Emergency Management of COLD-INDUCED INJURIES

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Overexposure to cold can result in injuries ranging from the easily correctable to the life-threatening. Rewarming, resuscitation, and possible referral to a burn specialist may all be required, depending on the degree and type of injury. The authors consider both systemic and localized injuries and their treatment, as well as the pathophysiology and classification of such injuries.

Injuries due to exposure to cold or damp can occur at any time, and patients with such injuries can present to any ED. Cold-induced injuries are not exclusive to persons in geographic areas with a cold climate. Each year in the United States, approximately 650 to 1,100 deaths are caused by primary accidental hypothermia, in which direct exposure to cold results in death, or by secondary hypothermia complicating another condition.[1] According to the CDC, of those who died from hypothermia during the years 1999 to 2002, 67% were men and 49% were at least 65 years of age. Although approximately 50% of hypothermia-related deaths occur in extremely cold weather, hypothermia can occur in any environment. With proper precautions, the majority of deaths and injuries from hypothermia are preventable.[1]

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It is important for emergency physicians to be skilled in recognizing and treating the various types of injuries related to overexposure to the cold. Cold-induced injury can be broadly classified as systemic or localized. These categories are not mutually exclusive, and injuries of both kinds may occur in the same patient.

SYSTEMIC INJURY (ACCIDENTAL HYPOTHERMIA)

Systemic cold-induced injury, or accidental hypothermia, is defined as a core body temperature below 35°C. The effects of hypothermia occur along a continuum ranging from mild and easily correctable to severe, life-threatening multiorgan failure.

Pathophysiology

Exposure to cold initially causes compensatory effects in the cardiovascular, muscular, and respiratory systems. These effects are mediated by the preoptic hypothalamus, which is

the temperature control center of the brain. Cutaneous cold receptors communicate with the preoptic hypothalamus, which induces an increase in circulating catecholamines, shivering, and the sensation of being cold.[2] The heart rate and cardiac output increase, and the peripheral cutaneous vasculature constricts, diverting blood to the central circulation. In addition, the respiratory rate increases to support the increased metabolic demand. Shivering begins to increase the basal metabolic rate and generate heat. Additionally, respiratory secretions increase as cold, dry air and rapid breathing quickly dry the mucous membranes.

These compensatory mechanisms have only a limited ability to maintain core temperature in prolonged cold exposure; overwhelming environmental conditions and exhaustion of the individual's energy stores will cause them to fail. In the absence of behavioral modification to seek warmth, clothing, or shelter, the core temperature continues to decrease. As the temperature decreases, the initial compensatory effects are suppressed due to direct dysfunction from the cold.

Special Populations—Those at risk for hypothermia include individuals who cannot compensate for heat loss and those who fail to alter their behavior to compensate for cooler ambient temperatures. The condition is most likely to occur in the very young, the very old, persons who are intoxicated, those with a physical or intellectual disability, and outdoor enthusiasts.[3] It is noteworthy that having more than one risk factor can dramatically increase an individual's risk for hypothermia.

Leaner persons cool more quickly, since they have less insulation in the form of fat. Conversely, a patient with very low muscle mass has a decreased ability to generate heat through muscle activity. The very young radiate heat readily due to a large ratio of surface area to mass. In addition, this population is entirely dependent on caregivers for clothing and heat. The elderly have decreased muscle mass for shivering and, like the very young, often lack the ability to seek warm clothing or shelter. Homeless persons frequently have inadequate clothing and can be caught unprepared by sudden temperature changes. In outdoor enthusiasts, even those who are well prepared, prolonged exposure can overwhelm heat generation capacity and diminish the protective benefit of clothing, especially in the event of injury or becoming lost.[2,3]

Effects of Alcohol and Sedatives—Alcohol and sedatives can inhibit behavioral responses to cold. Chemical impairment of heat generation occurs with use of these substances, as their anesthetic properties can compromise shivering. In particular, meperidine lowers the shivering threshold, a property that makes it unique among the opiates. Patients taking meperidine may not begin shivering until they have reached a body temperature too low for this compensatory mechanism to be effective. The mechanism by which meperidine lowers the shivering threshold is poorly understood but may involve effects on the κ-receptor. Central α_2-adrenergic agonists such as clonidine have been shown to lower the shivering threshold as well, and may also increase risk of hypothermia.[3,4]

Classification

Hypothermia is classified as mild, moderate, or severe, based on core temperature. Each level of hypothermia produces classic signs and symptoms (Table).[2,5-7]

Mild Hypothermia—Mild hypothermia (core temperature, 32°C to 35°C) is characterized by the signs and symptoms of attempted compensation, with early indications of compensation failure. Therefore, the mildly hypothermic patient will have an increased heart rate, blood pressure, and respiratory rate, as well as peripheral vasoconstriction. These changes lead to respiratory alkalosis and "cold diuresis." Increased central blood flow increases renal blood flow and suppresses antidiuretic hormone (ADH) release, causing production of dilute urine.[2,5] Behavioral manifestations in the otherwise-nonintoxicated patient may include confusion, ataxia, and apathy.

Moderate Hypothermia—As temperature decreases, the patient develops increasing signs of moderate hypothermia (core temperature, 28°C to 32°C). At these temperatures, the mechanisms of heat production become overwhelmed, and the body demonstrates the effects of cooling. Bradycardia and decreased cardiac output produce hypotension. The myocardium becomes irritable, increasing the incidence of arrhythmia. Common perfusing arrhythmias may include atrial fibrillation, but the patient is also at risk for ventricular fibrillation and ventricular tachycardia.[7] During this stage, an ECG will reflect these derangements with prolonged PR, QRS, and QT intervals, AV block, premature beats of atrial or ventricular origin, and, classically, a positive deflection between the QRS complex and ST segment known as the J wave, or Osborn wave (Figure). This wave, whose origin is poorly understood, appears in 80% to 86% of moder-

TABLE. Features of Hypothermia by Organ/Body System

	Level of Hypothermia (Core Temperature)		
System	**Mild (32°C to 35°C)**	**Moderate (28°C to 32°C)**	**Severe (<28°C)**
Cardiovascular	• Elevated heart rate, cardiac output, BP	• Decreased heart rate, cardiac output, BP • Prolonged PR, QRS, QT intervals • J waves • AF, VF	• Pulseless electrical activity • AF, VF • Heart block • Asystole (at <20°C)
Respiratory	• Elevated RR • Respiratory alkalosis • Bronchorrhea	• Hypoventilation • Respiratory acidosis • Hypoxemia • Decreased airway protection and coughing • Suppressed ciliary function	• Apnea (at <24°C) • Pulmonary edema • ARDS
Neurologic	• Ataxia • Apathy • Confusion • Dysarthria	• Loss of pupillary reflexes • Hallucinations, agitation • Progressive decrease in level of consciousness	• Areflexia • Coma • Decreased cerebral blood flow and EEG activity (no activity at <20°C)
Renal	• Cold diuresis	• Cold diuresis • Decreased GFR • Metabolic acidosis	• Oliguria
Metabolic	• Shivering • Decreased insulin release • Increased catecholamines • Hyperglycemia	• Decreased metabolic rate and shivering • Hyperkalemia • Hyperglycemia • Lactic acidosis	• Shivering stops • Acidosis worsens • Metabolic rate approaches 20% of BMR

BP = blood pressure; AF = atrial fibrillation; VF = ventricular fibrillation; RR = respiratory rate; ARDS = acute respiratory distress syndrome; EEG = electroencephalographic; GFR = glomerular filtration rate; BMR = basal metabolic rate.

Data from Kempainen and Brunette[2]; Danzl and Pozos[5]; Biem et al[6]; Mattu et al.[7]

ately to severely hypothermic patients and tends to resolve as the patient warms up.[7] Mattu et al note that J-point elevation can be confused with ST elevation and can appear to be consistent with an acute myocardial infarction.[7] Therefore, in selected patients with ST elevation, it may be useful to obtain a rectal temperature.

The respiratory system is also affected in moderate hypothermia, with slowing of the respiratory rate and conversion of the respiratory alkalosis to respiratory acidosis. Ciliary motion is impaired, as is cough reflex, resulting in poor clearance of secretions and possible aspiration. Cold diuresis continues, enhanced by failure of the cold pituitary gland to secrete ADH. Fluid loss can make the patient hypovolemic, exacerbating hypotension. Shivering lessens due to loss of central stimulus, and the patient becomes increasingly stuporous and eventually loses consciousness. The pupils dilate, and light reflexes are lost.[1,2]

Severe Hypothermia—In this final stage of hypothermia (core temperature <28°C), ventricular fibrillation is possible, or the heart may simply slow to asystole. Apnea and pulmonary edema develop, which may progress to acute respiratory distress syndrome, and cold diuresis gives way to oliguria as decreased cardiac output reduces the glomerular filtration rate. Neurologic manifestations include failed reflexes, cessation of shivering, loss of pain responses, and loss of spontaneous movement. Electroencephalography shows no brain activity at temperatures around 20°C. At this point, a patient may appear by most metrics to be dead.

Treatment

Rewarming Methods—The mainstay of treatment and resuscitation of the hypothermia patient is rewarming. Many of the physiologic insults that result from low core temperature resolve with correction. There are four broad categories of warming strategies: external rewarming, internal rewarming, extracorporeal rewarming, and cardiopulmonary bypass. Each can be used alone or in conjunction with the others, based on the severity of the hypothermia and resources available.

First, wet clothing should be removed and patients should be covered with warm blankets or reflective emergency thermal barriers. This is known as *passive external rewarming*. The scalp should also be covered to minimize heat loss, with the face remaining exposed to maintain access to the airway.[3,5] With appropriate insulation, natural shivering will rewarm patients at a rate of approximately 0.5°C/h.[2] The effectiveness of this method depends on the patient's energy stores and ability to continue to shiver. Consumption of warm food and drink can help the patient to maintain glucose reserves during this process.

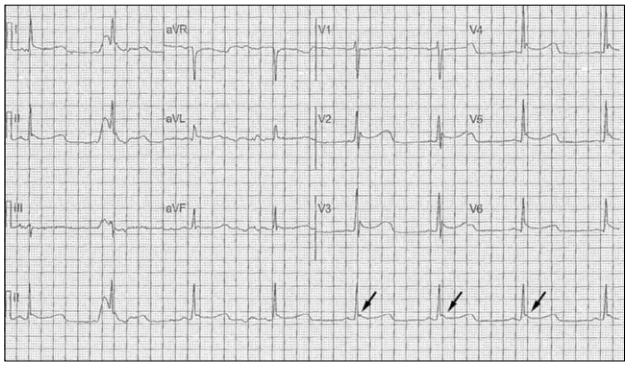

FIGURE. ECG exhibiting J waves (arrows).

The next level of intervention is *active external rewarming*. This involves using a heat source to add heat back to the body. In the prehospital or wilderness environment, hot water bottles, fire, space heaters, or heating pads may be used. In the ED, the most common method of active external rewarming is the forced-air blanket. Forced-air rewarming can achieve warming rates of 1°C to 2.5°C/h.[8,9]

Both the passive and active external rewarming methods can cause paradoxic cooling of the core or systemic acidosis when previously poorly circulated, comparatively cold blood from the extremities flows into the central circulation. For this reason, it is important to first apply interventions to the core (rather than the extremities), to allow the patient to heat "from the inside out," or proximally to distally.[2]

The *active internal rewarming* approaches involve direct warming of the patient's blood or body cavities. The two least invasive methods are warmed, humidified oxygen and warmed IV fluids, both warmed to approximately 40°C to 42°C. Warmed oxygen can improve core temperature by 0.5°C to 1.2°C/h and has the added benefit of reducing evaporative loss.[5] Warmed IV fluids can achieve warming rates of 1°C to 2°C/h, depending on rate of infusion.[10] To minimize cooling in transit, short IV lines are preferable. Research has examined the possibility of using fluids warmed as high as 65°C: While at least one study on beagles[11] shows this to be safe and effective when applied to the central circulation, safety data in humans are lacking. With fluids at such warm temperatures, there is the concern of endothelial heat damage, especially in

the peripheral circulation; thus, this approach cannot be recommended at this time.[12]

Increasingly invasive methods for rewarming in severe hypothermia include peritoneal and thoracic lavage with warm saline. In the case of hypothermic cardiac arrest, thoracotomy followed by open thoracic lavage in conjunction with direct cardiac massage has been shown to be effective, both as a lifesaving measure and a bridge to cardiopulmonary bypass. Warming rates of 8°C/h have been reported with this method.[13] Other approaches reported in the medical literature include peritoneal lavage and closed thoracic lavage, in which warm crystalloid is infused through a thoracostomy tube at the anterior axillary line in the second or third intercostal space; it then exits through a second tube of more traditional placement.

FAST TRACK

Hypothermic patients are prone to ventricular arrhythmia; therefore, care should be taken to move them gently, avoiding sudden jarring movements that may destabilize the irritated heart.

The *extracorporeal rewarming* approaches involve the most intensive methods available, but they offer the benefit of rapid rewarming that can be consistently maintained. Three methods of variable efficacy and availability are currently used. The easiest to initiate is hemodialysis, which warms the patient at rates of 2°C to 3°C/h. Placement of a single large-bore dialysis catheter is the only prerequisite, and emergent dialysis is available to most EDs.[9] Hemodialysis requires that the patient have a perfusing blood pressure. This requirement also exists for continuous arteriovenous rewarming and venovenous rewarming. In these two methods, the physician cannulates the femoral vein on one side and the femoral artery (or vein) on the other side. The patient's own blood pressure moves blood through a warming device and then back into circulation. This method can raise the core temperature by 3°C to 4°C/h.[9]

The fastest method of rewarming the very cold patient, who may also be in cardiac arrest and have no blood pressure, is *cardiopulmonary bypass*, which achieves warming rates from 7°C to 10°C/h, although rates up to 20°C/h have been cited in some sources.[13] With this method, blood is warmed, oxygenated, and circulated through the body while the heart can warm up to the point that it may produce adequate cardiac output. Disadvantages of cardiopulmonary bypass include its limited availability in some locations, the need for a perfusionist, and the need for anticoagulation.[2]

Resuscitation—The resuscitation of the hypothermic individual is similar to that of the normothermic patient, with a few notable differences. Hypothermic patients are prone to ventricular arrhythmia; therefore, care should be taken to move them gently, avoiding sudden jarring movements that may destabilize the irritated heart.[2] Patients who are areflexic should be gently intubated. It is not necessary to paralyze the hypothermia victim who requires intubation, as most agents do not work at temperatures below 30°C, and the patient's ability to metabolize them is severely limited as well.[2] ACLS protocols must be modified due to temperature. In the patient in pulseless electrical activity (PEA) or asystole with a core temperature of less than 30°C, one round of drugs may be given, but greater effort should be invested in rewarming the patient to a temperature above 30°C, as the patient's condition may improve on its own when the patient is warm.[14] Likewise, patients with severe hypothermia who are in ventricular fibrillation should receive one shock, but further intervention should be limited to CPR until the patient is warm. Additionally, CPR is indicated for asystole, but not PEA, as PEA in the very cold patient may represent a perfusing rhythm that will improve with warmth.

It is important to remember that substantial volume loss occurs in most patients with hypothermia; thus, volume resuscitation is often required.[2,14] Arterial and central venous catheterization will be helpful for monitoring volume status and blood pressure, as the cold heart has poor contractibility. Arterial blood gas and electrolyte panels can be useful in identifying correctable derangements—notably, acidosis, hyperkalemia, and hypercarbia. Coagulation panels are not useful, as this testing is performed at 37°C, and hypothermic patients are often coagulopathic until they are rewarmed.[1,3] Platelet count, fibrinogen values, and D-dimer assay are more useful, as they can indicate disseminated intravascular coagulation. The primary focus of resuscitation should be supportive care until the patient is rewarmed to a core temperature greater than 30°C. After that, more aggressive ACLS-type interventions can be instituted, as needed.[2,14]

LOCALIZED COLD INJURIES

Localized cold injuries represent a spectrum of injury from superficial to severe tissue destruction. The extremities are the most susceptible, but these injuries can be found anywhere on the body. Risk factors for localized injury are similar to those for accidental hypothermia (including intoxication, physical or intellectual disability, and participation in outdoor sports); however, they also include occupational hazards such as working in a cold environment (eg, meat processing) and direct contact with frozen objects or surfaces. Diabetes and cigarette smoking increase the risk for localized cold-induced injury due to vascular disease and neuropathy. The risk for localized cold injuries, unlike that for accidental hypothermia, is lower in the very young and very old because these groups are less likely than the rest of the population to have a single portion of the body exposed to freezing conditions.[15] Injuries related to nonfreezing temperatures include trench foot and chilblains, while injuries from exposure to freezing temperatures span from frostnip (a condition of numbness, pain, and pallor in cold-exposed skin that resolves easily without significant intervention) to frostbite (which involves destruction of localized tissue).

Exposure to cold causes peripheral vasoconstriction, a mechanism that helps preserve core temperature by diverting blood to the central circulation. This causes localized cooling and tissue damage, with the degree of cooling and damage dependent on the temperature and duration of exposure.

Chilblains and Trench Foot

When the temperature remains above freezing, chilblains or trench foot may develop. Chilblains, or pernio, are characterized by localized irritation, pain, itching, and redness in patches with repeated exposure to cold, dry conditions. Plaques, vesicles, and bullae may develop. Treatment involves rewarming the injured area and avoiding repeated exposure to damp and cold. Following chilblains, the patient may experience heightened sensitivity to cold in the area originally affected. Repeat exposure may lead to chronic lesions.

Coined during World War I, the term *trench foot* refers to a condition that affects the hands or feet and is brought on by prolonged exposure to wet conditions at temperatures just above freezing. The tissue initially becomes cold, pale, and anesthetic. Over the next several days, the tissue becomes hyperemic, painful, and edematous and may develop bullae or ulcers. This may lead to localized infection or gangrene. Treatment, as for chilblain, is removal from the damaging environment, elevation, and exposure to warm, dry air. In neither condition is it advisable to soak, rub, or rapidly rewarm the injured area.

Frostnip

At freezing temperatures, frostnip can occur, which is a superficial injury characterized by pain and pallor followed by anesthesia of the affected part. The affected area is cold to the touch. At this point, the condition remains reversible, and rewarming of the area results in full recovery.[3] If the condition is allowed to progress, ice crystals begin to form in the tissue. Initially, the crystals are extracellular, causing a shift of fluid out of the cell. Cells become dehydrated and supersaturated with electrolyte solute, which triggers cell death.[16] Once the ambient temperature in the tissue has fallen far enough, ice crystals begin to form inside the cells, lysing the cell membranes.

FAST TRACK

For frostbite, NSAIDs have been shown to reduce the initial cascade of inflammatory and coagulatory mediators, potentially reducing the extent of injury.

Frostbite

When a single part of the body is subjected to the drastic drop in temperature seen with frostbite, the initial response is vasoconstriction. This is followed by vasodilation, which has the effect of partially thawing the frozen area. Thawing allows inflammatory mediators to develop in response to the tissue injury and the development of microemboli. This injury leads to frostbite.

Superficial frostbite occurs when the frozen tissue is limited to the skin and subcutaneous layers. Deep frostbite is characterized by penetration to muscle, tendon, and bone. In both conditions, the extremity presents with pallor, stiffness, and anesthesia. Adjacent areas may sting or burn. Often, bullae develop with rewarming. Superficial and deep frostbite can be distinguished at this point: Superficial injuries develop blisters filled with clear fluid, whereas in deep injuries, the blister contains cloudy or hemorrhagic fluid.[15,16]

Treatment—The initial appearance of frostbite is very similar in both the superficial and deep forms, with the true extent of viable versus nonviable tissue sometimes taking weeks to manifest. For this reason, the initial management of frostbite is a single approach.[15] In the prehospital environment, any wet, nonadherent clothing should be removed from the area, and the area should be splinted or dressed to avoid mechanical trauma. Rewarming should be avoided during transport if there is any risk of refreezing. Once the patient is in the ED, the area should be aggressively rewarmed by soaking in warm water (40°C to 42°C) for 15 to 30 minutes or until the area looks and feels thawed. Once the area is rewarmed, any blisters containing clear fluid should be gently debrided, while hemorrhagic blisters should be left undisturbed.[16] Elevation of the affected extremity helps to reduce edema.

The patient may require significant analgesia. An approach to pain management similar to that used with burns may be adopted. NSAIDs have been shown to reduce the initial cascade of inflammatory and coagulatory mediators, potentially reducing the extent of injury.[16] Similarly, topical aloe vera in conjunction with NSAIDs has been shown to reduce tissue loss through inhibition of thromboxane. Tetanus vaccination should be updated as necessary. Frostbite patients will require close follow-up with a burn specialist, and those with extensive injury or signs of deep injury should be admitted for daily hydrotherapy and debridement.

Once an extremity is rewarmed and initially treated, the extent of damage can be surmised. Magnetic resonance imaging and magnetic resonance angiography 2 to 3 weeks after initial injury can assist in demarcating the border between recovering and ischemic tissue.[15,16] However, surgical debridement or amputation is usually withheld during the first 1 to 3 months, unless the injured area becomes gangrenous. It is important that frostbite be managed as early as possible. Delay in management over 24 hours is associated with surgical intervention 85% of the time.[3]

Several adjunct therapies have been proposed, including anticoagulation, hyperbaric oxygen, vasodilators, and thrombolytics, all intended to reduce post-thaw tissue ischemia.[17] Two small studies have addressed the use of adjunctive therapies to limit frostbite tissue loss. One study suggested that thrombolysis may be beneficial in the short term,[18] and a new French study of 47 patients showed a significant benefit with prostacyclin treatment.[19] These therapies may prove to be new avenues for frostbite treatment.

CONCLUSION

Cold-induced injuries can result in serious complications if they are not treated properly. The hypothermic patient must be resuscitated appropriately, with due attention paid to airway, breathing, and circulation, with the caveat that medications and electricity ordinarily associated with ACLS may not work if the temperature of the patient's heart is below 30°C. Localized cold injuries have varied presentations that often require long-term follow-up with specialized care and early appropriate intervention to reduce the extent of injury. When these conditions are recognized early, initiation of appropriate management can save limbs and lives. **EM**

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