high calorie enteral formula. Diabetic patients quickly become hyperglycemic after thermal injury and frequently require an insulin drip. The insulin infusion should be titrated to maintain tight control of serum glucose to avoid glycosuria. In diabetic burn patients, glycosuria can be present at seemingly insignificant elevations of serum glucose levels, frequently leading to hypovolemia and hyperosmolality. Diabetic intestinal dysmotility and delayed gastric emptying should be managed by postpyloric placement of a feeding tube and the selective use of metoclopramide. The use of insulin infusion to obtain tight control of serum glucose will usually allow delivery of standard enteral formulations. Diabetics with large burns may benefit from a decrease in carbohydrate calories and an increase in the proportion of calories from proteins and fatty acids.

CONCLUSION

Victims of major burns become markedly catabolic because of stress and inflammation. The catabolic state persists for an extended period, causing muscle wasting and immune suppression. The result of the catabolic state is poor wound healing, septic complications, multiple organ failure, and eventual death unless adequate nutritional support is initiated. Early intervention via enteral feeding that delivers appropriate mixture of nutrients will decrease protein loss and improve wound healing and immune function. Optimal outcomes require continuous reassessment and reformulation. Several adjuncts to the "fuel mixture" have great potential for further limiting the metabolic impact of severe thermal injury.

SUGGESTED READING

The American Burn Association Evidence Based Guidelines Group: Initial nutritional support of burn patients: practice guidelines for burn care, *J Burn Care Rehabil Suppl* 59s, 2001.

Curreri PW, et al: Dietary requirements of patients with major burns, *J Am Diet Assoc* 65:415, 1974.

Ramzy PL, Barret JP, Herndon DN: Thermal Injury, *Crit Care Clin* 15:333, 1999.

Zaloga GP: Immune-enhancing enteral diets: where's the beef? *Crit Care Med* 26:1143, 1998.

COLD INJURY

Navin K. Singh

In the medical literature, cold injury is written about even in the writings of Hippocrates, Aristotle, and Galen. Frostbite in military campaigns has been reported from Hannibal who lost half his army crossing the Alps in the third century BC, to World War I, World War II, and the Korean War. Napoleon's infamous retreat from Moscow in 1812 was related to the ravages of the winter. The German army performed more than 15,000 amputations for cold-related injuries on the Russian front in the winter of 1942.

Cold injury can be broadly classified into (1) accidental hypothermia, signifying systemic derangements of thermoregulation, and (2) local injury, such as frostbite with focal tissue destruction (Table 1). The physiology and risk factors for these processes are the same.

PHYSIOLOGY

Homeothermy is maintained for the essential viscerae and deep muscles. The poikilothermic shell includes the skin, subcutaneous tissues, and the superficial muscles and is responsible for temperature modulation. Heat loss is mediated via radiation (the dissipation of heat via infrared radiation), convection (via currents to environmental air or water), conduction (the transfer of heat via direct contact), and evaporation (facilitated by diaphoresis).

Radiation accounts for 55% to 65% of heat loss. Evaporation and respiration account for 20% to 30%, principally through sweating. Conduction accounts usually for less than 5% of heat loss. Convection from movement of air currents can lead to losses of up to 15%. Wet clothing increases heat loss fivefold, and water immersion, because of total conduction and convection, can accelerate heat loss by a magnitude of 25 times. When compared to conduction against air, cold-water immersion conduction is up to 32 times higher.

Integumentary blood flow has a profound range to facilitate homeothermy; due to cold stress, it can drop as low as 0.5 mm per minute per 100 ml of tissues under vasoconstriction. Or, it can range to 30% of the cardiac output when heat is being dissipated. With cooling, shivering induces thermogenesis, with a fivefold increase in heat production, but fails once glycogen stores are depleted. The basal metabolic rate (BMR) may be accelerated via catecholamines.

The homeothermic process is coordinated by the hypothalamus. These four mechanisms are either limited or facilitated in an effort to maintain core temperature at 37°C.

RISK FACTORS

Metabolic risk factors include hypothyroidism, hypoglycemia, or hypoadrenalism, due to an inability to meet the metabolic demands of thermoregulation. The patient may not be able to peripherally vasoconstrict, mount a shivering response, or have a low BMR. Lean body type (fat provides insulation) is also a risk.

Neurologic risk factors include head injury, spinal cord injury, stroke, hypothalamic lesions, Alzheimer's disease, fatigue, mind-altering substances, and alcohol. Consumption of alcohol is particularly problematic because it not only impairs cognition but also causes peripheral vasodilation, accelerating heat loss. Alcohol intoxication, substance abuse, or psychiatric illness may prevent individuals from seeking appropriate shelter to retreat from the cold or may create an indifference to the insult.

Age-related factors, though not conclusively borne out in the epidemiology literature, should be considered. The elderly are considered at risk because a decline in cognitive abilities may hinder the ability to seek protective clothing or shelter. Further, because of reduced muscle bulk, they may be unable to accomplish thermogenesis

Table 1: Cold Injury

Accidental Hypothermia	Local Cold Injury
1. Mild	1. Pernio or chilblain
2. Moderate	2. Trench foot (nontropical immersion foot)
3. Severe	3. Freezing injury/frostbite A. Frostnip B. Superficial frostbite C. Deep frostbite

by secondary mechanisms such as shivering. The very young have a high surface-to-body mass ratio, which allows greater heat loss. Epidemiologically, most cases of cold injury are reported in patients aged 30 to 49 years, with a 10:1 ratio of men to women.

Climatic hazards include temperature, wind, and altitude. For instance, high altitude (more than 17,000 feet) frostbite is reported in World War II aviators. Outdoor winter sports, especially those requiring prolonged exposure or coupled with high altitude or high windchill factor, predispose to hypothermia. Accident victims with lengthy extractions/rescues or delayed presentation are particularly susceptible. In an urban setting, the increasing numbers of homeless persons exceeds the capacity of shelters, leading to vulnerable, malnourished individuals facing cyclic and repetitive exposure in wintertime.

ACCIDENTAL HYPOTHERMIA

Accidental hypothermia is defined as a body core temperature below 35°C (95°F). Once seen only in military campaigns, because of a societal premium on outdoor recreation, urban problems of homelessness, and substance abuse, it is seen with increasing frequency in urban populations. Hypothermia is related to both duration and extreme of temperature. Thus hypothermia can occur even in cool weather. Surgical familiarity with the principles of resuscitation and management are emphasized in this discussion.

Classification

Mild hypothermia is defined as temperatures of 90°F to 94°F (greater than 32.2°C). Presenting signs include shivering, complaints of being cold, mental states ranging from slight confusion to clarity, and a normotensive physiologic examination. Moderate hypothermia ranges from 80°F to 89°F (26.7°C to 32.2°C). The patient may be combative, agitated, and delirious. Shivering has ceased. Muscle spasticity, dilated pupils, and bradypnea are found. Myocardial irritability is encountered. Severe hypothermia temperature is less than 80°F (less than 26.7°C). The patient here is comatose with flaccid paralysis, apnea, and spontaneous ventricular fibrillation.

Pathophysiology

Cardiovascular

After an initial increase in cardiac output with tachycardia, there is progressive decline with worsening severity of hypothermia. There is an accompanying hypotension. However, with full-body immersion in cold water including the head, the "diving response" is initiated. This "diving" reflex involves apnea, bradycardia, and increased total peripheral resistance with decreased stroke volume/cardiac output and an increased mean arterial pressure. The diving reflex also has associated cardiac arrhythmias with sudden death as a possibility. However, the diving reflex, along with cold exposure, may explain prolonged submersion survival, because of minimizing cardiac work, vasoconstricting all noncritical vascular beds, leading to oxygen conservation. It should be noted that 16°C is the lowest adult accidental hypothermia survival and 15°C is the lowest infant accidental hypothermia survival.

In severe hypothermia, signs of myocardial irritability such as atrial fibrillation and ventricular ectopy are noted. At temperatures less than 25°C, the heart spontaneously undergoes ventricular fibrillation or cardiac standstill with drops to 21°C.

There is a 2% increase in blood viscosity for each 1°C drop in temperature. Hemoconcentration is seen related to secondary cold diuresis. Rheologic blood flow is suboptimal with sludging and even thrombosis.

Pulmonary

After an initial tachypnea in sudden immersion in cold water, hypothermia creates pulmonary respiratory depression. Pulmonary edema is found after rewarming. Collection of secretions and suppression of the cough reflex creates "cold bronchorrhea." The metabolic rate is reduced to about half at 30°C. Because hypothermia overall decreases oxygen consumption by about 50% at 30°C (86°F), the shift in the oxygen hemoglobin disassociation curve to the left is not seen. This may lead to the finding of metabolic acidosis in one third of patients after rewarming because of respiratory depression and shivering.

Renal

"Cold diuresis" leads to fluid loss and hypovolemia in the setting of hypothermia. This may be mediated via decreased sensitivity to antidiuretic hormone. This develops despite decreased glomerular filtration rate and renal blood flow.

Central Nervous System

The progressive confusion seen with hypothermia is mediated via decreased cerebral blood flow, which decreases 6% to 7% for every 1°C drop in core temperature.

Treatment

Treatment consists of three phases of (1) field or prehospital care, (2) definitive care, and (3) long-term care.

Prehospital

Prehospital care consists of evacuation from a cold environment including removal of cold wet clothing. Trauma to the body and body parts is minimized by avoiding massage, friction rubbing, and manipulation. Frozen body parts should be immobilized, padded, and splinted. Those patients found in arrest should undergo cardiopulmonary resuscitation or advanced cardiac life support per protocol. It should be emphasized that the patient is not pronounced dead until "the patient is warm and dead." At this point, only passive rewarming is undertaken because active rewarming can lead to myocardial instability and fibrillation. Also, with rewarming, there may be a precipitous drop in blood pressure named "afterdrop," which might not be manageable in the field without large-bore peripheral intravenous lines or a central line.

Definitive

Definitive treatment in the emergency department begins with making the diagnosis. One must confirm that the thermometer recording the temperature has an adequate scale to measure extremes. Most hospital thermometers are manufactured within a physiologic range and may not report the low extremes of temperature encountered in accidental hypothermia. If shivering is present, the patient most likely has mild hypothermia. The absence of shivering prognosticates more severe hypothermia.

Routine laboratory investigations such as complete blood cell count, electrolytes, liver function tests, coagulation panel, and arterial blood gas (ABG) analyses are sent, as well as drug toxicology screens and blood alcohol levels. Large-bore intravenous access is established and a Foley catheter with urimeter is inserted for evaluation of volume status. One must be mindful that cold diuresis or alcoholic diuresis may mislead the treating physician into thinking the patient is adequately hydrated. Electrocardiograms (ECGs) and chest x-rays are obtained as well. The patient must be continuously monitored in an intensive care setting. Serial blood work including electrolytes and monitoring for hyperkalemia and rhabdomyolysis

are obtained. Serial ABGs and continuous ECG monitoring are indicated. Of note, pulmonary artery catheters (Swan-Ganz catheters) are contraindicated because they may trigger myocardial irritability.

The patient should be administered 5% dextrose or glucose to rule out hypoglycemia. In the apneic patient, naloxone might be indicated to rule out the narcotized patient. At this point, active rewarming measures can be undertaken. Rewarming techniques are classified into passive or active methodologies.

Passive rewarming in mildly hypothermic patients results in a slow increase in the body core temperature. This range is quoted from approximately 0.5°C to 2°C each hour.

Table 2 lists rewarming techniques for hypothermia. Warm humidified oxygen either by facemask or endotracheal tube in the intubated patient can reverse the cold-induced ciliary dysfunction seen with hypothermia. This humidified air is at 40°C (104°F) in the awake patient and 50°C (122°F) in the unresponsive patient. The advantages touted for active rewarming techniques are that the cardiovascular system is rewarmed first, thus mitigating against the propensity for fatal dysrhythmia.

One must be mindful that because hypothermia can be central nervous system protective from the lowered metabolic rate, resuscitation after durations of up to 45 minutes of hypothermia arrest have been reported in children. Thus again, "no patient is dead until warm and dead." Despite rough guidelines, treatment must be titrated to the individual patient, bearing in mind the premorbid risk factors, duration and severity of injury, and current physiology.

Long Term

Long-term care includes physical therapy, neurologic rehabilitation (although hypothermia can be neuroprotective) such as at a traumatic brain injury institute, and psychologic support (e.g., for posttraumatic stress disorder) and counseling for reintegration into society.

LOCAL INJURY

In the past, reports in the literature and scientific studies on frostbite were all on military personnel. Many military conflicts have had an incidence of frostbite, especially those fought in cold weather. Although cold injury can happen at any temperature, it is traditionally associated with cold weather battles. More and more, the reports in the literature are no longer about military conflict, since stemming from their vast experience on the battlefield, most modern militaries have undertaken measures to prevent or minimize cold injury. Consequently, there is an increased emphasis toward the occurrence and management of cold injury in the civilian population.

Table 2: Rewarming Techniques for Hypothermia

Passive
Warm environment
Shivering
Blanket or clothing insulation
Active
External
Heating pad
Immersion in warm bath
External convection heaters
Internal
Heated intravenous solutions
Hemodialysis
Gastric/colonic lavage
Peritoneal lavage
Mediastinal lavage
Warmed inhalational agents
Extracorporeal circulation

Source: Bickel KD: *Current surgical therapy,* ed 6, St Louis, 1998, Mosby.

The risk factors for frostbite/local injury are the same as those for accidental hypothermia. They include psychiatric disorders, alcohol or substance abuse, and head injury. Outdoor recreation including skiing, hunting, mountain climbing, and snowmobiling continue to be the most significant risk factors for sports enthusiasts. The homeless, elderly, and malnourished person remains the most common in urban cold injury. A previous frostbite injury doubles the risk for a second frostbite injury, whereas experience in cold weather climates such as in northern latitudes, through acclimatization, is considered to decrease the relative risk of frostbite.

Risk factors specific for local injury include peripheral vascular disease, Raynaud's phenomenon, vasoconstrictive drugs, constrictive clothing, neuropathy such as from diabetes, which may lead to the inability to perceive cold injury in the distal tissues, and smoking, which causes vasospasm. Immobilization, such as during military trench warfare, wounded status, or confinement, is also a significant risk factor.

Pathophysiology

The injurious mechanisms in frostbite are related to three processes, as follows:

1. Initial freeze
2. Vascular occlusion
3. Vascular and inflammatory changes

In the cooling phase of the tissues, they initially undergo vasoconstriction with cyclic vasoconstriction and dilation in periods of approximately 5 to 10 minutes, called "the hunting reaction." Once homeothermy of the core is threatened, the hunting reaction is abandoned and the body follows vasoconstriction only. The temperature of the freezing tissue continues to decline until about the freezing point, at which point extracellular ice crystals form. With the formation of crystals, there is a hyperosmolar gradient that causes fluid to be desiccated from the intracellular space into the extracellular space. The intracellular hyperosmolarity can increase up to tenfold; however, the cell itself may be salvageable. In extremely rapid freezing such as that mediated by liquid nitrogen, intracellular ice can also form, causing nonsalvageability of the cells via mechanical destruction. In the vascular occlusion phase, red blood cell sludging occurs because of cold-water diuresis and hemoconcentration, eventually leading to thrombus formation and obstruction of circulation. The inflammatory mechanism creates endothelial cell damage, creating extremely "leaky" vessels which lead to extravasation of proteins, causing massive third-spacing of fluids.

Zones of frostbite are characterized by arteriovenous shunting, which exacerbates the overall injury. Longer durations of exposure produce characteristically more severe injuries. However, most injuries happen from exposures of less than 12 hours in duration and can happen in as brief a period as 6 hours. Thus cold injury does not necessarily require a freezing temperature. Beyond temperature, the windchill factor is extremely predictive of the risk of injury to exposed areas. Other risk factors such as skin contact with metal or water, which leads to conductive heat loss, as well as immobilization and dependency increase the risk of injury. Higher altitude, due to decreased oxygen tension and decreased perfusion, also potentiates injury in a shorter time.

During the rewarming phase with heating in a warm bath at 40°C, the extracellular crystals melt, the capillaries dilate, and free radical–mediated ischemia reperfusion injury ensues. Diffuse thrombosis can lead to microcirculatory failure.

Diagnosis and Treatment

Chilblain, or pernio, is the moderate form of cold injury found in the presence of high humidity and usually occurs on the face, anterior shins, or the dorsal surfaces of the hands and feet. It is thought to be a chronic dermal vasculitis, a localized inflammatory lesion precipitated by the cold with arteriolar and venular constriction accompanied by erythrocyanosis, which can involve the fingers, toes, heel, lower legs, thighs, and ears. It shows erythema, edema with blisters, and ulcerations. This typically resolves in 2 weeks.

Trench foot is ordinarily seen in soldiers immobilized for lengthy periods in damp, cool environments. Trench foot or cold immersion foot (nontropical immersion) develops in wet environments at above freezing temperatures. Commonly affected individuals include sailors, fishermen, military personnel, and those in life rafts, among others. It is characterized by cold, numbness, swelling, and cramping. Synonyms for trench foot include *foxhole foot* or *sea boat foot.* Worsened by dampness, wind, high altitude, and immobility, trench foot disease has four stages, as follows:

1. Exposure to cold
2. Prehyperemic phase, typified by cold, blue, and numb feet
3. Hyperemic phase with edema, warmth, and painful symptoms of approximately 10 weeks in duration
4. Posthyperemic phase with cold intolerance and hyperhidrosis, lasting years

Frostnip is characterized by blue light discoloration of affected skin on the face and fingertips as a result of extreme cold temperature exposure. Treatment involves simple rewarming. Frostnip is completely reversible, whereas frostbite involves the deeper skin layers and causes serious damage to the tissues.

Ninety percent of frostbite occurs in the upper and lower extremities, in particular, in the most acral portions, namely, the fingers and toes. Hands and feet account for 90% of all recorded injuries, although it is also reported in nose, ears, cheeks, and genitalia. Various classification schemes exist for cold injury. Previously, cold injury was classified into first, second, third, and fourth degree. However, this classification scheme provides very little in terms of prognosis, degree of tissue loss, or protocols for treatment and is not correlated with clinical outcomes. Summarily, all frostbite is treated the same way until demarcation at about a month after injury.

Typically, the severity of frostbite injury is classified as either superficial or deep. Superficial injury is classified as partial- or full-thickness skin freezing that creates the symptoms of transient stinging and burning, throbbing, and aching or numbness. Superficial frostbite of the arms and hands is accompanied by clumsiness and lack of fine motor control, which is followed by a throbbing sensation that may last for several weeks. Frostbitten skin may have a waxy yellow or mildly blue appearance and will become hyperemic with rewarming. Blisters will follow in 6 to 24 hours. Good prognosticators in the physical examination include sensation to pinprick, skin with turgor, and capillary refill. Blisters with blood-tinged fluid, pallor, and woody skin auger poor results. Deep frostbite is characterized as initially insensate skin that may feel woody or firm. After severe injury, a black desiccated eschar may develop in about 2 weeks. Autoamputation takes a variable amount of time depending on the severity of injury and its location.

Treatment

Prehospital

Field or prehospital care is typified by mechanical protection and stabilization of the part via splints or bulky dressings and the avoidance of rewarming until definitive care can be delivered in an environment capable of resuscitation. If rewarming is attempted before the thawed body part can be kept thawed and refreezing prevented, cyclic episodes of freezing and thawing will contribute to multiple ischemia reperfusion injuries and worsen the degree of tissue necrosis. Postthaw pain for management of frostbite will prevent the patient from actually ambulating and bearing weight. Thus it is important if an individual needs to be evacuated for him or her to walk to rescue on the frostbitten feet before rewarming. Otherwise evacuation would become impossible secondary to pain. Typically after rewarming, the injured area becomes numb but will progress to aching and burning once edema is established.

Frostnip is the only form of frostbite that can safely be treated in the field. With frostnip, the victim feels severe local cold progressing to numbness and then pain. Clinically the skin is erythematous but not distorted and blister free. Treatment includes removal of wet garments and local warming measures, among others.

Definitive

At the time of admission, in addition to routine laboratory work, blood alcohol levels and toxicology screens are sent. The affected parts are x-rayed to rule out associated fractures. In-hospital care consists of rapid rewarming of the affected part at 40°C to 42°C (104°F to 108°F). Rapid rewarming enhances cutaneous dilation, restoring blood flow and enhancing the degree of tissue survival. The caveat must be issued against warm dry heat because this can cause uneven warming and may burn the compromised and insensate tissues. Whenever combined with accidental hypothermia, the hypothermia should be corrected before attempting treatment of frostbite injuries. Adjunctive analgesics will be required because of the severe pain and throbbing. Topical aloe vera and oral ibuprofen are administered. Tetanus immunization status should be brought up to date. The role for prophylactic antibiotics systemically is unclear. However, there is a role for topical antimicrobial therapy.

Supportive care consists of extremity immobilization and elevation to minimize edema, which can compound the effects of the ischemia reperfusion injury. Splints should not be constrictive. The skin should be protected with dry soft dressings to prevent maceration (Table 3).

Escharotomy might be required when mechanical impingement to circulation or chest excursion from the eschar is suspected. Similarly, fasciotomy might be indicated for compartment syndrome. Because of emphasis on protecting fragile parts, ambulation is limited via wheelchair or crutch ambulation. Smoking, because of the vasoconstrictive properties of nicotine, is *verboten.*

Many adjuvant therapies are considered, although none is demonstrated to increase tissue survival in any prospective randomized control trials. Modalities in this gray area include surgical sympathectomy, dextran, heparin, pentoxifylline streptokinase, and hyperbaric oxygen. There is questionable value to steroid and nifedipine use.

Because prediction of the degree of tissue slough is extremely difficult, radiologic techniques to predict the appropriate amputation level have been developed. These include plain x-rays, which can start to show osteoporosis or osteitis at several weeks. Technetium-99 radioscintigraphy or triple-phase bone scans have been advocated to obtain earlier results. These are useful only on postinjury days 5 to 7 to determine viable soft tissue and bone, because up to 5 days, persistent vascular spasm and instability are encountered, rendering findings equivocal. Angiography has also been advocated. Magnetic resonance imaging or magnetic resonance angiograms may eventually have a role in prognosticating the level of injury. However, no radiologic technique has been conclusively shown to be predictive, and the mainstay of treatment remains watchful waiting. An old adage "frostbite in January, amputate in July" remains the zeitgeist of management. Tissue demarcation is depicted in Figure 1. These tissues must be allowed to demarcate, and, oftentimes, a surprisingly greater level of tissue salvage is encountered than would be if amputation was undertaken acutely. A waiting period of 1 to 3 months will show mummification of the tissues and may even lead to autoamputation.

Table 3: Treatment Protocol for Frostbite

1. Admit frostbite patient to a specialist unit, if possible.
2. On admission, rapidly rewarm the affected areas in warm water at 40°C to 42°C (104°F to 108°F) for 15-30 minutes or until thawing is complete.
3. On completion of rewarming, treat the affected parts as follows:
   a. Débride white blisters and institute topical treatment with aloe vera (Dermaide aloe) every 6 hours.
   b. Leave hemorrhagic blisters intact and institute topical aloe vera (Dermaide aloe) every 6 hours.
   c. Elevate the affected part(s) with splinting as indicated.
   d. Administer antitetanus prophylaxis (toxoid or immunoglobulin).
   e. Analgesia: opiate, intramuscularly, or intravenously as indicated.
   f. Administer ibuprofen (400 mg orally every 12 hours).
   g. Administer benzyl penicillin (600 mg every 6 hours).
   h. Perform daily hydrotherapy for 30-45 minutes at 45°C.
4. For documentation, obtain photographic records on admission, at 24 hours, and serially every 2-3 days until discharge.
5. Prohibit smoking.

Source: Murphy JV, et al: *J Trauma* 48:171, 2000; adapted from McCauley RL, et al: *J Trauma* 23:143, 1983.

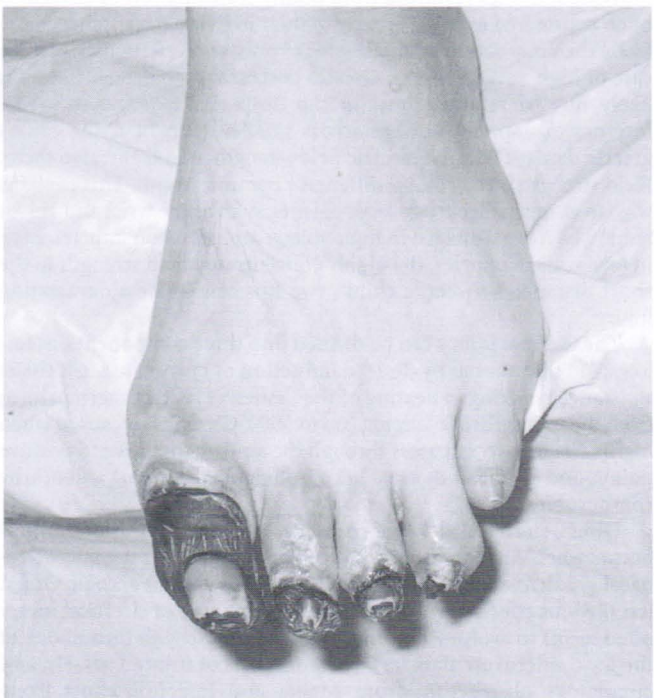

Figure 1 Demarcating deep frostbite, 2 months postinjury.

One must remain vigilant, however, that if evidence of infection develops around the eschar, such as ascending cellulitis or purulence, surgical débridement must be performed earlier than the proposed timeline.

Long Term

Long-term sequelae of frostbite include cold sensitivity, hyperhidrosis, arthritis, tissue loss, hyperesthesia, and chronic pain. Management may include calcium channel blockers for cold sensitivity and botulinum toxin for hyperhidrosis. Tingling may develop from ischemic neuritis, which can last for months. It may also predispose to chronic pain, which is often treated with gabapentin. Sunscreen should be used in the various areas because frostbitten areas may become hypopigmented or hyperpigmented related to inflammation. Heterotopic calcifications have also been noted.

Prevention

Prevention remains the watchword. Despite that hundreds of millions of people live in regions of the world with below-freezing temperatures at some portion of the year, frostbite or local cold injury is unusual because the ordinary denizen spends only 5% to 10% of the day outside of shelter. Thus it is the homeless, the outdoor sports enthusiasts, and members of the military who are at risk. Even in extremes of altitude, wind, and cold, such as in summitting the most inhospitable mountains on earth, frostbite has been avoided almost entirely. This is a testament to modern technologies, planning, and prevention.

Military principles to avoid morbidity from cold injury have been worked out, including education and use of multilayered clothing that traps air, creating insulation between each layer, topped off with an outer windproof or waterproof layer. Shoes must be well fitting; mittens are preferable to gloves. If feet become wet from sweating, they must be allowed to dry and new pairs of socks used. Thus prevention is critical via proper clothing, equipment, education, and instruction.

Suggested Reading

Britt LD, Dascombe WH, Rodriguez A: New horizons in management of hypothermia and frostbite injury, *Surg Clin North Am* 71:345, 1991.

Fritz RL, Perrin DH: Cold exposure injuries: prevention and treatment, *Clin Sports Med* 8:111, 1989.

Murphy JV, et al: Frostbite: pathogenesis and treatment, *J Trauma* 48:171, 2000.

Oumeish OY, Parish LC: Marching in the army: common cutaneous disorders of the feet, *Clin Dermatol* 20:445, 2002.

Reamy BV: Frostbite: review and current concepts, *J Am Board Fam Pract* 11:34, 1998.