In the United States, frostbite is often a disease of the indigent, the intoxicated, the mentally ill, and winter outdoor recreation enthusiasts. For both frostbite and hypothermia the literature consists of primarily case reports, case series, and reviews.

**EPIDEMIOLOGY**

Accidental hypothermia is generally manifested as progression from an initial state of catecholamine release and stimulation (mild hypothermia) to one marked by a predictable slowing of metabolism and all critical body functions to the extent that patients may appear dead. The profound physiologic changes and metabolic slowing all reverse with rapid rewarming, and reports of remarkable neurologically intact survival despite hours of pulselessness and resuscitation are not infrequent. This has given rise to the adage "No one is dead until they are warm and dead." Not surprisingly, there is little strong scientific evidence for treatment recommendations because trials cannot be conducted. The medical literature is composed mostly of animal experiments, case reports and series, and retrospective reviews. Still, rational approaches can be inferred from the extant literature for this uncommon but serious problem (Table 131.1).

Frostbite is a freezing injury to soft tissues secondary to cold exposure and results in loss of circulation to and therefore viability of the affected area. The extremities, nose, ears, and male genitalia are the most commonly affected areas.

**HYPOTHERMIA**

**PATHOPHYSIOLOGY**

**MECHANISMS OF HEAT LOSS**

The mechanisms of heat loss are as follows:

- **Radiation** of heat occurs when the ambient temperature is less than body temperature and heat is lost directly to the environment via electromagnetic radiation.
- **Conduction** is heat transfer from one (warmer) solid to another (cooler) when they are in contact.
• **Convection** is loss of heat from a surface to a (usually moving) gas or fluid, typically air or water. It can be considered an adjunct to conduction.
• **Evaporation** causes heat loss through the energy required to vaporize water (i.e., sweat).

As a person cools, a fairly predictable procession of pathophysiologic changes occurs, as seen in **Table 131.2** and **Figure 131.1**.

### PRESENTING SIGNS AND SYMPTOMS

Patients with mild hypothermia are awake, occasionally drowsy, uncomfortable, and shivering. They simply need insulation (blanket), dry clothes, and food. They will recover completely and can be discharged when normothermic and feeling better.

Patients with moderate hypothermia are generally confused and lethargic, often have slurred speech, and are typically not shivering. They require more energetic rewarming measures, including heated blankets, resistive and hot air blankets (Bair Hugger), and close monitoring, including core temperature. Though strictly considered active internal rewarming, the use of heated, humidified oxygen and warmed intravenous (IV) fluids is reasonable in this situation. Patients whose hypothermia responds to these measures may be discharged when normothermic, awake, alert, and ambulatory. Patients with severe hypothermia require prompt intervention, close monitoring, and potentially aggressive, invasive rewarming therapies.

It is of the utmost importance to learn the circumstances that led to the patient becoming hypothermic. The possibility of a verify drug overdose, trauma, infection, drowning, or decompensated comorbid conditions—to name but a few examples—must be considered, sought, and treated along with the hypothermia.

The critical element of the diagnosis of hypothermia is accurate measurement of core temperature. Several methods exist, all of which have potential drawbacks (**Table 131.3**). Laboratory and physiologic changes are correlated with the temperature. For example, a normal hematocrit value in a

### Table 131.1 Definitions of Hypothermia

<table>
<thead>
<tr>
<th>LEVEL OF HYPOTHERMIA</th>
<th>CORE TEMPERATURE (°C)</th>
</tr>
</thead>
<tbody>
<tr>
<td>General</td>
<td>&gt;35</td>
</tr>
<tr>
<td>Mild</td>
<td>32-35</td>
</tr>
<tr>
<td>Moderate</td>
<td>28-32</td>
</tr>
<tr>
<td>Severe</td>
<td>&lt;28</td>
</tr>
</tbody>
</table>

### Table 131.2 Pathologic Changes Seen with Hypothermia

<table>
<thead>
<tr>
<th>SYSTEM</th>
<th>MILD</th>
<th>MODERATE</th>
<th>SEVERE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cardiovascular</td>
<td>↑ HR</td>
<td>Progressive ↓ HR, ↓ CO, ↑ PVR</td>
<td>Profound ↓ HR, ↓ CO, ↓ PVR</td>
</tr>
<tr>
<td></td>
<td>↑ CO</td>
<td>Osborn waves possible on electrocardiogram</td>
<td>Ectopy (especially atrial fibrillation)</td>
</tr>
<tr>
<td></td>
<td>↑ PVR</td>
<td></td>
<td>Ventricular fibrillation</td>
</tr>
<tr>
<td></td>
<td>Drowsiness, shivering</td>
<td>Confusion</td>
<td>Asystole</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Dysarthria</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Muscular rigidity</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Ectopy (especially atrial fibrillation)</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Venticular fibrillation</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Coma</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Muscular rigidity</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Pupils fixed and dilated</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Electroencephalogram flat at −20° C</td>
<td></td>
</tr>
<tr>
<td>Central nervous system</td>
<td>In general, hematocrit ↑≈2% for every 1° C  ↓ in temperature</td>
<td>Continuum</td>
<td>Coagulopathy</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Coagulopathy</td>
<td>Thrombocytopenia</td>
</tr>
<tr>
<td>Hematologic</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Renal</td>
<td>Diuresis secondary to ↑ PVR with ↑ renal blood flow</td>
<td>Progressive loss of distal tubular resorption</td>
<td>Continued diuresis</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Resistance to antidiuretic hormone</td>
<td>Limitation of clearance of electrolytes and glucose</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Acute renal failure</td>
</tr>
<tr>
<td>Respiratory</td>
<td>Tachypnea</td>
<td>Progressive bradypnea</td>
<td>Profound bradypnea</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Loss of protective reflexes</td>
<td>Apnea</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Bronchorrhea</td>
<td>Pulmonary edema (rare)</td>
</tr>
<tr>
<td>Gastrointestinal</td>
<td>Clinically silent</td>
<td>Progressive hepatic impairment</td>
<td>Decreased lactate clearance and detoxification and metabolism of drugs Pancreatitis in 20-30% of cases</td>
</tr>
<tr>
<td>Acid-base</td>
<td>Can be either alkalotic or acidotic</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Endocrine</td>
<td>Patient usually hyperglycemic</td>
<td></td>
<td>Preexisting hypothyroidism or hypoadrenalism can impair rewarming</td>
</tr>
</tbody>
</table>

CO, Cardiac output; HR, heart rate; PVR, peripheral vascular resistance.
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severely hypothermic patient should prompt concern for hemorrhage because the hematocrit should rise in a predictable fashion with ever-lowering temperature. Alternatively, arterial blood gas values should be interpreted as though the patient is normothermic (the alpha-stat method) and not corrected for the actual core temperature (the pH-stat method). Evaluation for infection, metabolic derangement, and cardiac, neurologic, renal, and other organ system abnormalities is important because comorbid conditions are common as a cause, a consequence, or coincidence of hypothermia.

TREATMENT

MILD HYPOTHERMIA

Patients with mild hypothermia may be treated with passive external rewarming. Such treatment, which consists of the use of blankets, dry clothes, ambient warmth, oral hydration, and energy substrate (food), produces a rewarming rate of about 0.5° C to 1° C per hour. The approach uses the patient’s inherent ability to keep warm, primarily through shivering, and insulation.

MODERATE HYPOTHERMIA

Patients with moderate hypothermia should undergo active external rewarming. Heat is actively supplied to the body via electric blankets, forced-air blankets, and space heaters. This method achieves rewarming rates of about 1° C to 2° C per hour.

SEVERE HYPOTHERMIA

Active internal rewarming is needed for patients with moderate and severe hypothermia. Actions are directed toward heating the core preferentially over the periphery. This goal is accomplished via methods of variable invasiveness and complexity, as follows:

- Heated, humidified oxygen (40° C to 45° C) administered via face mask or endotracheal tube; it primarily serves to prevent additional heat loss.
- Administration of heated IV fluids (40° C to 42° C) adds negligible heat overall but does aid in preventing further heat loss.
- Gastric or bladder lavage (or both) via nasogastric tube and Foley catheter is relatively easily accomplished; however, the small volume of these cavities limits the effectiveness of these modalities.
- Peritoneal lavage with prepackaged dialysate or standard crystalloid fluids heated to about 45° C. This method is quicker if two catheters, one for afferent flow and one for effluent flow, are used. Rewarming rates average 2° C to 3° C per hour.3,4
- Closed thoracic cavity lavage (pleural lavage) of the left hemithorax is done with two 36-French thoracostomy tubes and isotonic fluid heated to about 42° C. Large volumes are required. The afferent tube is placed in the second intercostal space (ICS) in the midclavicular line. The efferent tube is placed in the usual location, the fourth or fifth ICS in the

Table 131.3  Methods of Measuring Core Temperature

<table>
<thead>
<tr>
<th>METHOD</th>
<th>COMMENTS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Esophageal probe</td>
<td>Easy to insert; falsely high temperature readings possible with warmed oxygen via an endotracheal tube</td>
</tr>
<tr>
<td>Rectal probe</td>
<td>Insert to 15-20 cm; if the probe is in or surrounded by cold stool, temperature recordings will lag behind true changes</td>
</tr>
<tr>
<td>Temperature-recording Foley catheter</td>
<td>Inflowing cold urine may falsely lower temperature recordings</td>
</tr>
<tr>
<td>Pulmonary artery catheter</td>
<td>Most accurate and most invasive method; higher potential for iatrogenic injury, especially ventricular fibrillation in cold, irritable myocardium</td>
</tr>
</tbody>
</table>

Fig. 131.1  Osborn wave seen on the electrocardiogram of a patient with hypothermia. The wave is usually seen at body temperatures lower than 30° C; however, it is neither diagnostic nor prognostic of hypothermia and is thus of academic interest only.
midaxillary line. Fluid is literally poured in by hand, infused with a large (60-mL) syringe, or administered directly with a rapid infuser (the hub of the rapid infuser fits snugly into the bore of a 36-French chest tube). Rewarming rates average about 3° C per hour.\(^7\)\(^8\)

- Left thoracotomy with mediastinal irrigation and internal cardiac massage is quite invasive with high attendant morbidity. It is very effective and self-explanatory and has rewarming rates as high as 5° C to 6° C per hour.\(^7\)\(^8\)
- Cardiopulmonary bypass (CPB) is the definitive method for rewarming. It is rapid, with rewarming rates of 9° C per hour or higher achieved, and supports blood pressure; however, CPB also requires specialized equipment and personnel that are not readily available in most hospitals.\(^9\)\(^10\)

The use of medications, particularly cardioactive medications and vasopressors, is theoretically unappealing and thought to be potentially dangerous in a patient with a core temperature lower than 30° C, primarily because of decreased metabolism, which can lead to toxic levels. Similarly, defibrillation is less likely to be effective at temperatures lower than 30° C. However, citing the solely theoretic basis of these concerns, the 2010 American Heart Association guidelines now state that in patients with persistent ventricular fibrillation or tachycardia after a single shock it may be “reasonable to perform further defibrillation attempts according to the standard BLS [basic life support] algorithm concurrent with rewarming strategies.” Furthermore, regarding medication administration “it may be reasonable to consider administration of a vasopressor during cardiac arrest according to the standard ACLS [advanced cardiac life support] algorithm concurrent with rewarming strategies.” It is clear that very little is known about the utility of defibrillation and administration of vasoactive medications in patients with hypothermic cardiac arrest. Providers will have to decide each case on an individual basis and be guided by any response to the therapy used.

The phenomenon of core temperature afterdrop refers to the observation that a patient’s temperature can fall after rewarming efforts have begun. It is believed to be due to a combination of temperature equilibration and return of cold blood from the periphery to the patient’s core as perfusion is restored and strengthened. The clinical importance of core temperature afterdrop is keenly contested, and no consistent recommendations can be made regarding it. Certainly, attempts to rapidly rewarm a patient should not be delayed for fear of this consequence.

**NEXT STEPS IN CARE**

Patients with mild hypothermia and (most) patients with moderate hypothermia can be treated in the emergency department and released when normothermic. This statement relies on the assumption that no other complicating social or medical problems are present that must be addressed. All patients with severe hypothermia should be admitted to the hospital, usually to an intensive care unit. Successful revival of these patients will require considerable time and resources.

Clear, universally accepted criteria on declaration of death in hypothermic patients are lacking, beyond the obvious recommendations regarding terminal injuries, rigidity that precludes chest compressions, and physical blockage of the mouth or nose by ice.

Numerous case reports have described neurologically intact survival after prolonged, severe hypothermia with cardiac arrest.\(^7\)\(^12\) A serum potassium level higher than 10 mmol/L has been postulated to be a marker of irreversible cell death and therefore patient death;\(^14\) however, another case report has called this approach into question.\(^19\) Pronouncement of death in a severely hypothermic patient should be made with reluctance until the patient’s core temperature has been warmed to higher than 30° C to 32° C and signs of life remain absent.

A reasonable approach to the hypothermic patient is presented in Figure 131.2.

**FROSTBITE**

**PATHOPHYSIOLOGY**

Frostbite is a freezing injury to tissues. During this process it is believed that deposits of ice crystals causing interstitial, cellular, and vascular endothelial cell damage are one part of the pathophysiological process.\(^15\) The vascular endothelial damage results in activation of the clotting cascade with resulting thrombosis, which leads to hypoperfusion, ischemia, and eventually tissue necrosis. The prominence of the clotting that can cause vascular occlusion can be seen on angiography and is the basis for the concept of treating selected patients with thrombolysis.

**PRESENTING SIGNS AND SYMPTOMS**

The classic victim of frostbite has either a dusky or a white affected area that is brawny in texture, insensate, and without capillary refill. Variations are time dependent, and patients seen in delayed fashion may have blisters that are either hemorrhagic or clear and even some tissue loss or frank necrosis already evident. Classification systems exist for frostbite, but they are controversial and are also problematic for the emergency physician because the initial clinical appearance can be misleading and it takes time for the full extent of the damage to become clear. It is simpler and more realistic to start to treat the affected part and consult surgeons for ongoing wound care and treatment (Fig. 131.3).

**TREATMENT**

Frostbite is a clinical diagnosis. Other cold-related tissue injuries to be considered are frostnip, pernio (chilblains), and trench foot. These injuries, however, are nonfreezing ones, in contrast to frostbite.
The evidence for recommendations is graded using the following scale. **Class I**: Definitely recommended. Definitive, excellent evidence provides support. **Class II**: Acceptable and useful. Good evidence provides support. **Class III**: May be acceptable, possibly useful. Fair to good evidence provides support. **Indeterminate**: Continuing area of research.

This clinical pathway is intended to supplement rather than substitute for professional judgment and may be changed depending on a patient’s individual needs. Failure to comply with this pathway does not represent a breach of the standard of care.

**HYPOTHERMIA CLINICAL PATHWAY**

**Cold patient**
- Check core temperature
- Remove cold clothing
- Insulate to prevent further loss
- Minimize patient jostling
- Keep horizontal
- Treat disorders

**Temperature < 35° C (95° F) = Hypothermic**
- Intubate gently (class I)
- Hold resuscitation
- Check for organized rhythm or sign of life

**Responsive**
- Handle gently
- Cardiac monitor

**Unresponsive**
- Patient frozen solid
- Ice in nose/mouth
- Core temperature < 10° C (50° F)
- Submerged >1 hour
- Obvious lethal injury

**Check temperature**
- Mild: 32° C to 35° C (89.6° C to 95° C)
- Moderate: 28° C to 32° C (82.4° C to 89.6° C)
- Severe: < 28° C (82.4° C)

**Check for organized rhythm or sign of life***
- YES
- Consider holding CPR (class III)
- Start CPR (class III)
- PEA Bradycardia
- Check temperature
- Consider pacing (in determinant)
- NO
- Vfib Asystole
- Shock ≥1
- ALCS drugs ≥1

**Pulse present?**
- Pulse
- NO
- Consider holding CPR (class III)
- Start CPR (class III)
- PACS drugs ≥1
- Check pulse
- NO
- Organized Vfib/asystole

**Pulseless**
- Check for organized rhythm or sign of life
- YES
- Consider holding CPR (class III)
- Start CPR (class III)
- PEA Bradycardia
- Check temperature
- Consider pacing (in determinant)
- NO
- Vfib Asystole
- Shock ≥1
- ALCS drugs ≥1

**Active internal rewarming**
- Active external rewarming
- Extracorporeal blood warming (CAVR)

**Passive rewarming**
- Active external rewarming
- Passive rewarming

**Spontaneous ventilation**
- Spontaneous movement/sound
- Audible beat on auscultation

* Signs of life

PREHOSPITAL MANAGEMENT
Treatment of frostbite in the field consists of the application of dry sterile dressings to separate the involved digits and elevation of the affected extremity. Avoid rewarming with dry heat, such as with fires or heaters, and also assiduously prevent further cold injury. Rubbing the affected part with snow is soundly condemned. It is paramount to avoid a freeze-thaw-freeze cycle, which worsens the tissue damage.

HOSPITAL MANAGEMENT
Once the patient is in the hospital, the mainstay of treatment is rapid rewarming with a circulating bath of water heated to 40° C to 42° C for 10 to 30 minutes until the involved area is erythematos and pliable. Because rewarming is extraordinarily painful, liberal use of parenteral analgesics is usually necessary. Clear, large blisters should generally be débrided, but hemorrhagic ones should be left intact (their presence implies much deeper damage, and desiccation of the area is a concern). Débridement removes fluid that is rich in thromboxanes and prostaglandins, which are thought to be destructive to tissue. Aloe vera may be applied topically every 6 hours and the wounds bandaged.

Prophylactic antibiotics are controversial. Penicillin G has been advocated. Additionally, ibuprofen, 400 mg by mouth twice daily, is recommended in an attempt to interrupt the arachidonic acid cascade. Both catheter-directed intraarterial and systemic thrombolytic therapies have been used with impressive success in preventing amputations (Figs. 131.4 and 131.5). This novel therapy holds considerable promise but does have several limitations, including restriction to patients initially seen within 24 hours of the injury and risk for bleeding.

Most recently, a small, randomized trial of frostbite therapies showed remarkable success with intravenous iloprost, a prostacyclin (no digit amputations), over an IV nonsteroidal antiinflammatory drug (=40% digit amputation rate) or recombinant tissue plasminogen activator plus iloprost (=3% digit amputation rate). Whether this promising success can be repeated and confirmed elsewhere remains to be seen.

Early surgical management is not indicated for frostbite because of the difficulty of ascertaining the full extent of the
Frostenip is a superficial freezing injury that appears pale and can be associated with discomfort. It resolves with rewarming without sequelae.

Pernio (chilblains) is due to repeated, intermittent exposure to wet, nonfreezing temperatures. Localized edema, erythema, nodules, plaques, cyanosis, and possibly vesicles and ulcerations appear up to 12 hours after exposure. Burning paresthesias and itching may be present, and rewarming can result in the formation of bluish nodules. Care is supportive and consists of rewarming, bandaging, and elevation. Nifedipine, pentoxifylline, or limaprost has been advocated as potential therapy. Topical and oral corticosteroids may be useful.

Trench foot is direct soft tissue injury secondary to prolonged immersion in cold water. It develops slowly, but the damage, though initially reversible, may become permanent if not treated. Early in the course, paresthesias develop in a pale, mottled, insensate, and possibly pulseless foot. It becomes hyperemic after rewarming, with return of sensation (proximal more than distal) and severe burning pain. Edema and blisters can form, and in severe cases, tissue sloughing and gangrene can develop. Prevention is paramount, but when it does occur, trench foot is treated supportively much like chilblains.
tissue damage initially. Typically, the affected area is left to mummify and essentially autoamputate before the formal procedure is carried out. Some newer imaging modalities, such as nuclear scanning and magnetic resonance angiography, may be able to shorten the time to definitive surgery by delineating viable tissue earlier than possible with simple observation.

**NEXT STEPS IN CARE**

Because of the severity of frostbite, the need for daily hydrotherapy and wound care, and the often tenuous social circumstance of those afflicted, most patients with frostbite should be admitted to the hospital under the care of a physician skilled in treating this illness. In many cases, burn centers are an excellent option.

**SUGGESTED READINGS**


**REFERENCES**

References can be found on Expert Consult @ www.expertconsult.com.
REFERENCES