Accidental hypothermia in adults

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INTRODUCTION

Death from exposure and accidental hypothermia occurs throughout the world and can present significant management problems [1-4]. 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 [5]. Cases of hypothermia can occur during the summer months and in hospitalized patients [6]. The rate of neurologically intact survival in patients with hypothermic cardiac arrest has been estimated to be 47 to 63 percent with the use of extracorporeal life support (ECLS) using extracorporeal membrane oxygenation (ECMO) or cardiopulmonary bypass (CPB) [7-9].

The definition, pathophysiology, and management of accidental hypothermia will be discussed here. A table and algorithm outlining the emergency management of hypothermia in adults are provided (table 1 and algorithm 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)".)
- (See "Frostbite: Emergency care and prevention".)
- (See "Hypothermia in children: Clinical manifestations and diagnosis" and "Hypothermia in children: Management".)
- (See "Hypothermia in children: Management".)

DEFINITIONS AND STAGES OF HYPOTHERMIA

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 [10,11]:

- 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 <28°C (82°F)

In addition, some experts regard a core temperature <24°C (75°F) or <20°C (68°F) as "profound hypothermia" [12,13].

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 should measure core temperature, preferably using an esophageal probe in the lower third of the esophagus, in an unresponsive patient. (See 'Temperature measurement' below.)

If temperature measurement is not feasible, pre-hospital personnel should estimate the severity of hypothermia by observing clinical signs. Because the response to hypothermia varies widely among patients, any clinical staging system can only provide a rough estimate of severity (table 2) [14].

- **Cold stress (not hypothermia)** – Normal mental status with shivering. Functioning normally. Able to care for self. Estimated core temperature 35 to 37°C (95 to 98.6°F).
- **Mild hypothermia** – Alert, but mental status may be altered. Shivering present. Not functioning normally. Not able to care for self. Estimated core temperature 32 to 35°C (90 to 95°F).
- **Moderate hypothermia** – Decreased level of consciousness. Conscious or unconscious, with or without shivering. Estimated core temperature 28 to 32°C (82 to 90°F).
- **Severe/profound hypothermia** – Unconscious. Not shivering. Estimated core temperature <28°C (<82°F).

Pre-hospital personnel, especially in Europe, may use the clinical staging scheme described

by the International Commission for Mountain Emergency Medicine (this is sometimes referred to as "the Swiss system") (table 3) [12]:

- **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).

A potential limitation of the Swiss 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 [15,16].

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 [17]. The lowest observed core temperatures were 28.1°C for HT 1, 22.1°C for HT II, and 19.3°C for HT III. A further analysis of 305 hospital and published cases found that only 61 percent of patients were assigned to the correct stage [18]. There were large overlaps among the four stages. Core temperature was overestimated in 18 percent and underestimated in 21 percent of patients. The authors calculated that the optimal temperature thresholds to classify patients into the four stages were 32.1, 27.5, and 24°C, which were similar to the original cutoffs of 32, 28, and 24°C.

The Swiss system is being replaced by the Revised Swiss System (RSS) as a recommendation of the International Commission for Mountain Emergency Medicine [19]. The RSS stages hypothermia based on the level of consciousness (using the Alert, Verbal, Painful, Unconscious [AVPU] scale) and the presence or absence of vital signs (figure 1). Shivering will no longer be a stage-defining sign. The emphasis of the RSS is on the risk of cardiac arrest rather than core temperature. Alert patients (mild hypothermia) have a low risk of cardiac arrest, patients who respond to verbal stimuli have a moderate risk, and patients who respond only to painful stimuli or have no response (unconscious) have a high risk. Patients who are unconscious without detectable vital signs are considered to be in

hypothermic cardiac arrest. As with all hypothermia grading systems, the RSS applies only to uncomplicated hypothermia and not to patients with comorbidities such as trauma or sepsis. The risk of hypothermic cardiac arrest would likely be overestimated in patients with comorbidities that might affect the level of consciousness.

A similar system, unrelated to the RSS but only using level of consciousness, has been used in Denmark since 2004 [20]. The Danish system has three grades: mild (awake), moderate (unconscious), and severe (lifeless). Severe patients were treated with cardiopulmonary resuscitation (CPR) and extracorporeal membrane oxygenation (ECMO). Retrospective studies have found that level of consciousness alone may be as accurate as core temperature in predicting the risk of cardiac arrest and mortality [21,22]. In one study of 114 patients, for example, none of the 33 fully alert patients had a cardiac arrest, 3 of the 12 patients who were alert but confused had cardiac arrests, and two-thirds of the 43 patients who were unresponsive had cardiac arrests [21].

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 [23]:

- 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 [24].

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 [24,25]. 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

Clinical signs and their progression with worsening hypothermia — As the compensatory mechanisms preventing hypothermia are overwhelmed, the following changes typically occur (table 4) [8,10,26]:

- 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 at the lower end of the moderate hypothermia core temperature range. Paradoxical undressing may be observed. Atrial fibrillation, junctional bradycardia, and other arrhythmias can occur. The pupillary light reflex is depressed through slowing of both constriction and dilation [27]. Dilated pupils are seen below a core temperature of approximately 29°C.

- Severe hypothermia can lead to pulmonary edema, oliguria, areflexia, coma, hypotension, bradycardia, ventricular arrhythmias (including ventricular fibrillation), and asystole [24,28]. Loss of corneal and oculocephalic reflexes may be noted but do not correlate with temperature [29]. Signs that might portend a poor prognosis in normothermic patients (eg, fixed, dilated pupils; loss of brainstem reflexes; and apparent rigor mortis) reverse with rewarming if the patient survives.

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

retrospective study of 216 hypothermic patients found generally positive linear correlations between core temperature and heart rate, systolic blood pressure, respiratory rate, and Glasgow Coma Scale (GCS) score (table 5) [30]. A calculator to estimate the expected vital signs based on body temperature can be found at the Hypothermia Outcome Prediction after ECLS project at the University Hospital of Lausanne.

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 [2,31].

Geriatric population — Older adults are at increased risk of developing hypothermia and its complications and should be urgently assessed if found to be hypothermic [31,32]. 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 older adult 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 — 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.

Clinical findings associated with the progressive stages of hypothermia are described above. (See 'Clinical signs and their progression with worsening hypothermia' above.)

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: Emergency care and prevention".)

Temperature measurement — Effective diagnosis and management of hypothermia depend upon the use of a low-reading thermometer to determine core temperature [33,34]. 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 (approximately 24 cm below the larynx) provides a near approximation of cardiac temperature [13]. Esophageal temperature is the most accurate method to track the progress of rewarming. An instructional video demonstrating esophageal temperature measurement can be found in the following reference [35,36].

Rectal probe readings may rise following peritoneal lavage or fall if adjacent to cold feces; esophageal probes advanced to the upper two-thirds 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 [13].

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.

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
- Blood urea nitrogen (BUN) and creatinine
- Serum hemoglobin, white blood cell, and platelet counts
- Serum lactate

- Fibrinogen
- Creatine kinase (CK)
- Lipase
- 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. Serum cortisol and thyroid function studies might be obtained in a patient with moderate or severe hypothermia who did not have an environmental exposure or 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 [37]. Hyperglycemia that persists during rewarming suggests pancreatitis or diabetic ketoacidosis. Pancreatitis is common in hypothermia [38]. Pancreatitis may lead to diabetic ketoacidosis in hypothermic patients [39]. 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 6) [24-26,28,40-42]. 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 [23].

Inhibition of the enzymes of the coagulation cascade from hypothermia leads to a bleeding diathesis [41]. 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 [43]. 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, partial pressure of carbon dioxide ($PaCO_2$), and partial pressure of oxygen (PO_2) of a

blood sample all vary with the temperature, as gas tensions and hydrogen ion concentration decline as temperature drops (table 7) [44,45]. Use uncorrected values (those measured by the blood gas analyzer at 37°C) 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 [44]. An uncorrected pH of 7.4 and a $PaCO_2$ of 40 mmHg reflect normal acid-base balance. Use of uncorrected pH values (ie, alpha-stat strategy) appears to be a better prognostic indicator compared with correcting for the patient's temperature (ie, pH-stat strategy) [46]. (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 [47].

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) [48]. The height of the J wave is roughly proportional to the degree of hypothermia [49,50]. 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 [51]. (See "Electrocardiogram in the diagnosis of myocardial ischemia and infarction", section on 'Early repolarization'.)

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 35°C (95°F). Proper diagnosis depends upon the use of a low-reading thermometer to determine the core temperature accurately. (See 'Assessment' above.)

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 [52]. Hypothermia has even been misdiagnosed as psychiatric illness, including attempted suicide. The differential diagnosis of hypothermia is summarized in the following table (table 8) [24,26,33,40,53-55].

Certain medications directly or indirectly cause hypothermia 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 [56].

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 carbon dioxide 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.

MANAGEMENT

While managing the patient with significant hypothermia, keep in mind that 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.

Resuscitation — 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 [14,40,57]. A table and algorithm outlining the emergency management of hypothermia in adults are provided (table 1 and algorithm 1). (See "Advanced cardiac life support (ACLS) in adults" and "Rapid sequence intubation in adults for emergency medicine and critical care".)

Endotracheal intubation is performed in patients with respiratory distress or those 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.

Cardiopulmonary resuscitation — 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 "do not resuscitate" (DNR) 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. Fixed and dilated pupils are not a contraindication to starting CPR. Apparent rigor mortis is not a reliable sign of death. Mandibular rigidity can simulate rigor mortis [58].

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 is likely to be transient. There is little downside to withholding CPR briefly in these hypothermic patients. Should pulseless electrical activity 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

blood pressure monitoring or with Doppler ultrasound. Transesophageal echocardiography (TEE) is more sensitive than standard bedside echocardiography. TEE is preferred if it is available at the bedside.

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. Only if continuous CPR is not possible, CPR can be delayed up to 10 minutes while rescuers move the patient to a safer location. In a patient with a core temperature of 20 to 28°C, or if the core temperature is not known, administer CPR continuously for periods of at least five minutes, alternating with periods of no longer than five minutes without CPR. In patients with a core temperature below 20°C, administer CPR continuously for periods of at least five minutes, alternating with periods of no longer than 10 minutes without CPR [11,59]. Once the patient has reached an ambulance or hospital, resume continuous CPR. (See "Therapies of uncertain benefit in basic and advanced cardiac life support", section on 'Mechanical compression devices'.)

Ventricular arrhythmias and asystole — In hypothermic patients in cardiac arrest, ventricular arrhythmias and asystole may be refractory to conventional therapy until the patient has been rewarmed. The definitive management of ventricular arrhythmia is focused on aggressively rewarming 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 "Adult basic life support (BLS) for health care providers".)

Given the lack of evidence regarding management of ventricular arrhythmias in hypothermic patients, it is reasonable to treat patients in cardiac arrest according to advanced cardiac life support (ACLS) guidelines, including defibrillation and administration of epinephrine 1 mg intravenously (IV). Vasopressin 40 units IV may be used instead of epinephrine [60]. Defibrillation may be effective below 30°C [61-63]. Most published cases of successful defibrillation below 30°C involve core temperatures in the mid-20s, but there is a case report of successful defibrillation by an automatic implantable cardioverter defibrillator in a patient with a core temperature of 18.2°C [64].

It is easier to resuscitate a patient who is not in cardiac arrest than one who requires ongoing circulatory support. Therefore, it is 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 antiarrhythmic medication, such as amiodarone [65]. There was no advantage to using intermediate or high-dose epinephrine (45 mcg/kg or 200 mcg/kg).

Hypotension — Patients with moderate or severe hypothermia frequently become disproportionately hypotensive during rewarming due to severe dehydration and fluid shifts [10,24,26]. Two large (14- or 16-gauge) peripheral IV lines 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 IV 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. An indwelling bladder catheter is helpful to assess urinary output and fluid shifts. (See "Intraosseous infusion".)

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. (See "Placement of femoral venous catheters".)

Norepinephrine should be used to maintain blood pressure in cases refractory to volume resuscitation. If norepinephrine is not available, other vasopressors are acceptable. Only two studies have addressed this question, and low-dose dopamine was used in both [66,67]. (See "Use of vasopressors and inotropes".)

Arrhythmias not causing cardiac arrest — 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 lifesaving interventions. The management of hypothermic patients with cardiac arrest is discussed above. (See 'Ventricular arrhythmias and asystole' above.)

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 [24,26,33,68]. Transcutaneous pacing is less hazardous than transvenous intracardiac pacing for severe bradycardia associated with hypotension that is profoundly

disproportionate to temperature [69]. (See "Advanced cardiac life support (ACLS) in adults", section on 'Bradycardia'.)

Temperature monitoring — Core temperature, ideally esophageal temperature in intubated patients, should be monitored closely to assess the adequacy of therapy and to prevent iatrogenic hyperthermia. (See 'Temperature measurement' above.)

An instructional video demonstrating esophageal temperature measurement can be found in the following reference [35,36]. 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 [8,70-75]. Resuscitative efforts should only be withheld if there is a nonsurvivable injury or fatal illness, if the body is so frozen that chest compressions are impossible, or if the nose and mouth are blocked with snow or ice [11,76]. Efforts should be continued, sometimes for several hours [24]. Unlike in normothermic patients with cardiac arrest, do not terminate resuscitation for low end-tidal CO_2 (less than 10 mmHg) in hypothermic patients with cardiac arrest. Low end-tidal CO_2 may reflect a low metabolic rate rather than poor perfusion [46].

A number of biochemical markers can help inform the decision whether to continue resuscitation [12,77,78]. Extreme hyperkalemia reflects cell lysis and may predict a futile resuscitation. There are no reported cases of survival when the patient's serum potassium concentration exceeds 12 mEq/L (mmol/L). Case reports suggest that lower cutoffs (eg, 8 mEq/L) should not be used [79], except for avalanche victims because cardiac arrest in these patients is usually caused by asphyxia rather than hypothermia. For avalanche victims, a retrospective study reported that the optimal cutoff for potassium is 7 mEq/L (mmol/L) [80].

A single marker should not be used alone to decide whether to continue resuscitation. Reported serum potassium values may be falsely elevated due to hemolysis. In addition, measurements may vary by over 3 mmol/L depending on the sampling site (central venous, peripheral venous, or arterial). When available, a central venous blood sample should be used to measure the serum potassium as these appear to be most consistent [81].

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 [82].

Rewarming interventions based on severity of hypothermia — A table and algorithm outlining the emergency management of hypothermia in adults are provided (table 1 and algorithm 1).

Individuals should be extracted from the cold 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 [10].

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 [8,83]:

- Cold-stressed patients and those with mild hypothermia are treated with passive external rewarming, but active external rewarming should be added to decrease metabolic load and increase comfort. Active external rewarming may be necessary for older adults, malnourished patients, and patients with cardiovascular disease or other comorbidities.
- Moderate and refractory mild hypothermia are treated with active external rewarming.
- Severe (and some cases of refractory moderate) hypothermia is treated with active internal rewarming and possibly extracorporeal rewarming. (See 'Definitions and stages of hypothermia' above.)

Passive external rewarming — Passive external rewarming is used for mild hypothermia. It is also used 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.

Passive external rewarming requires physiologic reserve sufficient to generate heat by shivering or by increasing the metabolic rate. Passive external rewarming alone may be unsuccessful in the setting of glycogen depletion, sepsis, or hypovolemia, especially in older adult patients. Many older adult patients lack normal metabolic and cardiovascular homeostasis and require active rewarming.

The recommended rate of rewarming varies between 0.5 and 2°C/hour. If available, active external rewarming is routinely added to passive rewarming for patient comfort and to

decrease cardiovascular energy requirements. Active rewarming measures should be implemented if the rate of rewarming falls below 0.5°C/hour, dysrhythmias are present, or the patient fails to respond to passive rewarming [28]. Failure to rewarm should alert the clinician to the possibility of additional causes of hypothermia in addition to environmental exposure (table 8). (See 'Differential diagnosis' above.)

Active external rewarming — 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 lack physiologic reserve or fail to respond to passive external rewarming. Active external rewarming provides an effective initial strategy for most spontaneously perfusing, severely hypothermic patients.

There is insufficient evidence to endorse an optimal active external rewarming technique. Temperature regulation devices traditionally used for therapeutic hypothermia have been used successfully but are far more expensive than forced air warming systems [84].

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 [24,26,85]. With forced air warming systems, the extremities can be left uncovered initially. This minimizes afterdrop but allows heat transfer.

Hazardous core temperature afterdrop is a particular risk of active external rewarming. This complication occurs when the extremities and trunk are warmed simultaneously. Cold, acidemic 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 [10]. These phenomena may explain the fatal dysrhythmias that sometimes occur during rewarming [86,87]. Combining active core rewarming techniques with active external rewarming can minimize rewarming shock and afterdrop in patients with severe hypothermia [88] (see 'Active internal (core) rewarming' below). However, there is no evidence that prehospital external rewarming is dangerous for patients with accidental hypothermia [89].

Body surface burns are a 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 active external rewarming. 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. Colder patients tend to rewarm at a faster rate [13]. There is no evidence that increasing the rate of rewarming improves outcomes in spontaneously perfusing patients.

Active internal (core) rewarming — Active internal rewarming (also called active core rewarming) is the most aggressive strategy. It can be used alone or combined with active external rewarming in patients with severe hypothermia (<28°C) or patients with moderate hypothermia who fail to respond to less aggressive measures. Endovascular rewarming is the method of choice for patients not requiring extracorporeal life support (ECLS), or if ECLS is not available.

Endovascular temperature control catheters are widely available. If not available, alternative rewarming methods include irrigation of the peritoneum or the thorax (via the pleural space) with warmed isotonic crystalloid.

If available, ECLS should be used to rewarm patients in cardiac arrest or with cardiovascular instability and in special cases, such as patients with frozen limbs preventing IV or IO access. ECLS should also be used when rewarming is inadequate despite the other measures. Transferring patients to tertiary care centers is generally unnecessary unless they require extracorporeal blood rewarming or ECLS. (See 'Extracorporeal life support' below.)

Endovascular temperature-control catheters are an effective and less invasive alternative to extracorporeal blood rewarming in patients who are not in cardiocirculatory arrest [90,91]. 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 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. However, endovascular

rewarming has not been demonstrated to be more rapid than the other rewarming techniques described here [92].

When endovascular warming devices are unavailable, alternative approaches include peritoneal and pleural irrigation with warmed isotonic fluid. 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 [26]. Catheter placement is similar to that used for diagnostic peritoneal lavage. (See "Diagnostic peritoneal lavage (DPL) or aspiration (DPA)", section on 'DPL/DPA technique'.)

Pleural irrigation can be accomplished by placing two thoracostomy tubes large enough to allow unimpeded rapid infusion and drainage of saline (36 to 40 French) in one or both hemithoraces [93]. One tube is placed high and anterior, and the other is 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 [94]. This technique should be reserved 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. (See "Thoracostomy tubes and catheters: Placement techniques and complications".)

Some interventions have limited effect or pose a risk of complications. Airway rewarming and heated IV or IO fluids should not be used as primary rewarming methods. They are used primarily to decrease heat loss when active rewarming methods are used. Airway rewarming with heated, humidified oxygen decreases insensible heat loss but provides limited benefit as most humidifiers cannot exceed 41°C without modification and the ideal setting is 45°C. As with heated oxygen, the benefit of administering warm IV fluid (generally heated to 40 to 42°C) is limited. Even large volumes have minimal effect on raising temperature.

Gastric or colonic irrigation can cause fluid and electrolyte fluctuations, and both are best avoided. In addition, gastric lavage risks pulmonary aspiration in critically ill patients. The surface available for heat exchange in the bladder is too small to be of much benefit.

A minimally invasive method of rewarming in a case of severe accidental hypothermia, using an esophageal heat transfer device, has been described [95]. The device is an orogastric tube with additional lumens designed to circulate temperature-controlled water in a closed circuit

that was developed for cooling in targeted temperature management. This device shows promise for use in accidental hypothermia. As with endovascular warming devices, use of an esophageal heat transfer device avoids volume overload and electrolyte shifts.

Extracorporeal life support — Several ECLS 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) [71,96-102]. CPB and venoarterial ECMO are usually reserved for 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 [103]. The technique selected depends upon clinical circumstance and available resources. (See "Extracorporeal membrane oxygenation (ECMO) in adults".)

- **Venovenous rewarming** – 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 can achieve similar rewarming rates [104-106]. CAVR requires a systolic blood pressure of 60 mmHg [96]. 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.

- **Cardiopulmonary bypass** – CPB can rewarm at rates up to 9.5°C/hour [97,107]. 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 [71]. 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 [108].

- **Extracorporeal membrane oxygenation** – 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 [99,109-111]. In a meta-analysis of 23 observational studies with 464 patients, overall survival after

hypothermic cardiac arrest was better with ECMO compared with CPB (44 versus 31 percent, RR 1.41, 95% CI 1.11-1.80). In survivors, 80 percent had good neurologic outcomes that were slightly worse with ECMO compared with CPB (75 versus 87 percent, RR 0.86, 95% CI 0.75-0.99) [112]. Use of ECMO requires the administration of heparin, limiting its use in cases of hypothermia associated with bleeding problems including trauma [113].

- **Optimal rewarming rate with ECLS** – Slower rewarming rates with ECLS are associated with improved survival and neurologic outcomes, but evidence is insufficient to determine the optimal rewarming rate. A secondary analysis of a meta-analysis of 658 patients with accidental hypothermia treated with ECLS-assisted rewarming found the optimal cutoff value for good neurological outcomes was less than 5°C/hour [114].

Complications of rewarming — As mentioned above, patients with moderate or severe hypothermia frequently become disproportionately hypotensive during rewarming from severe dehydration and fluid shifts [10,24,26]. 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".)

Takotsubo cardiomyopathy has been reported [115]. (See "Clinical manifestations and diagnosis of stress (takotsubo) cardiomyopathy" and "Management and prognosis of stress (takotsubo) cardiomyopathy".)

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 in adults".)

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

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 8). (See 'Rewarming interventions based on severity of hypothermia' 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 0.7°C/hour, despite appropriate rewarming efforts, should be treated empirically with broad-spectrum 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 [28]. 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 8). In addition to sepsis, conditions amenable to emergency 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'.)

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 [116]. Frostbite is discussed in detail elsewhere. (See "Frostbite: Emergency care and prevention".)

PROGNOSIS AND OUTCOME

General population — Evidence is limited and conflicting regarding the prognosis of patients with severe hypothermia [102,117,118]. Factors associated with a worse prognosis are summarized in the table (table 9).

In a large retrospective study of hypothermia outcomes performed over 25 years ago, factors associated with death within 24 hours of presentation included prehospital cardiac arrest, low or absent blood pressure on presentation, elevated blood urea nitrogen (BUN), and the need for endotracheal intubation [119]. 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 [99,120,121]. Among healthy patients who develop accidental hypothermia and are hemodynamically stable at presentation, evidence suggests that almost all survive neurologically intact [121,122].

Outcomes of accidental hypothermia sustained during outdoor activities have not been systematically studied. In the International Hypothermia Registry, which included 201 non-consecutive cases, the main cause of hypothermia was "mountain accidents" mostly in young males [123]. Survival in patients without cardiac arrest was 95 percent.

There are a few observational studies of outcomes among urban dwellers. In a registry study involving 358 patients treated at 12 Japanese emergency departments, factors associated with increased in-hospital mortality from accidental hypothermia included age ≥75 years, frailty, hemodynamic instability, and hyperkalemia [124]. A retrospective, single-center study of 67 patients found that increased in-hospital mortality was associated with age ≥70 years, mean arterial pressure <90 mm Hg, pH <7.35, creatinine >1.5 mg/dL (133 micromol/L), and

confusion [125].

Cardiac arrest — Witnessed cardiac arrest is associated with relatively favorable neurologic outcomes, while unwitnessed cardiac arrest and asphyxia are associated with worse outcomes [121]. When cardiac arrest occurs during rescue, it is referred to as "rescue collapse" or "circumrescue collapse."

Survival in 73 patients with cardiac arrest was 36 percent in the International Hypothermia Registry [123]. Patients with witnessed cardiac arrest were more likely to survive (71 versus 30 percent). Other predictors of survival included return of spontaneous circulation before rewarming, normal potassium, and absence of asphyxia.

A systematic review including 214 patients with accidental hypothermia and witnessed cardiac arrest reported a mean core temperature at arrest of 24±3°C, with a range of 15.2 to 32.2°C [126]. No patient with uncomplicated hypothermia and a core temperature above 30°C sustained cardiac arrest. The highest core temperature in a survivor was 29.4°C. Survival to hospital discharge was approximately 73 percent. Neurologic outcomes were described as "favorable" for 102 of 115 patients (89 percent) discharged from the hospital for whom outcome data was available.

A systematic review of 221 patients with unwitnessed hypothermic cardiac arrest rewarmed with extracorporeal life support (ECLS) found a survival rate of 27 percent. Most of the survivors (83 percent) had no neurologic deficit, including patients whose presenting rhythm was asystole. Predictors of survival included female sex, non-asphyxial mechanism of cooling, pulseless electrical activity as the initial rhythm, and normal serum potassium [127].

Using data from a Japanese registry of patients with out-of-hospital cardiac arrest, a study of a subset of 754 hypothermic patients reported that one-month survival was approximately 6 percent [128]. Factors associated with increased mortality included pH <6.9, lactate >13.3 mmol/L (120 mg/dL), and potassium >7 mmol/L (7 mEq/L).

Witnessed cardiac arrest **after** extrication from an avalanche in patients with a core temperature below 24°C is associated with a relatively favorable neurologic outcome [129]. Good neurologic outcome after unwitnessed cardiac arrest in an avalanche is very rare [130].

Extracorporeal life support — Although there are many reports of neurologically intact survival without the use of ECLS following cardiac arrest associated with hypothermia [94,122,131-133], such interventions may improve survival in some patients, as suggested by the following studies:

- In a meta-analysis of 44 observational studies and 40 case reports, including a total of 658 patients with accidental hypothermia treated with ECLS, the rate of survival with good neurologic outcome was 40.3 percent [102]. Absence of asphyxiation, serum potassium <5 mmol, and female sex were associated with increased survival with good neurologic outcome. None of these factors, alone or in combination, should be used as the sole basis to decide whether to initiate ECLS. Slower rewarming rates using extracorporeal circulation were also associated with higher rates of neurologically intact survival, but the effect size was small.

- In a study of 204 patients with hypothermia initially treated by the Danish Air Force Search and Rescue Service, 47 were considered unsalvageable and 157 were treated, of whom 108 (69 percent) survived [20]. Of the 54 patients treated with ECLS, 21 (39 percent) survived to hospital discharge.

- A retrospective registry (HELP registry) study of 98 non-asphyxiated patients in hypothermic cardiac arrest treated with ECLS reported a survival rate of 53 percent, with 94 percent of survivors having good neurologic outcomes [134]. In multivariate analysis, the only favorable prognostic factors were lower age and lower serum lactate concentration. The mean age of survivors was approximately 50 (±13 years), while the mean age of non-survivors was 58 (±15 years).

- In a multicenter, prospective study in Japan (ICE-CRASH) of 242 mostly older adult patients (mean age 81 years) with severe accidental hypothermia (defined as cardiac arrest, systolic blood pressure <60 mm Hg, or heart rate <50 beats per minute), the rate of survival at 28 days was 65 percent and rate of favorable neurologic outcome was 53 percent [111]. In the 57 patients with cardiac arrest, those who received ECLS had better 28-day survival (58 versus 21 percent, OR 0.17, 95% CI 0.05-0.58) and favorable neurologic outcome (42 versus 15 percent, OR 0.22, 95% CI 0.06-0.81). In patients without cardiac arrest, ECLS did not improve 28-day survival, led to more adverse events, and increased the frequency of bleeding complications. These results may not be generalizable to other populations given the advanced age of the patients and the predominance of indoor occurrence of accidental hypothermia.

Among the subset of patients who sustain cardiac arrest from accidental hypothermia, the rate of neurologically intact survival has been reported to be significantly higher among those treated with ECLS compared with other treatment methods [99]. However, this purported difference is likely to be more apparent than real, as patient populations differ substantially among studies. Some studies included hypothermic patients with associated

trauma or asphyxia [122], as in the case of avalanche victims, while others focused on isolated hypothermia [134]. Studies of treatment with extracorporeal techniques have included predominantly young, healthy, non-asphyxiated patients, while studies involving more standard treatments typically involved older patients with more comorbidities. In addition, the worse outcomes among patients who sustain cardiac arrest do not vary based on treatment with ECLS [135].

In Europe, the most widely used instrument to decide whether to use ECLS to treat in patients hypothermic cardiac arrest is the Hypothermia Outcome Prediction after ECLS (HOPE) survival probability score [117]. The HOPE score was developed based on the outcomes of 286 patients: 237 patients from 18 published studies and 49 additional patients from hospital data. Overall survival was 37 percent. Favorable prognostic factors at hospital admission were female sex, mechanism other than asphyxiation (ie, exposure or immersion rather than submersion or avalanche burial), greater age (in contrast to HELP registry study [134]), lower serum potassium concentration, shorter duration of cardiopulmonary resuscitation (CPR), and lower core temperature. Witnessed arrest and cardiac activity (ie, pulseless electrical activity or ventricular fibrillation rather than asystole) were associated with improved survival but were not included in the score. The score involves a complex equation that can be calculated online at www.hypothermiascore.org.

Individual factors such as age have limited prognostic value. The median age of survivors was 40 (range 18 to 56), while the median age of non-survivors was 29.5 (range 13 to 54). In a retrospective, external validation study of the HOPE score using data from 122 patients, overall survival was 42 percent, and the negative predictive value was 97 percent [118].

Compared with serum potassium alone (a criterion commonly used to determine eligibility for ECLS), use of the HOPE score would have avoided unsuccessful rewarming in 27 percent of patients with a potassium ≤12 mmol/L without any additional deaths [117]. As the HOPE score is based on data from retrospective observational studies, it has methodologic limitations and should be interpreted with caution. Possible problems include selection bias and substantial overlap between groups for some parameters, such as age, duration of CPR (median 106 minutes for survivors and 120 minutes for non-survivors), and core temperature (median 23°C for survivors and 25°C for non-survivors).

SOCIETY GUIDELINE LINKS

Links to society and government-sponsored guidelines from selected countries and regions

around the world are provided separately. (See "Society guideline links: Management of environmental emergencies" and "Society guideline links: Hypothermia".)

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

- **Emergency care** – A table and algorithm outlining the emergency management of hypothermia in adults are provided (table 1 and algorithm 1).
- **Definitions** – Hypothermia is defined as a core temperature below 35°C (95°F) and can be further classified by severity (table 2):
  - Mild hypothermia – Core temperature 32 to 35°C (90 to 95°F); findings include confusion, tachycardia, and increased shivering.
  - Moderate hypothermia – Core temperature 28 to 32°C (82 to 90°F); findings include lethargy, bradycardia and arrhythmia, decreased or absent pupillary reflexes, and decreased or absent shivering.
  - Severe hypothermia – Core temperature below 28°C (82°F); findings include coma, hypotension, arrhythmia, pulmonary edema, and rigidity. (See 'Definitions and stages of hypothermia' above and 'Clinical presentation' above and

'Pathophysiology' above.)

- **Causes and risk factors** – 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 older adult patients (table 8). (See 'Differential diagnosis' above.)

- **Diagnosis with low-reading thermometer** – 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' above and 'Temperature measurement' above.)

- **Laboratory testing** – 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.)

- **Resuscitation** – The initial management of hypothermia is directed toward resuscitation, assessment of the extent of injury, and rewarming (table 1 and algorithm 1). 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 'Resuscitation' above.)

- **Mild hypothermia management** – 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' above.)

- **Moderate and refractory mild hypothermia management** – Active external rewarming is used to treat patients with moderate hypothermia and refractory mild hypothermia, and as an adjunct in severe hypothermia (table 1 and algorithm 1). 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' above.)

- **Severe hypothermia management** – For severe and recalcitrant moderate hypothermia, initiate treatment with less invasive rewarming techniques (eg, warmed intravenous [IV] isotonic crystalloid) and progressively add more invasive ones as needed (table 1 and algorithm 1).

  When endovascular warming devices are available, we suggest they be used to rewarm patients with severe accidental hypothermia and a perfusing rhythm (**Grade 2C**). Endovascular rewarming catheters are effective and less invasive and more easily monitored than alternative techniques (eg, peritoneal irrigation with warmed isotonic fluid).

  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 patients with severe hypothermia and a non-perfusing cardiac rhythm, we suggest treatment with extracorporeal membrane oxygenation (ECMO) or cardiopulmonary bypass (CPB) when that option is readily available (**Grade 2C**). (See 'Active internal (core) rewarming' above.)

- **Arrhythmia and cardiac arrest** – 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°C (86°F), at which point renewed attempts at defibrillation and resuscitation with advanced cardiac life support (ACLS) medications are undertaken. The neuroprotective effects of low temperature may allow recovery following prolonged arrest. (See 'Resuscitation' above.)

- **Failure to rewarm** – For patients who fail to rewarm appropriately despite aggressive rewarming measures, we suggest treatment with empiric broad-spectrum antibiotics (**Grade 2C**) and also a single dose of glucocorticoid (eg, dexamethasone 4 mg IV or hydrocortisone 100 mg IV) (**Grade 2C**). Such patients may also need treatment for hypoglycemia, myxedema coma, or other contributing causes. (See 'Failure to rewarm' above.)

- **Post-resuscitation care** – 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.)

- **Frostbite** – 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: Emergency care and prevention".)

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Topic 178 Version 47.0

GRAPHICS

Initial management of hypothermia: Rapid overview of emergency management

General
Definition: core temperature lower than 35°C (95°F) ▪ Mild: 32 to 35°C (90 to 95°F) ▪ Moderate: 28 to 32°C (82 to 90°F) ▪ Severe: below 28°C (82°F)
In patients who are not completely alert and oriented, measure core temperature with a low-reading, digital temperature probe if available. Measure esophageal temperature in patients with tracheal tube or supraglottic airway in place.* Standard oral thermometers do not read below 34°C (93°F).
Consider hypothermia secondary to other conditions (eg, infection, hypoglycemia, adrenal insufficiency, hypothyroidism, overdose, trauma). Older adults are at higher risk. Hypothermia with associated comorbidities or trauma is considered more severe.
Clinical aspects
Physical examination ▪ Vital signs • Mild hypothermia: tachypnea, tachycardia, hyperventilation • Moderate hypothermia: expect bradycardia (tachycardia suggests hypoglycemia, hypovolemia, or overdose), hypotension, hypoventilation • Severe hypothermia: hypotension, cardiovascular collapse ▪ Neurological examination • Mild hypothermia: ataxia, dysarthria, impaired judgement; suspect CNS pathology if patient comatose • Moderate hypothermia: CNS depression • Severe hypothermia: areflexia, coma
Laboratory evaluation
For patients with moderate or severe hypothermia, studies to obtain include: fingerstick glucose, coagulation studies, CBC, basic electrolytes, BUN and creatinine, serum lactate, electrocardiogram, plain chest radiograph. Additional studies may be needed. ▪ Clinical coagulopathy may be present despite normal measured coagulation times ▪ Increased hematocrit may reflect hemoconcentration ▪ May see low bicarbonate, suggesting anion-gap acidosis; if so, obtain venous or arterial blood gas

Electrocardiogram

- Rhythm abnormalities (atrial fibrillation, sinus bradycardia) may be present
- Intervals (PR, QRS, and QTc) may be prolonged
- Osborn J waves are characteristic of hypothermia (but can occur with other conditions)
  - Occur at junction of QRS and ST segments, most prominent in V2 to V5
  - Distortion of the earliest phase of membrane depolarization
  - Computer may misinterpret as ischemic injury pattern

Treatment

Endotracheal intubation may be necessary in obtunded or unconscious patients and those with bronchorrhea

Treat hypotension with warmed, isotonic crystalloid (40 to 42°C) initially, vasopressors (norepinephrine preferred) if necessary

Avoid rough movements and activity, which may induce ventricular fibrillation

Rewarming techniques are based on degree of hypothermia

- Mild hypothermia
  - Remove wet clothing, cover with warm blankets, keep room temperature at approximately 28°C (82°F).
  - Provide active external rewarming, with forced air warming systems if available. Warmed blankets, heating pads, radiant heat sources can also be used. Avoid burning skin.
- Moderate hypothermia
  - Provide active external rewarming as described above.
  - Give warmed IV fluids (40 to 42°C) and warmed humidified oxygen as adjuncts (these are not primary rewarming methods).
  - Beware of afterdrop, a drop in core temperature caused by return of cold blood from the extremities to the core circulation. Rewarm trunk first to minimize afterdrop.
- Severe hypothermia: active external rewarming and active internal rewarming (active core rewarming)
  - Perform interventions for moderate hypothermia.
  - For hemodynamically stable patients, rewarm with endovascular temperature catheter whenever available.
  - For hemodynamically unstable patients, rewarm with ECMO (preferred approach) or CPB if possible. If ECMO or CPB not available, can perform continuous venovenous rewarming, hemodialysis, continuous arteriovenous rewarming.
  - If other interventions unavailable, may perform peritoneal or pleural irrigation with warmed, isotonic saline (40 to 42°C).

Treatment of arrhythmias

- Arrhythmias may persist until patient rewarmed
- Ignore atrial arrhythmias with slow ventricular response

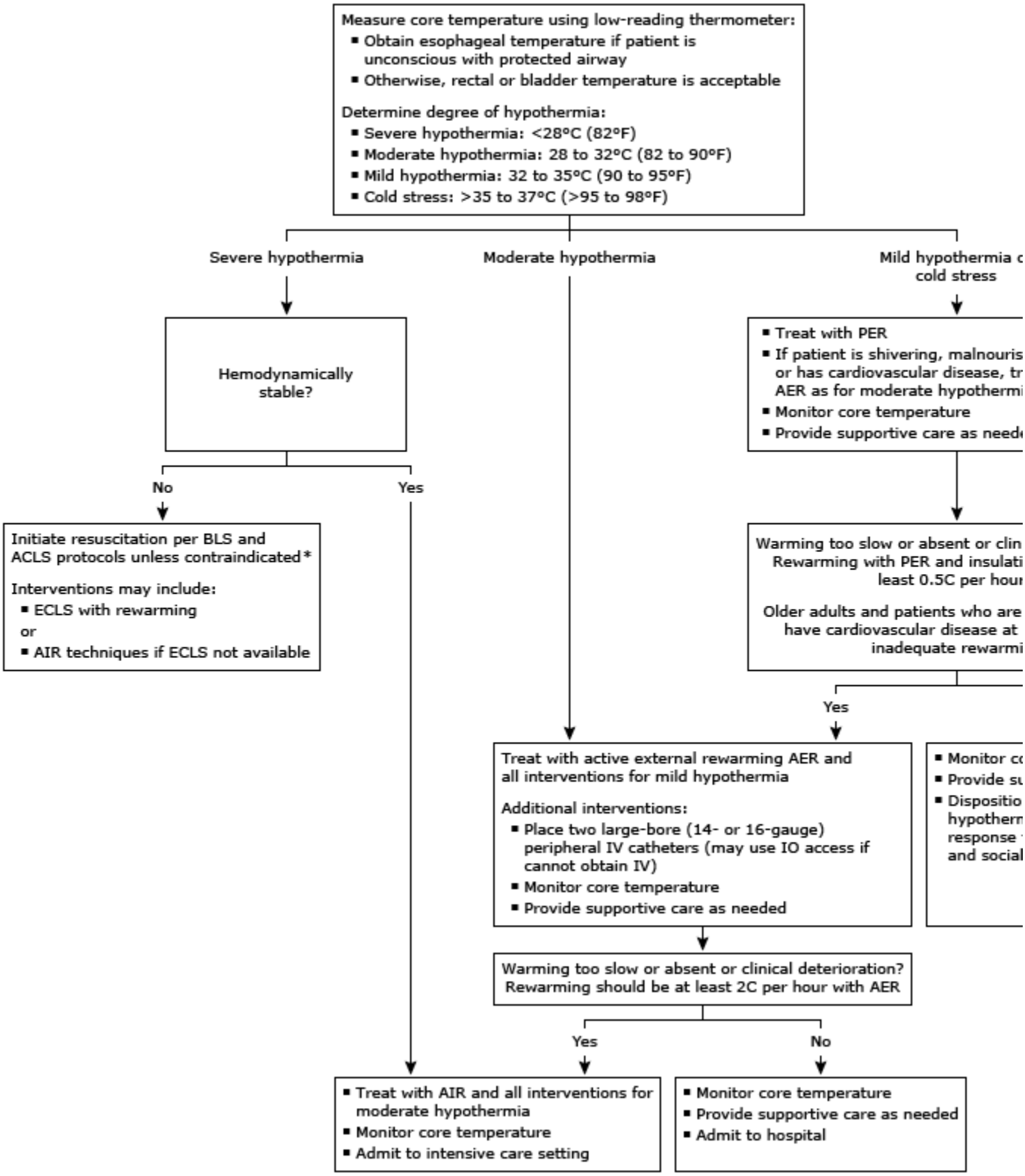
- Ventricular fibrillation is common rhythm
  - Electrical defibrillation may be attempted but is rarely successful until core temperature is above 30°C.
  - Initiate CPR in all patients with cardiac arrest; do not perform chest compressions if an organized rhythm is present on the cardiac monitor.*

CNS: central nervous system; CBC: complete blood count; BUN: blood urea nitrogen; IV: intravenous; CPB: cardiopulmonary bypass; ECMO: extracorporeal membrane oxygenation; CPR: cardiopulmonary resuscitation.

* Refer to UpToDate topic about accidental hypothermia for details about temperature measurement and management of cardiac arrest in severe hypothermia.

Graphic 51697 Version 6.0

Rewarming of adults with accidental hypothermia



PER: passive external rewarming; AER: active external rewarming; BLS: basic life support; ACLS: advanced rewarming; IV: intravenous; IO: intraosseous; ECMO: extracorporeal membrane oxygenation.

* We do not advocate chest compressions in patients with an organized, regular cardiac rhythm. Refer to

¶ Inadequate temperature rise may be due to any number of causes, including sepsis, trauma, intoxicati details.

Graphic 131322 Version 2.0

Hypothermia scale and clinical findings

Stage	Core temperature	Clinical findings
Cold stressed (but not hypothermic)	35 to 37°C (95 to 98.6°F)	Normal mental status with shivering. Functioning normally. Able to care for self.
Mild hypothermia	32 to 35°C (90 to 95°F)	Alert with shivering. Not able to care for self.
Moderate hypothermia	28 to 32°C (82 to 90°F)	Altered level of consciousness. May be conscious or unconscious, with or without shivering.
Severe hypothermia	<28°C (<82°F)	Unconscious. Not shivering.

Graphic 121569 Version 2.0

ICAR-MEDCOM prehospital hypothermia (HT) scale

International Commission for Mountain Emergency Medicine hypothermia scale		
Hypothermia (HT) stage	**Clinical assessment**	**Expected core temperature, C° (F°)***
HT I	Clear consciousness with shivering	35-32 (95-89.6)
HT II	Impaired consciousness without shivering	32-28 (89.6-82.4)
HT III	Unconsciousness	28-24 (82.4-75.2)
HT IV	Apparent death	<24-13.7 (<75.2-56.7)¶
HT V	Death due to irreversible hypothermia	<13.7-9 (56.7-48.2)¶

* Clinical assessment may be used to stage hypothermia when core temperature measurement in the field is not available, but be aware that clinical findings can vary substantially.

¶ The core temperature at which irreversible hypothermia with death occurs is **not** well defined. Please refer to UpToDate topics on hypothermia for discussions regarding prehospital declaration of death and withdrawal of resuscitation.

Graphic 88073 Version 8.0

Revised Swiss System for hypothermia and risk of cardiac arrest

	Stage 1	Stage 2	
Clinical findings*	"Alert" from AVPU	"Verbal" from AVPU	"Pai
Risk of cardiac arrestΔ	Low	Moderate	

AVPU: alert, verbal, painful (ie, responds to pain), unconscious; GCS: Glasgow Coma Scale.

* In the Revised Swiss System, "alert" corresponds to a GCS score of 15, "verbal" corresponds to a GCS sc "painful" and "unconscious" correspond to a GCS score <9. While shivering is not used as a stage-defining temperature is >30°C, a temperature at which hypothermic cardiac arrest is unlikely.

The estimated risk of cardiac arrest is based on accidental hypothermia being the only cause of clinical fi as asphyxia, intoxication, high-altitude cerebral edema, or trauma, the Revised Swiss System may falsely patients who remain "alert" or "verbal" but show signs of hemodynamic or respiratory instability (eg, bra transitional stage with a higher risk of cardiac arrest.

¶ No respiration, no palpable carotid or femoral pulse, no measurable blood pressure. Check for signs of

Δ The transition of colors between stages represents the overlap of patients within groups.

Graphic 134314 Version 1.0

Clinical manifestations of accidental hypothermia

	Mild	Moderate	Severe
Neurologic	Confusion, slurred speech, impaired judgment, amnesia	Lethargy, hallucinations, loss of pupillary reflexes, EEG abnormalities	Loss of cerebrovascular regulation, decline in EEG activity, coma, loss of ocular reflexes
Cardiovascular	Tachycardia, increased cardiac output and systemic vascular resistance	Progressive bradycardia (unresponsive to atropine); decreased cardiac output and BP; atrial and ventricular arrhythmias, J (Osborn) wave on ECG	Decline in BP and cardiac output, ventricular fibrillation (<28°C; 82.4°F) and asystole (<20°C; 68°F)
Respiratory	Tachypnea, bronchorrhea	Hypoventilation, decreased oxygen consumption and CO2 production, loss of cough reflex	Pulmonary edema, apnea
Renal	Bladder atony, cold diuresis	Cold diuresis	Decreased renal perfusion, oliguria
Musculoskeletal	Increased shivering	Decreased shivering (<32°C; 89.6°F) muscle rigidity	Patient may appear dead ("pseudo-rigor mortis")
Metabolic	Increased metabolic rate, hyperglycemia		Decreased metabolic rate, hyper- or hypoglycemia
Hematologic	Increase in hematocrit, decreased platelet count and white blood cell count, bleeding diathesis, DIC		
Gastrointestinal	Ileus, pancreatitis, gastric stress ulcers, hepatic dysfunction		

BP: blood pressure; CO2: carbon dioxide; DIC: disseminated intravascular coagulation; ECG: electrocardiogram; EEG: electroencephalogram.

Graphic 55728 Version 5.0

Expected vital signs according to hypothermia stage

Vital sign*	35°C	32°C	30°C	28°C	26°C	24°C	22°C	20°C
Respiratory rate, breaths/minute (95% CI)	20 (8-32)	18 (6-30)	17 (5-29)	16 (4-28)	15 (3-27)	14 (2-26)	13 (1-25)	12 (0-24)
Heart rate, beats/minute (95% CI)	82 (35-129)	74 (27-121)	69 (22-116)	64 (17-111)	59 (12-106)	54 (7-101)	49 (2-96)	44 (0-91)
Systolic blood pressure, mmHg (95% CI)	139 (86-192)	126 (73-179)	118 (65-171)	109 (52-162)	100 (47-153)	91 (38-144)	83 (30-136)	74 (21-127)
Glasgow Coma Scale (GCS) score (95% CI)	15 (9-15)	13 (7-15)	11 (5-15)	9 (3-15)	8 (3-14)	6 (3-12)	4 (3-10)	3 (3-9)

* Mean vital signs can be used as a guide. Vital signs that fall within the 95% confidence intervals should not necessarily be considered normal for a given core temperature, especially at colder core temperatures.

Graphic 138434 Version 1.0

Laboratory findings in hypothermia

Arterial blood gas	Metabolic acidosis, respiratory alkalosis, or both
Electrolytes	No consistent abnormality
Glucose	Increased, decreased, or no change
White blood cell and platelets counts	Decreased due to splenic sequestration
Hemoglobin, hematocrit	Increased due to hemoconcentration
Lipase	May be increased due to hypothermia-induced pancreatitis
Prothrombin and partial thromboplastin times	Increased in vivo due to inhibition of coagulation cascade, despite normal reported values
Electrocardiogram	Prolongation of PR, QRS, QT intervals
	ST segment elevation
	T wave inversions; Osborn J wave
	Atrial fibrillation or sinus bradycardia
Chest radiograph	Aspiration pneumonia, vascular congestion, pulmonary edema

Redrawn from: Mechem CC. Hypothermia and hyperthermia. In: The Intensive Care Unit Manual, Lanken PN, Manaker S, Hanson CW III (Eds), WB Saunders, Philadelphia 2000. p.627.

Graphic 67294 Version 4.0

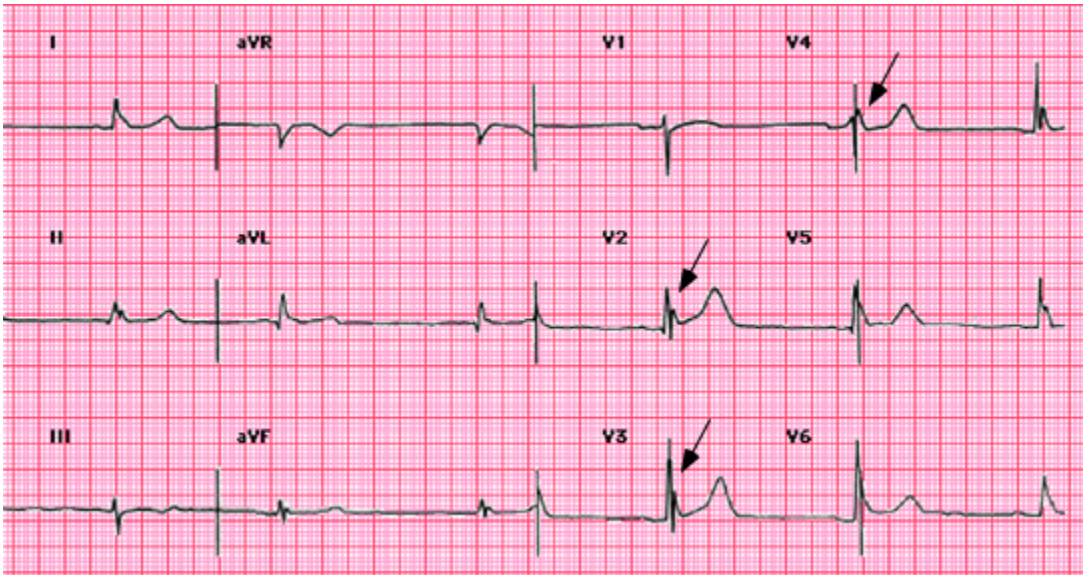
The effect of temperature on blood gas measurements

Temperature		pH	PCO_2	PO_2
ºC	ºF			
20	68	7.65	19	27
30	86	7.50	30	51
35	95	7.43	37	70
36	97	7.41	38	75
37	98	7.40	40	80
38	100	7.39	42	85
40	104	7.36	45	97

Adapted from: Shapiro, Peruzzi, Kozelowski-Templin: Clinical Application of Blood Gases, ed 5. St. Louis, Mosby-Year Book, 1994, p. 128.

Graphic 59630 Version 2.0

Electrocardiogram in hypothermia

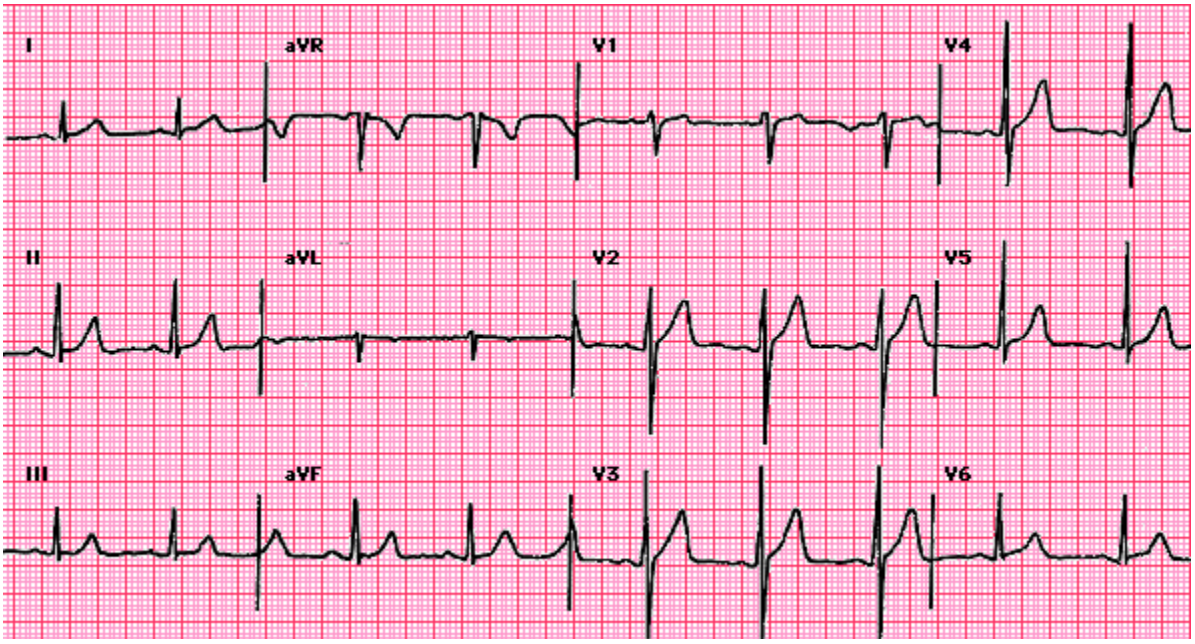


The ECG reveals marked sinus bradycardia (about 40 beats/minute) with first-degree atrioventricular block (PR interval = 0.23 seconds). The slow heart rate in this patient is due to hypothermia (90°F, 32.2°C), which also produces prominent convex deflections at the J point (junction of QRS and ST segments) that are best seen in the precordial leads. The J waves or Osborn waves (arrows) are characteristic of severe hypothermia and resolve with rewarming.

Courtesy of Ary Goldberger, MD.

Graphic 64520 Version 4.0

Normal ECG

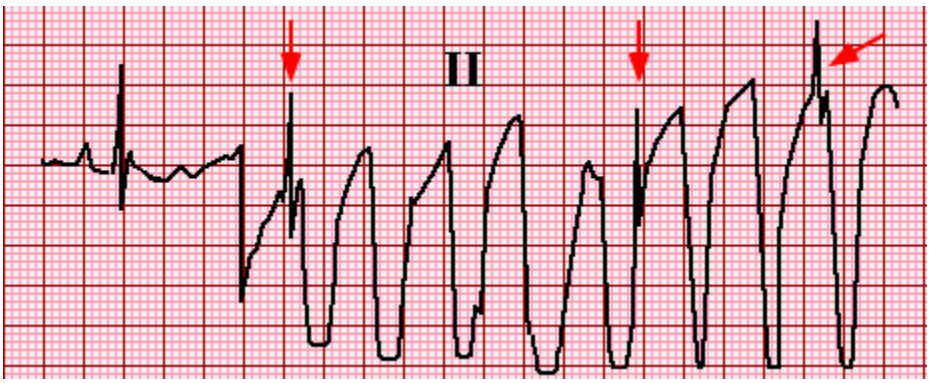


Normal electrocardiogram showing normal sinus rhythm at a rate of 75 beats/minute, a PR interval of 0.14 seconds, a QRS interval of 0.10 seconds, and a QRS axis of approximately 75°.

Courtesy of Ary Goldberger, MD.

Graphic 76183 Version 4.0

Tremor artifact



This dramatic example of tremor artifact demonstrates complexes that simulate a run of ventricular tachycardia. However, QRS complexes (arrows) can clearly be seen marching through the rhythm strip.

Graphic 62575 Version 3.0

Causes of hypothermia

Mechanism	Clinical disorder
Increased heat loss	Environmental exposure
	Induced vasodilation
	Drugs
	Alcohol
	Toxins
	Skin disorders
	Burns
	Psoriasis
	Exfoliative dermatitis
	Iatrogenic
	Cold infusion
	Emergent deliveries
	Cardiopulmonary bypass
	Continuous renal replacement therapy
Decreased heat production	Endocrinologic disease
	Hypopituitarism
	Hypoadrenalism
	Hypothyroidism
	Insufficient fuel
	Hypoglycemia
	Malnutrition
	Neuromuscular inefficiency
	Extremes of age

Mechanism	Clinical disorder
Impaired regulation	Peripheral
	Spinal-cord transection
	Neuropathies
	Diabetes mellitus
	Central
	Cerebrovascular accident
	Subarachnoid hemorrhage
	Parkinsonism
	Hypothalamic dysfunction
	Multiple sclerosis
	Anorexia nervosa
	Drugs: Intoxicants, anxiolytics, antidepressants, antimanic agents, antipsychotics, opioids, oral antihyperglycemics, beta blockers
Other	Sepsis
	Pancreatitis
	Carcinomatosis
	Uremia
	Vascular insufficiency
	Trauma

	Impaired shivering
	Inactivity

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Graphic 58684 Version 2.0

Accidental hypothermia prognostic factors in adults

Asphyxia (drowning, avalanche burial)
Unwitnessed cardiac arrest
Asystole
Hyperkalemia
Elevated serum lactate
Hemodynamic instability
Age >75 years
Kidney injury (elevated creatinine)

While evidence is limited, the factors listed above are associated with worse outcomes following hypothermia due to environmental exposure.

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* Evidence about accidental hypothermia is limited. These factors are drawn primarily from retrospective, observational studies. They are not necessarily independent risk factors. There are many potential confounders.

Graphic 130815 Version 1.0

Contributor Disclosures

Ken Zafren, MD, FAAEM, FACEP, FAWM No relevant financial relationship(s) with ineligible companies to disclose. **C Crawford Mechem, MD, FACEP** No relevant financial relationship(s) with ineligible companies to disclose. **Daniel F Danzl, MD** No relevant financial relationship(s) with ineligible companies to disclose. **Michael Ganetsky, MD** No relevant financial relationship(s) with ineligible companies to disclose.

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