Out of the cold: management of hypothermia and frostbite

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Case

In mid-January a 70-year-old man was brought by ambulance to the emergency department. He had been found lying on a sidewalk in a confused state. An acquaintance said the patient drank alcohol heavily. On examination the patient was drowsy, but he responded to voice commands by opening his eyes. His limbs were cold, blanched and pulseless. Muscle tone was increased, but the patient was not shivering. Reflexes were absent. His blood pressure was 90/52 mm Hg, pulse 50 and respiratory rate 8. His rectal temperature was 31°C. Crackles were heard at the right lung base. Bowel sounds were absent. The hemoglobin level was 182 g/L, and the leukocyte count was 12×10^6/L. On room air, the arterial blood pH was 7.25, partial pressure of carbon dioxide 52 mm Hg and partial pressure of oxygen 60 mm Hg. The anion gap was 18 mmol/L, the glucose level 12 mmol/L, the creatinine level 210 μmol/L, the creatine kinase level 1200 U/L and the ethanol level 21 mmol/L. The platelet count, international normalized ratio, partial thromboplastin time, aspartate aminotransferase level and amylase level were normal. An electrocardiogram showed sinus bradycradia and J waves. A chest radiograph showed a consolidation in the right lower lobe.

What is the severity of the patient's hypothermia and frostbite? How should he be managed acutely? What rewarming techniques should be used? How should the frostbite be treated?

Although cold-induced injuries and hypothermia are usually accidental, they can result from social (e.g., homelessness, inadequate home heating),[1–3] occupational,[4–7] recreational,[8–11] iatrogenic[12,13] and even criminal causes.[14] It is difficult to obtain data on the incidence of cold-induced injuries and hypothermia. Hospital data are the "tip of the iceberg," representing only the most severe cases. According to Statistics Canada, there were 411 deaths from hypothermia, frostbite and other cold-induced injuries during 1992–1996.[15] Several case series in western Canada of patients admitted to hospital with frostbite identified the main associated factors to be alcohol consumption, psychiatric illness, and motor vehicle accident or breakdown.[16–18]

There are many types of cold-induced injury. In this article, we review the pathophysiology of the most common forms — hypothermia and frostbite — and provide strategies for the diagnosis and management of these conditions.

Pathophysiology

At temperatures below –10°C, wind speed increases the risk of cold-induced injuries.[19] Environment Canada now reports a wind chill index in temperature equivalents. With a wind chill index of –25, there is a risk of frostbite, and with a wind chill index of –45, exposed skin will freeze in minutes (www.msc.ec.gc.ca/education/windchill). Humidity and skin moisture increase the rate of skin cooling through evaporation.[20] Hypothermia develops faster with submersion in water than with exposure to cold air. Clothing with wind resistance and an insulating layer is protective.

Underlying conditions that increase susceptibility to cold

Many underlying conditions increase a person's susceptibility to cold (Box 1). Infants have a high ratio of body surface area to mass, and elderly people have a decreased capacity for metabolic heat production and vasoconstriction.[21] The amount of muscle and fat a person has may be more important than his or her age in defending against hypothermia.[22] Less body fat decreases tissue insulation. Malnutrition and exertion decrease the fuel available for heat generation.[23] Alcohol and sedative drugs dull mental awareness of cold and impair the judgement necessary to seek shelter or put on warm clothing. In addition, alcohol inhibits shivering, perhaps by causing hypoglycemia,[24] and causes cutaneous vasodilatation, which counteracts the thermoregulatory response.[25] The opioid meperidine and the α-adrenergic blocker clonidine also inhibit shivering.[26] Neuroleptic drugs may predispose to hypothermia through antagonism of the serotonin receptor.[27] Endocrine conditions such as hypothyroidism, adrenal insufficiency and hypoglycemia are accompanied by decreased metabolic heat production. People with diabetes mellitus may have peripheral or autonomic neuropathy. Peripheral neuropathy impairs the cold-induced nociception that might trigger retreat from cold. Both peripheral neuropathy and spinal cord damage may impair cutaneous vasoconstriction.[28] Autonomic neuropathy impairs reflex peripheral vasoconstriction.[29] Central nervous system degeneration, trauma or neoplasms may affect the hypothalamic thermoregulatory centre. Falls may be accompanied by hypothermia because of immobility and conductive heat loss.[30] Hip fractures are more common during cold weather because of a greater risk of slipping or slower reaction times.[31] Infection is common and can either be a precipitant or a complication of hypothermia.[32]

Clinical manifestations

Cold-induced injury leads to clinical manifestations related to thermoregulation, cellular dysfunction, ischemia and edema (Fig. 1).[33]

Thermoregulatory response

The preoptic hypothalamus controls heat conservation, triggers sympathetic activity and is activated by cold stimulation of cutaneous thermoreceptors. Sympathetic activity causes peripheral and visceral vasoconstriction, hypertension, tachycardia, ileus, and bladder atony.

Cellular and tissue effects

Cold damages tissues through cellular injury and vascular impairment.[34] Cellular injury may be due to intracellular water crystallization, temperature-induced protein changes and membrane damage.[35] Slow cooling crystallizes extracellular water, which decreases interstitial water in the liquid phase and draws water out of cells. This effect alters intracellular electrolyte concentrations, which modifies cellular protein structure.[36]

Vasoconstriction, endothelial injury and thromboembolism contribute to vascular insufficiency and ischemia. Inflammatory mediators such as prostaglandins may have a role as well.[37] Vasoconstriction causes hypoperfusion and stasis. Endothelial injury causes thrombosis and loss of vascular integrity. Thromboembolism, from stasis and endothelial injury, may also be promoted by hemoconcentration and hyperviscosity. However, cold-induced inhibition of coagulation-cascade enzymes[38] and platelet dysfunction can also cause bleeding.[39]

As tissues thaw, marked edema occurs because of melting of water crystals, cellular damage, loss of endothelial integrity and thrombosis. Hemorrhage may occur as well. Over time, necrosis and gangrene become apparent.

Systemic effects

Fluid sequestration in damaged tissues may cause volume depletion. Despite a decreased glomerular filtration rate, "cold diuresis" may occur, believed to be due to a defect in distal tubular sodium and water reabsorption, cold-induced glycosuria or inhibition of antidiuretic hormone.[40] Acidemia may be caused by a combined lactic and respiratory acidosis.[41]

Although hypertension and tachycardia occur initially, hypothermia directly decreases cardiac contractility, which, along with volume depletion from fluid sequestration in tissues, decreases cardiac output and blood pressure and may progress to shock. Shock may also occur after rewarming,

owing to capillary leakage of fluids and plasma proteins or disturbed calcium and phosphate homeostasis.[42] Edema and ischemia cause muscles to stiffen. Ischemia may lead to rhabdomyolysis. Cognitive performance decreases,[43] and nerve conduction slows. Patients may appear dead in cases of severe hypothermia.[44]

Diagnosis

Hypothermia

Hypothermia, defined as a core body temperature of less than 35°C, has multi-systemic clinical features (Table 1).[33,45,46] It may be difficult to detect because clinical thermometers are inaccurate below 35°C.[47] The presentation may be subtle in older patients[32] and in patients suspected of alcohol intoxication. Although cutoff points vary, hypothermia can be classified as mild (core body temperature > 32°C and < 35°C), moderate (core temperature 28°C–32°C) or severe (core temperature < 28°C).[33]

Frostbite

Frostbite, one of the many types of local cold-induced injury (Table 2), most commonly affects the extremities.[17] Although frostbite has been classified by degree,[48] it may be more usefully classified as superficial or deep.[49] Superficial frostbite affects the skin and subcutaneous tissues; deep frostbite also affects bones, joints and tendons. With the superficial form, rewarmed skin has clear blisters, whereas with deep frostbite the rewarmed skin has hemorrhagic blisters. Favourable prognostic factors include retained sensation, normal skin colour and clear rather than cloudy fluid in the blisters, if present. Poor prognostic features include nonblanching cyanosis, firm skin and dark, fluid-filled blisters. However, no prognostic features are entirely predictive,[36] and weeks may pass before the demarcation appears between viable and nonviable tissues.[50] To define the extent of ultimate tissue loss, some authors advocate triple-phase bone scanning[51] or MRI scanning.[52] Although such techniques may permit earlier surgical management, controlled clinical trials are lacking.

Management

Management demands attention to hypothermia and local cold-induced injury as well as to coexisting intoxication, trauma, infection and central nervous system disease.

Box 1: Factors that increase susceptibility to cold

General: Infancy, advanced age, malnutrition, exhaustion

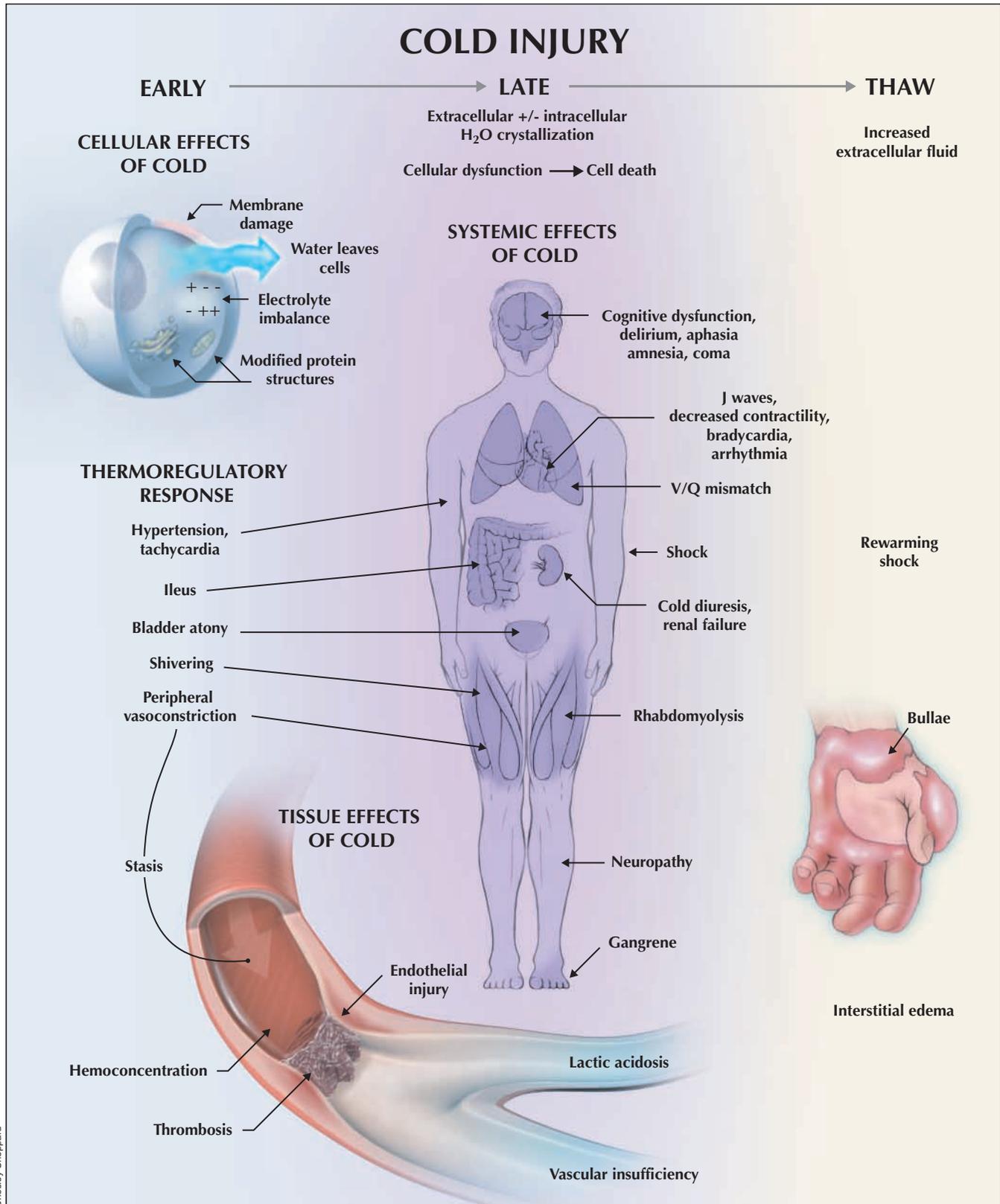**Drug use:** Alcohol, sedatives, meperidine, clonidine, neuroleptics

Endocrine system: Hypoglycemia, hypothyroidism, adrenal insufficiency, diabetes

Cardiovascular system: Peripheral vascular disease, nicotine use

Neurologic system: Peripheral neuropathy, spinal cord damage, autonomic neuropathy, hypothalamic disease

Trauma: Falls (head or spinal injury), fracture causing immobility

Infection: Sepsis (diaphoresis, hypothalamic dysfunction)

COLD INJURY

EARLY → LATE → THAW

CELLULAR EFFECTS OF COLD

Extracellular +/- intracellular H_2O crystallization

Cellular dysfunction → Cell death

Increased extracellular fluid

Membrane damage

Water leaves cells

+ - -
- ++

Electrolyte imbalance

Modified protein structures

SYSTEMIC EFFECTS OF COLD

Cognitive dysfunction, delirium, aphasia amnesia, coma

J waves, decreased contractility, bradycardia, arrhythmia

V/Q mismatch

THERMOREGULATORY RESPONSE

Hypertension, tachycardia

Ileus

Bladder atony

Shivering

Peripheral vasoconstriction

Shock

Cold diuresis, renal failure

Rewarming shock

TISSUE EFFECTS OF COLD

Stasis

Rhabdomyolysis

Bullae

Neuropathy

Gangrene

Endothelial injury

Hemoconcentration

Thrombosis

Lactic acidosis

Vascular insufficiency

Interstitial edema

Chesley Sheppard

Fig. 1: Cold-induced injuries such as hypothermia and frostbite lead to thermoregulatory response (e.g., shivering and increased sympathetic activity), cellular and tissue effects (e.g., membrane damage, electrolyte imbalance, endothelial injury and thrombosis) and systemic effects (e.g., shock, arrhythmia and neuromuscular dysfunction).

Hypothermia

Moderate to severe hypothermia is a medical emergency necessitating maintenance of airway, breathing and circulation. The core body temperature is monitored by means of an esophageal or rectal probe.

Because of the risk of arrhythmia, patients should have cardiac monitoring (needle electrodes may be necessary) and should be transferred gently. Venous access lines and arterial pressure monitoring should be established for fluid resuscitation in case of shock, which can be present initially or develop later with rewarming.[41] Pulseless electrical activity may be caused by severe vasoconstriction and volume depletion. Cardiac life support for hypothermia is unique for several reasons. First, careful monitoring of the carotid pulse is needed because bradycardia is common and shivering artifact may simulate ventricular fibrillation. Second, a frozen thorax makes chest compression difficult. Although some have suggested withholding chest compressions, fearing precipitation of ventricular fibrillation,[53] we believe it is reasonable to follow current guidelines for advanced cardiac life support,[54] arguing for maintenance of cerebral blood flow. Third, ventricular fibrillation may not respond to electroshock or drugs until the patient is rewarmed. Fourth, if feasible, the best treatment may be cardiopulmonary bypass.[46,55] On the electrocardiogram, positive deflections following the QRS complex (J, or Osborn, waves) are a clue to hypothermia, but they also may be confused for ST-segment elevation of myocardial infarction. Although J waves are more prominent with severe hypothermia, they do not reliably predict prognosis.[56] Arrhythmias such as atrial fibrillation and junctional bradycardia are associated with poor prognosis,[57] and asystole and ventricular fibrillation occur with severe hypothermia.

Blood work should include a complete blood count, measurement of electrolyte, glucose, creatinine and creatine kinase levels, and determination of the coagulation profile (international normalized ratio and partial thromboplastin time). Arterial blood gas analysis assesses ventilation and acid–base balance. Elevations in liver enzyme and amylase levels indicate hepatic dysfunction and pancreatitis respectively. Urinalysis and cultures of urine and blood should be done to identify infection, which can be both a precipitant and a complication of hypothermia. Measurement of serum ethanol levels detects alcohol intoxication, which is a common precipitant of hypothermia.

A chest radiograph may reveal signs of aspiration pneumonia. Other radiographs may be needed to exclude trauma (e.g., head injury, hip or spine fracture).

Rewarming techniques

There is a paucity of controlled trials of rewarming techniques in accidental hypothermia.[58,59] Some evidence of the relative effectiveness of these techniques comes from studies

Table 1: Multi-systemic features of hypothermia and rewarming techniques

System*	Mild hypothermia (core > 32°C)	Moderate hypothermia (core 28°C–32°C)	Severe hypothermia (core < 28°C)
Thermoregulatory	Shivering intact	Shivering lost, rapid cooling	Shivering lost, rapid cooling
Respiratory	Tachypnea	Hypoventilation, respiratory acidosis, hypoxemia, aspiration pneumonia, atelectasis	Apnea, adult respiratory distress syndrome
Cardiovascular	Tachycardia, hypertension	Hypotension, bradycardia, prolonged QT interval, J waves (leads II and V_6)	Pulseless electrical activity, atrial fibrillation, heart block, ventricular fibrillation, asystole
Gastrointestinal	Ileus	Pancreatitis, gastric erosions	Pancreatitis, gastric erosions
Genitourinary and fluids/electrolytes	Bladder atony, cold diuresis	Hyperkalemia, hyperglycemia, lactic acidosis	Hyperkalemia, hyperglycemia, lactic acidosis
Muscular	Hypertonia	Rigidity	Rhabdomyolysis
Hematologic	–	Hemoconcentration, hypercoagulability	Disseminated intravascular coagulation, bleeding
Neurologic	Hyperreflexia, disorientation, ataxia, dysarthria	Hyporeflexia, agitation, hallucination, dilated pupils	Areflexia, coma, absent pupil responses, brain-dead–like state
Rewarming technique†	Passive rewarming (active external rewarming may be required if the patient is unable to generate heat through shivering because of exhaustion or advanced age)	Active external and noninvasive internal rewarming (invasive internal rewarming may be required if there is cardiovascular compromise)	Active external, noninvasive internal, invasive internal or extracorporeal blood rewarming (venovenous or arteriovenous hemodialysis, cardiopulmonary bypass)

*The systemic effects are a continuum and depend not only on core temperature but also on patient features. Rewarming methods need to be decided on an individual basis.
†Passive: blanket (covering body and head). Active external: forced air or heating blanket; warm baths are not recommended because they make monitoring difficult.
Noninvasive internal: warmed oxygen and warmed intravenous fluids. Active internal: peritoneal lavage, pleural lavage, esophageal warming tubes.

of intraoperative hypothermia,[60,61] experimental hypothermia,[62,63] case series[64] and case reports.[65] Rewarming may be passive or active. Passive rewarming involves the use of blankets to cover the body and head; depending on shivering thermogenesis the warming rate may be 0.5°C–2°C per hour with this technique.

Active rewarming can be external or internal. Active external warming includes the use of heating blankets or a heated forced-air system. In a randomized trial involving 100 patients with minor trauma, of whom 80 had hypothermia, heating blankets warmed at a rate of 0.8°C per hour, as compared with a temperature drop of 0.4°C per hour with the use of wool blankets.[58] In another randomized trial, involving 16 adult patients with moderate or severe accidental hypothermia, rewarming rates were 1°C per hour faster with a forced-air system than with cotton blankets.[59] Warm baths are not recommended because they make monitoring difficult.[33]

Active internal rewarming techniques can be simple or invasive. Simple techniques may be initiated immediately and include the use of warmed intravenous fluids (heated to 43°C by microwave or heat infusion pump)[66] and warmed oxygen,[67] which together warm at a rate of 1°C–2°C per hour. Invasive techniques warm faster. For example, heated irrigation techniques warm at a rate of 1°C–4°C per hour and include peritoneal exchange,[68] thoracostomy lavage[64] and the use of esophageal warming tubes.[60] Extracorporeal rewarming of blood warms at a rate of 1°C–2°C per 5 minutes and includes continuous arteriovenous or venovenous rewarming (a pulse must be present), heated hemodialysis (useful in cases of renal failure or hyperkalemia) and cardiopulmonary bypass (in cases of cardiac arrest).[46]

Table 2: Local cold-induced injuries

Injury	Symptoms and signs
Axonal degeneration	Numbness, dysesthesia, cutaneous vasomotor instability; sensitivity to cold may persist for years
Chilblains (pernio)	Pruritic patches of erythema and cyanosis, especially on hands and feet, that may blister, ulcerate, scar or atrophy
Cold-contact adhesion†	Erosion or ulcer on forcible separation
Frostbite	
Superficial	Pallor, edema, blistering, desquamation
Deep	Hemorrhagic blisters and anesthesia, followed later by hyperesthesia, ulceration and gangrene
Frostnip	Transient numbness and tingling without residual tissue damage
Immersion syndrome (trench foot)*	Alternating vasoconstriction (cold, pallor, cyanosis and pulselessness) and vasodilatation (warmth, erythema and edema), ecchymosis, blistering, lymphangitis, cellulitis, thrombophlebitis, gangrene

*Limb, often inactive, is immersed for prolonged period in non-freezing cold water or mud.
†Contact between metal and moist skin or mucosa.

For mild hypothermia (core body temperature > 32°C), passive external rewarming is usually sufficient. However, active external rewarming may be required if patients are unable to generate heat through shivering (e.g., they are too exhausted or are elderly).

For moderate hypothermia (core temperature 28°C–32°C), active external rewarming should be applied to the trunk rather than the extremities because an "afterdrop" in core temperature may occur when blood supply to the cold periphery is recirculated to the core. External heat may also decrease the stimulus for shivering thermogenesis. For patients in whom shock occurs after rewarming,[41] volume expansion with warmed intravenous fluids is required.[69]

For severe hypothermia (core temperature < 28°C), or for circulatory arrest, invasive internal rewarming techniques are recommended. Walpoth and associates[46] described 32 hypothermic patients with circulatory arrest who underwent cardiopulmonary bypass; about half survived and had no significant long-term sequelae.

Frostbite

In the pre-hospital care of frostbite, nonadherent wet clothing should be removed. Local rewarming should begin only if refreezing will not occur in transit. Rubbing affected areas worsens tissue damage. In hospital, rapid rewarming of a frostbitten extremity in a bath of water between 40°C and 42°C for 15 to 30 minutes may minimize tissue loss.[70] Narcotics and tetanus toxoid should be given as required. Splinting and elevation of the affected part may reduce edema and promote tissue perfusion.

Management of frostbite blisters is controversial. Some advocate their removal because of the high concentrations of prostaglandin $F_{2\alpha}$ and thromboxane A_2 in the exudate.[71] However, removal of blisters is associated with a risk of desiccation of exposed tissue. If the blisters do not interfere with range of motion, it may be reasonable to leave them intact. If they are large and need to be removed, or if they rupture, the area underneath should be débrided and covered with a topical antimicrobial and gauze.

Hospital stays for patients with deep frostbite are often prolonged because of immobility and because of the slow evolution of the injury. Escharotomy may be needed in cases of vascular impairment. Mummification and autoamputation may occur. Surgical amputation may be required many weeks after the injury.

The case revisited

Our patient had moderate hypothermia and deep frostbite. Mechanical ventilation was started, and he was given warmed oxygen and intravenous fluids and covered with a forced hot-air blanket. A nasogastric tube and Foley catheter were inserted. The patient was given antibiotics for pneumonia as well as thiamine. He was gently transported to the intensive care unit, where he was monitored for 24 hours.

Fig. 3: Surgical amputation of left midfoot and right toes 2 months after presentation with deep frostbite.

During this time he became more alert, and ventilatory support was no longer needed. His blood work and electrocardiogram findings returned to normal. Within 24 hours his feet and hands had large bullae (Fig. 2), which ruptured spontaneously over the next few days. During his second month in hospital, he had amputation of his left midfoot and right toes (Fig. 3). Social services arranged for the patient to live in a supervised group home after discharge.

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Fig. 2: Large bullae on hands and feet of man with moderate hypothermia and deep frostbite.

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