalloid with or without lidocaine immediately after insertion to open the marrow space and to help ensure good flow.

A central venous catheter may aid with fluid resuscitation. Temporary placement of a femoral venous catheter is preferred to avoid precipitating an arrhythmia from irritation of the right atrium. Low-dose (2 to 5 mcg/min) dopamine can be used to maintain blood pressure in cases refractory to volume resuscitation. Other vasopressors are acceptable, but studies to support their use have not been performed [45, 46]. An indwelling bladder catheter is helpful to assess urinary output and fluid shifts.

Core temperature, ideally esophageal temperature in intubated patients, should be monitored closely to assess the adequacy of therapy and to prevent iatrogenic hyperthermia (see 'Assessment' above). Rough movement and activity must be avoided, as they may provoke ventricular fibrillation.

**Duration of resuscitation** — Because of the neuroprotective effects of hypothermia, complete recovery of patients with hypothermia and cardiac arrest has been well documented despite prolonged resuscitation [19, 47-50]. Therefore, under usual circumstances, resuscitative efforts should be continued (occasionally for several hours) until the patient's core temperature reaches 32 to 35°C (90 to 95°F) [16]. Resuscitation should be withheld if the body is frozen so completely that chest compressions are impossible or if the nose and mouth are blocked with ice [51].

A number of biochemical markers can help inform the decision whether to continue resuscitation [11, 52, 53]. Extreme hyperkalemia reflects cell lysis and may predict a futile resuscitation. Survival is rare when the serum potassium concentration exceeds 12 mEq/L (mmol/L). Case reports suggest that lower cutoffs (eg, 8 mEq/L) should not be used [54]. Evidence of intravascular thrombosis (eg, fibrinogen concentration below 50 mg/dL [1.5 mmol/L]), ammonia concentrations above 420 mcg/dL (250 mmol/L), and elevated blood lactate or serum sodium or creatinine are additional indicators of a poor prognosis [55].

**Rewarming** — Individuals should be extracted from the hypothermic environment in the horizontal position whenever possible. Even low intensity use of peripheral muscles should be avoided, as muscular perfusion and consequently core temperature afterdrop is accelerated by exertion [9].

Rewarming should begin as soon as possible. Rewarming techniques are divided into passive external rewarming, active external rewarming, and active internal core rewarming. The degree of hypothermia determines the techniques implemented: mild hypothermia is treated with passive external rewarming; moderate and refractory mild hypothermia are
treated with active external rewarming; and severe (and some cases of refractory moderate) hypothermia is treated with active internal rewarming [56]. (See ‘Definitions’ above.)

**Passive external rewarming (PER)** — Passive external rewarming is the treatment of choice for mild hypothermia. It is also used as a supplemental method, when feasible, in patients undergoing aggressive rewarming for moderate to severe hypothermia. After wet clothing is removed, the patient is covered with blankets or other types of insulation. The resulting reduction in heat loss combines with the patient's intrinsic heat production to produce rewarming. Room temperature should be maintained at approximately 28°C (82°F), if possible.

PER may be unsuccessful in the setting of glycogen depletion, sepsis, or hypovolemia, especially in elderly patients. Many elderly patients lack normal metabolic and cardiovascular homeostasis, and will require active rewarming. The recommended rate of rewarming varies between 0.5 and 2°C/hour. Strongly consider implementing active rewarming measures if the rate falls below 0.5°C/hour or if dysrhythmias are present [20].

Passive external rewarming requires physiologic reserve sufficient to generate heat by shivering or by increasing the metabolic rate. Initially, the clinician often will not know if sufficient reserve is present. The elderly, malnourished, and those with severe cardiovascular disease or other major comorbidities are more likely to lack sufficient physiologic reserve.

Should the patient fail to respond to passive rewarming, more aggressive measures are implemented. Failure to rewarm should alert the clinician to the possibility of additional causes of hypothermia in addition to environmental exposure (table 5). (See ‘Differential diagnosis’ above.)

**Active external rewarming (AER)** — During active external rewarming, some combination of warm blankets, heating pads, radiant heat, warm baths, or forced warm air is applied directly to the patient's skin. These methods are indicated for moderate to severe hypothermia (<32°C) and for patients with mild hypothermia who are unstable, lack physiologic reserve, or fail to respond to passive external rewarming.

Especially in chronic hypothermia with dehydration, rewarming of the trunk should be undertaken **BEFORE** the extremities. These actions are performed in order to minimize core temperature afterdrop with associated hypotension and acidemia due to arterial vasodilation [16,18,57]. With forced air warming systems, the extremities can be left uncovered initially. This minimizes afterdrop but allows heat transfer.

Core temperature afterdrop is a particular risk of active external rewarming. This complication occurs when the extremities and trunk are warmed simultaneously. Cold, acidic blood that has pooled in the vasoconstricted extremities of the hypothermic patient returns to the core circulation, causing a drop in temperature and pH. At the same time, removal from the cold environment results in peripheral vasodilation, potentially contributing to precipitous hypotension, inadequate coronary perfusion, and ventricular fibrillation [9]. These phenomena may explain the fatal dysrhythmias that sometimes occur during rewarming [58,59]. Combining active core rewarming techniques with AER can minimize
rewarming shock and afterdrop in patients with severe hypothermia [60]. (See 'Active internal (core) rewarming (ACR)' below.)

Body surface burns are another possible complication of rewarming with heating pads. The combination of decreased sensation and reduced blood flow (which minimizes local heat dissipation) predisposes the hypothermic patient to surface burns. Therefore, the condition of the patient's skin should be frequently assessed during AER. Forced warm air systems are preferable.

The core temperature of patients rewarmed using either active external or active internal rewarming techniques generally increases at a rate of at least 2°C/hour. More hypothermic patients tend to rewarm at a faster rate [61].

**Active internal (core) rewarming (ACR)** — Active internal rewarming (also called active core rewarming) is the most aggressive strategy. It can be used alone or combined with active external rewarming (AER) in patients with severe hypothermia (<28°C) or patients with moderate hypothermia who fail to respond to less aggressive measures. In addition to IV administration of warmed crystalloid (40 to 42°C), effective techniques include irrigation of the peritoneum or the thorax (via the pleural space) with warmed isotonic crystalloid, and extracorporeal blood rewarming.

In general, we advocate a stepwise approach beginning with less invasive rewarming techniques. The combination of ACR, using heated IV infusions and warmed humidified oxygen, and ACR provides an effective initial strategy for most spontaneously perfusing, severely hypothermic patients. There is inadequate evidence to suggest that rapidly increasing the rate of rewarming improves outcomes in this population. In a patient who fails to respond to this approach, irrigation of the peritoneal and pleural cavities with heated crystalloid can be performed. Extracorporeal blood rewarming is performed in extreme cases (eg, cardiac arrest, frozen limb) or when rewarming is inadequate despite the other measures described here. Transferring patients to tertiary care centers is generally unnecessary unless they require extracorporeal rewarming. (See 'Extracorporeal blood rewarming' below.)

Although every increment of heat helps, some interventions have limited effect or pose a risk of complications. Airway rewarming with heated humidified oxygen decreases insensible heat loss and is a useful adjunct. However, airway rewarming provides only a modest benefit since most humidifiers cannot exceed 41°C without modification. The ideal setting is 45°C. Similarly, the amount of heat provided by IV fluid heated to 40 to 42°C only reaches significance during large volume resuscitations. Gastric or colonic irrigation can cause fluid and electrolyte fluctuations and thus is best avoided. In addition, gastric lavage risks pulmonary aspiration in these critically ill patients. The surface available for heat exchange in the bladder is too small to be of much benefit.

Peritoneal and pleural irrigation are the ACR techniques most likely to be employed in the emergency department (ED) should more basic interventions fail. Peritoneal irrigation can be performed by infusing 10 to 20 mL/kg of isotonic saline warmed to approximately 42°C. The fluid is left in the peritoneal cavity for 20 minutes and then removed. The overall exchange rate is 6 L/hour and is most easily accomplished with two catheters, one for instillation and
one for drainage [18]. Catheter placement is similar to that used for diagnostic peritoneal lavage.

Pleural irrigation can be accomplished by placing two thoracostomy tubes (36 to 40 French) in one or both hemithoraces [62]. One tube is placed high and anterior and the other placed low and posterior in the chest cavity. Warm isotonic saline at a temperature of 40 to 42°C is infused in 200 to 300 mL amounts through the anterior tube and allowed to drain posteriorly [63]. Reserve this technique for the severely hypothermic patient who is not rewarming unless the patient has an alternative indication for a chest tube. Do not insert left-sided tubes in a perfusing patient, since the heart is irritable. Leave the inferior tube for drainage after rewarming.

Endovascular warming devices are an effective and less invasive alternative to extracorporeal blood rewarming in patients who are not in cardiocirculatory arrest [64,65]. These devices are designed to aid in the use of therapeutic hypothermia. They use a femoral catheter that circulates temperature-controlled water inside a closed catheter tip in the femoral vein, warming or cooling blood as it flows past the tip. The thermostat is connected to an esophageal temperature probe and the machine is set to rewarm rapidly until it approaches the target temperature. The rewarming rate is then slowed in order to avoid overshooting. Some devices have a fail-safe mechanism that shuts the machine down when the core temperature sensor reads <30°C. This mechanism can be defeated by connecting the machine to a probe in warm water at 30°C until esophageal temperature reaches 30°C. Rewarming rates have been reported in the range of 2 to 3°C/hour.

**Extracorporeal blood rewarming** — Several techniques can be used to treat hypothermic patients by rewarming blood outside the body: venovenous rewarming, hemodialysis, continuous arteriovenous rewarming (CAVR), cardiopulmonary bypass (CPB), and extracorporeal membrane oxygenation (ECMO) [48,66-71] (see "Extracorporeal membrane oxygenation (ECMO) in adults").

Cardiopulmonary bypass and venoarterial ECMO are usually reserved for potentially salvageable patients with hemodynamic instability or cardiac arrest, patients who do not rewarm with less invasive active internal rewarming techniques, and those with completely frozen extremities or severe rhabdomyolysis with hyperkalemia. The technique selected depends upon clinical circumstance and available resources.

A venovenous rewarming circuit (without ECMO) requires two IV lines, one of which must be a central venous catheter. A femoral catheter is preferred to avoid the precipitation of dysrhythmia from irritation of the right atrium. Although venovenous rewarming provides no direct oxygenation or circulatory support, flow rates average 150 to 400 mL/minute and rewarming occurs at a rate of 2 to 3°C/hour. Hemodialysis achieves similar rewarming rates [72]. CAVR requires a systolic blood pressure of 60 mmHg [66]. Percutaneous 8.5 French femoral catheters direct blood through a countercurrent heat exchanger, yielding a rewarming rate of 3 to 4°C/hour. Venovenous ECMO is similar to CAVR but can perform both oxygenation and rewarming.

CPB can rewarm at rates up to 9.5°C/hour [67,73]. One retrospective series documented a 47 percent long-term survival rate with minimal sequelae among 32 patients with severe
hypothermia and cardiac arrest treated with CPB [48]. This relatively high survival rate following cardiac arrest likely reflects that many patients in the series were young and previously healthy and asphyxia and anoxic encephalopathy rarely preceded the development of hypothermia. Portable CPB units have been used to treat hypothermia [74].

Venoarterial ECMO, when available, is generally preferred to CPB because it allows prolonged oxygenation as well as circulatory support and can be used to treat noncardiogenic pulmonary edema, a common complication of hypothermia, and appears to improve survival [69]. Use of ECMO requires the administration of heparin, limiting its use in cases of hypothermia associated with bleeding problems including trauma [75].

Complications of rewarming — As mentioned above, patients with moderate or severe hypothermia frequently become disproportionately hypotensive during rewarming from severe dehydration and fluid shifts [9,16,18]. Aggressive fluid resuscitation with isotonic crystalloid is first line treatment. Use of vasopressors may be necessary. (See "Treatment of severe hypovolemia or hypovolemic shock in adults" and "Use of vasopressors and inotropes".)

Electrolyte abnormalities may develop during rewarming and should be treated as in normothermic patients. Atrial arrhythmias often develop during rewarming, but are generally benign and do not require treatment except in rare cases of rapid ventricular response. (See "Clinical manifestations and treatment of hypokalemia in adults" and "Treatment and prevention of hyperkalemia in adults" and "Treatment of hypocalcemia" and "Treatment of hypercalcemia" and "Overview of the treatment of hyponatremia in adults" and "Treatment of hypernatremia".)

Rhabdomyolysis and multi-organ system failure can develop during rewarming. Clinicians should not rely solely on initial laboratory values to exclude these conditions. (See "Clinical manifestations and diagnosis of rhabdomyolysis".)

Late, often fatal, complications, especially pulmonary, renal, and neurologic, are common after rewarming. Patients with significant hypothermia should be monitored closely in an intensive care setting throughout their resuscitation, including cardiac monitoring and serial laboratory measurements. (See 'Laboratory studies and monitoring' above.)

Failure to rewarm — In patients who fail to rewarm, the clinician should first ensure that appropriately aggressive rewarming techniques are in progress. Readily reversible contributing causes, such as hypoglycemia, must be addressed. The clinician next investigates other potential causes, including sepsis, adrenal insufficiency, and hypothyroidism (table 5). (See 'Rewarming' above.)

Hypothermic patients with an obvious source of infection should receive antibiotics. In addition, patients who fail to raise their core body temperature greater than 0.67°C/hour, despite appropriate rewarming efforts, should be treated empirically with broad spectrum intravenous (IV) antibiotics.

Rapid rewarming depends partly on the patient's inherent capacity for thermogenesis, which is compromised by underlying infection. This is suggested by a prospective observational
study of 88 consecutive hypothermic patients treated at a single major urban emergency department [20]. Despite the use of similar rewarming techniques, patients with hypothermia and an underlying infection had slower rewarming rates (below 0.67°C/hour) compared with patients without infection (above 1.67°C/hour). Patients with infection accounted for the great majority of deaths, and no patient died from a hypothermia-induced arrhythmia. The authors conclude that among urban patients with hypothermia, infection is a major cause of death that often manifests with slower rewarming rates.

Patients who fail to respond to aggressive rewarming measures may also have impaired thermogenesis from noninfectious causes, such as endocrine dysfunction, toxins, and lesions of the central nervous system (table 5). In addition to sepsis, conditions amenable to emergent treatment may include adrenocortical insufficiency and hypothyroidism.

Although no evidence exists to guide practice, we believe it is reasonable to treat potential adrenocortical insufficiency with a single dose of glucocorticoid in patients with moderate or severe hypothermia who fail to rewarm at an expected rate despite aggressive rewarming measures. Treatment with dexamethasone 4 mg IV or hydrocortisone 100 mg IV is reasonable. Dexamethasone is preferable because it is not measured in serum cortisol assays, if testing to determine adrenal function is performed. (See "Treatment of adrenal insufficiency in adults", section on 'Adrenal crisis'.)

If history, the patient's medication list, or a surgical scar in the area of the thyroid suggests that hypothyroidism may be contributing to a patient's failure to rewarm, it is reasonable to provide treatment. Severe hypothyroidism (myxedema coma) may be treated empirically with levothyroxine 250 mcg IV, after blood is drawn for thyroid function studies. Treatment for adrenal insufficiency is also required. (See "Myxedema coma", section on 'Treatment'.)

**Treatment of arrhythmia** — Hypothermic patients may exhibit cardiac irritability. Rough handling runs the risk of precipitating ventricular arrhythmias, including ventricular fibrillation. However, this concern should not prevent the performance of potentially life-saving interventions.

Bradycardia may be physiologic in severe hypothermia. Cardiac pacing generally is not required unless the bradycardia persists despite rewarming to 32 to 35°C (90 to 95°F). Atrial fibrillation and flutter do not cause a rapid ventricular response and often resolve spontaneously with rewarming, but the management of ventricular arrhythmias can be problematic [16,18,23,76]. Transcutaneous pacing is less hazardous than transvenous intracardiac pacing for a severe bradycardia associated with hypotension that is profoundly disproportionate to temperature [77].

Ventricular arrhythmias and asystole may be refractory to conventional therapy until the patient has been rewarmed. Thus, the definitive management of arrhythmia is focused on aggressively rewarmed the patient in conjunction with standard cardiac life support, including, most importantly, basic cardiopulmonary resuscitation using excellent technique. (See "Advanced cardiac life support (ACLS) in adults" and "Basic life support (BLS) in adults".)
Given the lack of evidence regarding management of ventricular arrhythmias in hypothermic patients, it is reasonable to treat patients according to ACLS guidelines, including defibrillation and administration of vasopressors (eg, epinephrine 1 mg IV or vasopressin 40 units IV) [42,78]. Defibrillation may be effective below 30°C [79,80]. It is easier to resuscitate a patient who is not in cardiac arrest than one who requires ongoing circulatory support. It is therefore reasonable to attempt defibrillation with a single shock, even in severe hypothermia. Further single attempts can be made with every 1 to 2°C increase in core temperature. Above 30°C, follow ACLS guidelines for normothermic patients.

Evidence to support drug therapy prior to successful rewarming is limited and consists primarily of animal studies. A systematic review of these studies found that vasopressor therapy had a higher association with return of spontaneous circulation than antiarrhythmia medications [81].

**Local injuries** — The management of cold-induced cutaneous injuries is generally supportive. Patients should receive tetanus toxoid and analgesia as indicated. Water bath rewarming (40 to 42°C) of affected areas for 15 to 30 minutes following stabilization of the core temperature may limit tissue loss [82]. Frostbite is discussed in detail elsewhere. (See "Frostbite").

**PROGNOSIS AND OUTCOME** — Few recent studies have assessed the prognosis of patients with moderate to severe hypothermia. In the largest study, performed over 35 years ago, factors associated with death within 24 hours of presentation included the following: prehospital cardiac arrest, low or absent blood pressure on presentation, elevated BUN, and the need for endotracheal intubation [83]. Notably, outcome did not correlate with core temperature at presentation. However, these results should be interpreted with caution as patients were not followed beyond 24 hours and no validation study was ever performed. In general, hypothermia associated with asphyxia by drowning or avalanche burial has a poor prognosis [69,84]. Otherwise, among healthy patients who develop accidental hypothermia and are hemodynamically stable at presentation, observational evidence suggests that almost all survive neurologically intact [85]. Among patients who sustain cardiac arrest from accidental hypothermia, the rate of neurologically intact survival is estimated to be approximately 50 percent when they are treated with extracorporeal circulation, and less than 37 percent with other treatment methods. However, this difference is likely to be more apparent than real as the patient populations studied differed substantially. Studies of treatment with extracorporeal techniques included predominantly young, healthy non-asphyxiated patients, while studies involving more standard treatments typically involved older patients with more comorbidities. There are many reports of neurologically intact survival without the use of extracorporeal circulation following cardiac arrest associated with hypothermia [63,85-87].

**INFORMATION FOR PATIENTS** — UpToDate offers two types of patient education materials, "The Basics" and "Beyond the Basics." The Basics patient education pieces are written in plain language, at the 5th to 6th grade reading level, and they answer the four or five key questions a patient might have about a given condition. These articles are best for patients who want a general overview and who prefer short, easy-to-read materials. Beyond
the Basics patient education pieces are longer, more sophisticated, and more detailed. These articles are written at the 10th to 12th grade reading level and are best for patients who want in-depth information and are comfortable with some medical jargon.

Here are the patient education articles that are relevant to this topic. We encourage you to print or e-mail these topics to your patients. (You can also locate patient education articles on a variety of subjects by searching on "patient info" and the keyword(s) of interest.)

- Basics topic (see "Patient education: Hypothermia (The Basics")

SUMMARY AND RECOMMENDATIONS — A table outlining the emergency management of hypothermia in adults is provided (table 1).

- Hypothermia is defined as a core temperature below 35°C (95°F), and can be further classified by severity:
  - Mild hypothermia: core temperature 32 to 35°C (90 to 95°F); findings include confusion, tachycardia, and increased shivering.
  - Moderate hypothermia: 28 to 32°C (82 to 90°F); findings include lethargy, bradycardia and arrhythmia, loss of pupillary reflexes, and decreased shivering.
  - Severe hypothermia: below 28°C (82°F); findings include coma, hypotension, arrhythmia, pulmonary edema, and rigidity (table 3). (See 'Definitions' above and 'Clinical presentation' above and 'Pathophysiology' above.)

- Causes and factors contributing to the development of hypothermia include outdoor exposure, cold water submersion, medical conditions (eg, hypothyroidism, sepsis), toxins (eg, ethanol abuse), and medications (eg, oral antihyperglycemics, sedative-hypnotics). Risk increases if the ability to autoregulate core temperature is impaired by medications or underlying disease, as is common among elderly patients (table 5). (See 'Differential diagnosis' above.)

- Proper diagnosis and management of hypothermia depend upon the use of a low-reading glass or electronic thermometer to determine core temperature. Many standard thermometers only read down to a minimum of 34°C (93°F). An esophageal temperature probe is preferred for severe hypothermia. (See 'Physical examination and temperature measurement' above.)

- Laboratory evaluation is used to identify potential complications and comorbidities, including lactic acidosis, rhabdomyolysis, bleeding diathesis, and infection. Moderate and severe hypothermia can cause dysrhythmia and prolongation of all electrocardiogram intervals. A list of studies to obtain is included in the text. (See 'Laboratory studies and monitoring' above and 'Electrocardiographic changes' above.)

- The initial management of hypothermia is directed toward resuscitation, assessment of the extent of injury, and rewarming. Endotracheal intubation is performed in patients with respiratory distress or who cannot protect their airway. Patients with moderate or severe hypothermia frequently become hypotensive; aggressive fluid resuscitation is appropriate. (See 'Airway, breathing, circulation' above.)

- Passive external rewarming is the treatment of choice for mild hypothermia and is a supplemental method in patients with moderate to severe hypothermia. After wet
clothing is removed, the patient is covered with blankets or other types of insulation. Room temperature should be maintained at approximately 28°C (82°F), if possible. (See 'Passive external rewarming (PER)' above.)

● Active external rewarming is used to treat patients with moderate hypothermia, refractory mild hypothermia, and as an adjunct in severe hypothermia. It consists of some combination of warm blankets, radiant heat, or forced warm air applied directly to the patient's skin. Rewarming of the trunk should be undertaken BEFORE the extremities to minimize the risk of core temperature afterdrop, hypotension, and acidemia. (See 'Active external rewarming (AER)' above.)

● For severe and recalcitrant moderate hypothermia, we suggest initiating treatment with less invasive rewarming techniques (eg, warmed IV isotonic crystalloid), and progressively adding more invasive ones (eg, warmed pleural lavage) as needed. Extracorporeal blood rewarming techniques are used to treat perfusing patients who fail to rewarm, have completely frozen extremities, or have severe rhabdomyolysis and hyperkalemia. For salvageable, non-perfusing patients with severe hypothermia, we suggest treatment with extracorporeal membrane oxygenation (ECMO) or cardiopulmonary bypass when that option is readily available (Grade 2C). (See 'Active internal (core) rewarming (ACR)' above.)

● Rough handling of the moderate or severe hypothermic patient can precipitate arrhythmias, including ventricular fibrillation, that are often unresponsive to defibrillation and medications. Cardiopulmonary resuscitation (CPR) should continue until the patient is rewarmed to 30 to 32°C (86 to 90°F), at which point renewed attempts at defibrillation and resuscitation with ACLS medications are undertaken. The neuroprotective effects of low temperature may allow recovery following prolonged arrest. (See 'Treatment of arrhythmia' above.)

● For patients who fail to rewarm appropriately despite aggressive rewarming measures, we suggest treatment with empiric broad spectrum antibiotics and a single dose of glucocorticoid (Grade 2C). Such patients may also need treatment for hypoglycemia, myxedema coma, or other contributing causes. (See 'Failure to rewarm' above.)

● After resuscitation, pay careful attention to potential complications, including hypotension during active rewarming, arrhythmia, hyperkalemia, hypoglycemia, rhabdomyolysis, bladder atony, and bleeding diathesis. (See 'Complications of rewarming' above.)

● Local cold-induced injuries vary in severity independent of the degree of systemic hypothermia. Severe injuries may require prolonged supportive management, escharotomy, or amputation. (See "Frostbite").

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REFERENCES

50. Hilmo J, Naesheim T, Gilbert M. "Nobody is dead until warm and dead": prolonged resuscitation is warranted in arrested hypothermic victims also in remote areas—a retrospective study from northern Norway. Resuscitation 2014; 85:1204.