Cold Injuries

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Introduction

Exposure to cold can produce various injuries that occur as a result of the human inability to adapt to cold.[1] These injuries can be divided into localized injury to a body part or parts (peripheral cold injuries), generalized cooling of the entire body (systemic hypothermia), or a combination of both. Among the civilian population, especially in densely populated cities, cold injuries are quite frequent, although sporadic. Unlike civilians, who spend 5-10% of each day outdoors, soldiers in tactical situations can be exposed to an extreme environment for 30% or more of each day, which may precipitate epidemics of cold injuries.

References

Systemic Hypothermia

Body temperature may fall as a result of heat loss by radiation, evaporation, conduction, and convection.[2] Radiation causes 55-65% of the body's heat loss. Evaporation occurs via the skin and airway and accounts for 30% of the heat loss. Normally, in a dry environment, only 15% of the body's heat loss results from conduction. However, the thermal conductivity of water is approximately 30 times that of air, so the body loses heat rapidly when immersed in water or covered in wet clothing, leading to a rapid decline in body temperature. Convection accounts for a minor amount of heat loss, but it becomes more significant in a windy environment. The amount of heat dissipated by any of these mechanisms is proportional to the temperature difference between the body and environment.

Opposing the loss of body heat are the mechanisms of heat conservation and gain. In general, a thermostat in the preoptic region of the hypothalamus controls these mechanisms. This human thermostat is set to a precise reference temperature, usually very close to 37°C (98.6°F). It responds to thermoregulatory mechanisms, the temperature of blood, and temperature receptors deep within the body and in the skin.

When the preoptic area of the hypothalamus is stimulated, various heat conservation and production mechanisms become activated. When the sympathetic nerves are excited, they cause the blood vessels in the skin to markedly constrict. The flow of warm blood from the core of the skin is depressed, thereby reducing the transfer of heat to the body surface. This reduction of blood flow in the skin is the prime physiologic regulator of heat loss from the body. The temperature of the skin decreases to approach the temperature of surrounding air, which lowers the temperature gradient and further decreases heat loss.

Stimulation of the sympathetic nerves also causes secretion of epinephrine and norepinephrine by the adrenal medullae. These hormones increase the metabolic rate of all cells, thereby enhancing heat production. Impulses from the preoptic hypothalamus also activate the primary motor center for shivering, which, in turn, increases the tone of muscles. The resulting enhancement of muscle metabolism can increase heat production by as much as 500%.

Causes

Hypothermia or systemic cold injury is a clinical condition in which the core body temperature has decreased to 35°C (95°F) or less. The causes of hypothermia are either primary or secondary. Primary, or accidental, hypothermia occurs in healthy individuals inadequately clothed and exposed to severe cooling.[3] Accidental hypothermia can be divided into immersion and nonimmersion cold exposure. The high thermal conductivity of water leads to the rapid development of immersion hypothermia. Although the rate of heat loss is determined by water temperature, immersion in any water less than 16°C (60.8°F) may lead to hypothermia within minutes.

When individuals are buried in the snow of an avalanche, they must be extricated from the scene of the avalanche accident as soon as possible.[4] In fact, rapid extrication is the most important determinant of positive outcome in snow avalanche victims. To facilitate the rapid localization of avalanche victims, avalanche transceivers are widely

used during off-piste and back country activities.

Hohlrieder et al conducted a retrospective study to analyze the influence of transceivers on the mortality of avalanche victims.[4] In the 194 accidents in Austria between 1994-2003, 278 victims were totally buried. Avalanche transceivers were used by 156 victims (56%), and transceiver use was associated with a significant reduction in the median burial time, which decreased from 102 minutes to 20 minutes ($P < .001$). Transceiver use was also associated with a significant reduction in mortality, which decreased from 68.0% to 53.8%. This reduction reflects a decrease in mortality during back country activities that involved ski tourists in free alpine areas. Transceivers did not significantly reduce mortality when they were used in off-piste activities beside or near organized ski slopes.

Even if a person is using a transceiver, mortality is significantly higher if burial depth exceeds 1.5 m. Despite a significant reduction in mortality, mortality still exceeds 50% even with transceivers. Consequently, even with the use of emergency equipment and life transceivers, avoiding avalanches is critically important. The authors conclude that the fairly modest influence of the use of transceivers on survival probability may also be due to the high efficiency of the mountain rescue service in the Austrian Alps.

In secondary hypothermia, another illness predisposes the individual to accidental hypothermia. The mechanism of secondary hypothermia appears to be an acute failure of thermoregulation; shivering does not usually occur in these patients. In many reports, alcohol seems to be a predominant cause of cutaneous vasodilation, loss of shivering, hypothalamic dysfunction, and lack of concern regarding the environment.[5] Other factors that predispose an individual to acute hypothermia include the following:

- Endocrine diseases (eg, hypothyroidism, pituitary insufficiency, Addison disease, diabetes mellitus[6])
- Cardiovascular diseases (eg, myocardial infarction,[7] congestive heart failure, vascular insufficiency)
- Neurologic diseases (eg, cerebrovascular accident, head injury, tumor, spinal cord injury, Alzheimer disease[8])
- Drugs (eg, phenothiazines, barbiturates, antidepressants)
- Pancreatitis
- Cirrhosis
- Hypoglycemia

Clinical presentation

Hypothermia affects multiple organs.[9] Initially, the metabolic rate increases, with tachycardia, tachypnea, increased muscle tone, and peripheral vascular resistance to generate maximal shivering. With continued hypothermia, the metabolism progressively declines, with bradycardia and hypoventilation and subsequent carbon dioxide retention. The heart rate drops to half its normal rate at 28°C (82.4°F), and ventricular contractility decreases. The risk of ventricular fibrillation increases at temperatures below 28°C (82.4°F). Cerebral metabolism is decreased 6-7% per 1°C drop in temperature, which results in a declining level of consciousness. Autoregulation of cerebral blood flow is impaired at temperatures below 25°C (77°F). The shivering mechanism of thermoregulation stops at 31°C (87.8°F).

The symptoms of hypothermia vary depending on the severity of the cold injury. In mild hypothermia, clinical symptoms are often vague and include dizziness, fatigue, joint stiffness, nausea, and pruritus. The skin is pale and cool as a result of peripheral vasoconstriction. The patient may exhibit lethargy, flat affect, impaired judgment, and mild confusion progressing to motor incoordination, ataxia, and slurred speech.

In severe hypothermia, mental status is further impaired, leading to hallucinations, stupor, and even coma. Atrial and ventricular arrhythmias are common with moderate hypothermia. The Osborn (J) point, an upward deflection at the junction of the QRS complex and the ST segment, can usually be seen on the ECG. The patient may appear clinically dead, with nonpalpable peripheral pulses, fixed and dilated pupils, loss of ocular reflexes, and stiff extensor posturing. Cardiac standstill usually occurs at 20°C (68°F), but one report described a survivor whose temperature was 15°C (60.8°F).

Diagnosis

The diagnosis of hypothermia is easy if the patient is a mountaineer who is stranded in cold weather.[10,11] However, it may be more difficult in an elderly patient who has been exposed to a cold external environment. In either case, the rectal temperature should be checked with a low-reading thermometer. The diagnosis of accidental hypothermia has proved elusive, largely because clinical thermometers do not record temperatures below 35°C (95°F). The only inexpensive low-reading thermometer is the Zeal (Zeal Group Ltd; London). Electronic thermometers with digital readouts and remote electric probes are made by several companies. Rectal temperature measurements are influenced by lower body temperature and probe placement. An inaccurate reading may result if the rectal probe was inserted in cold feces or to a depth of less than 15 cm.

Other methods of determining core body temperature include infrared tympanic thermometers, esophageal probes in intubated patients, and bladder thermistors embedded in a urinary catheter. The tympanic probe accurately measures hypothalamic temperature and most rapidly changes to reflect variations in core body temperature. On the basis of temperature measurements, the arbitrary classification of the level of hypothermia is mild (<34°C [93.2°F]), moderate (28-34°C [82.4-93.2°F]), and severe, with a boundary core temperature of less than 28°C (82.4°F).

Treatment

Field First Aid and Prehospital Management

The general principles of prehospital management are to (1) prevent further heat loss, (2) rewarm the body core

temperature in advance of the shell, and (3) avoid precipitating ventricular fibrillation. The application of these principles depends on the patient's core temperature, the equipment available, and the presence of complicating illnesses or injuries.

Mild hypothermia

For a person with mild hypothermia (≥ 33°C [91°F]), found in a cold environment, the first priority is to search for other injuries in that person and/or other affected individuals. The second priority is to increase the patient's core temperature to normal, before and during transport to the hospital. The patient should be moved into a tent or other dry shelter for protection from the wind. Wet clothing should be removed by cutting along the seams. Insulation, such as a sleeping bag, should be placed under and over the patient, who should not be allowed to stand or sit and whose head should be covered. A fire should be built or a stove lit. No fluids should be given by mouth. The patient should be transported to the hospital in an ambulance heated to 30°C (86°F).

Severe hypothermia

Severe hypothermia (<28°C [82.4°F]) should be treated as a life-threatening emergency. Attention should be directed first to the cardiopulmonary system. If the patient is breathing, humidified vented oxygen (10 L/min) should be administered using a nonrebreathing reservoir mask. If the patient is not breathing, ventilation should be initiated with a bag-valve-mask ventilator or a pocket mask connected to a humidified, heated oxygen delivery system.

The patient should not be hyperventilated because this may induce ventricular fibrillation. If bradycardia and hypotension are evident, do not perform cardiac compressions because this, too, may precipitate ventricular fibrillation. To avoid inappropriate chest compressions, prehospital personnel must examine the patient for a full minute before diagnosing pulselessness. If the patient is pulseless, external cardiac compressions with ventilations should be initiated unless the exterior wall is frozen and not compressible. Cardiopulmonary resuscitation should continue until the patient is evaluated and treated in the hospital.

Always follow the dictum that patients with severe hypothermia are never dead until they are warm and dead. Even with prolonged cardiac arrest, resuscitation is possible. Many physicians who are specialists in hypothermia believe that patients with severe hypothermia should not be rewarmed in the field, but, rather, kept in a "metabolic icebox" until in a hospital setting in which physiologic monitoring and advanced life-support equipment are available.

Hospital Treatment

Upon the patient's arrival in the emergency department, the physician must focus on cardiopulmonary function. If the patient is still not breathing, endotracheal intubation should be undertaken to maintain the airway. Frozen skin may require cricothyroidotomy. External cardiac compressions must be continued in pulseless patients with compressible chest walls. A central venous access line should be established to obtain blood for laboratory studies and to administer fluids for volume expansion. A venous cutdown may be necessary because vasoconstriction and hypovolemia may make percutaneous catheterization impossible. Central venous pressure monitoring is useful to determine the results of volume expansion in patients with severe hypothermia and hypovolemic shock.

Baseline laboratory determinations should include a complete blood cell count; levels of blood glucose, electrolyte, phosphorus, creatinine, amylase, lactic dehydrogenase isoenzyme, and creatine kinase isoenzyme; prothrombin time; activated partial thromboplastin time (aPTT); and arterial blood gas values. These laboratory studies should be repeated as indicated. A toxicologic evaluation is recommended for any patient whose history is unknown or who may have ingested a drug or poison. An initial 12-lead ECG should be obtained, after which the patient should have continuous cardiac monitoring. Intensely cold skin may preclude adequate transmission of electrical impulses to the electrocardiographic electrodes. A Foley catheter is introduced into the bladder to monitor urine output and to provide specimens for urinalysis and toxicology screening, including urine myoglobin levels.

(For detailed descriptions of catheterization, see Clinical Procedures articles Urethral Catheterization, Women, and Urethral Catheterization, Men.) Expired carbon dioxide analysis may help determine the level of asphyxiation.

Treatment of hypothermic patients should include volume expansion, cardiopulmonary support, and rewarming. Blood volume should be expanded using heated crystalloid solutions, such as dextrose in saline, to maintain blood pressure and coronary perfusion. In adults, 300-500 mL should be administered rapidly, with the subsequent rate of infusion adjusted according to the blood pressure. The solution should be warmed to 45°C (111°F) by heat exchangers or blood warmers. Fluid replacement may lead to increases in ventricular filling pressures, increased cardiac work, and pulmonary edema.

Supplemental oxygen is necessary to prevent hypoxia, reduce the risk of ventricular fibrillation, and treat pulmonary edema. Oxygen delivery to hypothermic patients should be maximized by the use of 100% oxygen during rewarming. Hyperventilation should be avoided because it may trigger ventricular fibrillation.

Although severe cardiac arrhythmias in hypothermic patients may represent an immediate threat to life, most rhythm disturbances (eg, sinus bradycardia, atrial fibrillation or flutter) require no therapy and revert spontaneously with rewarming. Ventricular fibrillation may be refractory to therapy until the patient is rewarmed to at least 34°C (93.2°F). At core body temperatures below 30°C (86°F), the heart is usually unresponsive to defibrillation, pacemaker stimulation, and cardioactive drugs.

Patients with ventricular fibrillation should receive 1-2 attempts at electrical defibrillation once the temperature is above 28°C (82.4°F). One dose of 10 mg/kg of bretylium (Bretylol) may be given; it is the only antiarrhythmic drug effective at low temperatures. If this is unsuccessful, cardiopulmonary resuscitation should be started or continued with active rewarming until the patient's core body temperature is above 32°C (89.6°F). As the myocardium warms, the rhythm may revert spontaneously or in response to electrical defibrillation.

The hypothermic heart is poorly responsive to the pharmacologic effects of medications. Excessive medication levels can accumulate as a result of decreased hepatic metabolism and increased protein binding, resulting in

toxicity when rewarming occurs. Therefore, nonessential medications should not be given until after rewarming. For information on various medication toxicities, see the eMedicine Emergency Medicine Toxicology section.

Coagulopathy is an underappreciated cause of morbidity in patients with moderate and profound hypothermia. Platelets are sequestered in frostbitten areas and in the lungs, causing thrombocytopenia and failure of platelets to clot. The aPTT becomes prolonged as core temperatures decrease. The combination of a prolonged aPTT and thrombocytopenia produces clinical disseminated intravascular coagulation. The passage of a nasogastric tube through friable nasal passages may produce torrential bleeding.

Rewarming Techniques

The 2 general techniques of rewarming are passive rewarming and active rewarming.[12] The capacity of humans with mild-to-moderate hypothermia to rewarm spontaneously after removal from the hypothermic condition accounts for the beneficial effects of passive rewarming. Because patients often become hypothermic over a period of days or hours, passive rewarming is physiologically sound, avoiding rapid changes in the cardiovascular status and the complications associated with active rewarming methods. Passive rewarming is a safe and simple method of treating mild hypothermia, and it is frequently the only method available for field management. Passive rewarming and noninvasive methods can also be used for patients with severe hypothermia (<28°C [91.4°F]) with a stable cardiac rhythm (including sinus bradycardia and atrial fibrillation) and stable vital signs. However, it is not recommended for patients with cardiovascular compromise.

Passive rewarming involves effective insulation of the patient, allowing the patient's spontaneous metabolic heat to rewarm the body. With this technique, the patient is covered with 1-2 blankets and rewarmed at a room temperature of 25-33°C (77-91.4°F). The increase in core temperature varies from 0.5-2°C/h; 24 hours may be required to achieve a normal temperature. If the increase in temperature is less than 0.5°C/h, the presence of a complicating disease, such as hypothyroidism, should be considered.

Active rewarming involves the internal or external addition of heat to the body. Active external rewarming works best for patients with mild or moderate hypothermia because it applies exogenous heat to the surface of the body in the form of warm packs, heating blankets, radiant heat, and warm water immersion. Concern has been raised about the efficacy of actively rewarming from the surface because of inherent physiologic changes that may aggravate the effects of hypothermia on dermal tissues that are poorly perfused because of vasoconstriction.

Active external rewarming may precipitate hypovolemic rewarming shock by decreasing the circulating blood volume secondary to peripheral vasodilation in an already hypovolemic patient. This peripheral vasodilation paradoxically causes central cooling by shunting stagnant, cold blood to the core, thus further chilling the myocardium, depressing contractility, and increasing its vulnerability to ventricular fibrillation.

The safest method of active rewarming of patients with severe hypothermia is internal rewarming that increases the core temperature. Active core rewarming should be used in patients with core temperatures lower than 32°C (90°F), who are hemodynamically unstable, or in whom more conservative rewarming methods have failed. Internal or core rewarming has the advantage of minimizing rewarming shock by warming the central core circulation first. Heat may be added internally by heated humidified inhalation, peritoneal dialysis, mediastinal irrigation, gastrointestinal tract irrigation, arterial venous shunting including hemodialysis, or extracorporeal bypass.

Airway rewarming at 40-45°C (104-113°F) prevents respiratory heat loss and raises body temperature 1-2°C/h. It also provides warm blood to the coronary arteries. In patients with severe hypothermia, air rewarming should be used as an adjunct to more rapid rewarming methods.

Peritoneal lavage with warmed potassium-free dialysate 40-45°C (104-113°F) is more effective, raising the body temperature 2-4°C/h. Irrigation of the stomach or colon with warm fluids produces minimal rewarming because the surface area available for heat exchange is small; also, this may cause mucosal sloughing in the very cold tissues. Arterial venous shunts and hemodialysis warm the blood directly but require cannulas to be inserted into arteriotomies.

Extracorporeal rewarming is the most rapid and efficient method of rewarming and is indicated in patients with cardiac arrest or impending cardiac arrest. Cutdowns may be necessary to place cannulas in the heart, aorta, or femoral vessels. A cardiopulmonary bypass circuit can achieve rewarming of 1-2°C every 3-5 minutes, but it mandates anticoagulation. Cardiopulmonary bypass resuscitation has been successful even after prolonged cardiac arrest unresponsive to other resuscitative and rewarming methods. Contraindications to the use of cardiopulmonary bypass for rewarming are severe brain injury, hyperkalemia (potassium level >7 mEq/L), and clotted or gelled blood in the arteries.

Gilbert et al reported the results of resuscitation of a 29-year-old skier who sustained accidental hypothermia after skiing down a waterfall gully.[13] She responded to a treatment using extracorporeal membrane oxygenator (ECMO) for 5 days. During that time, several organ dysfunctions developed that required hemodiafiltration and respiratory support, in addition to ECMO. Hemodiafiltration is an extracorporeal renal-replacement technique using a highly permeable membrane in which diffusion and convection are conveniently combined to enhance solute removal in a wide spectrum of molecular weights. Transitory hemorrhagic diathesis, atrophic gastritis, ischemic colitis, and polyneuropathy also occurred. At follow-up, 5 months after the accident, the patient had residual but improving partial pareses of the upper and lower extremities. Her mental function was excellent, and she was gradually returning to work. She also resumed hiking and skiing.[13]

ECMO has even been reported to be successful in near drowning associated with deep hypothermia. Thalmann et al described a case of near drowning of a 3-year-old girl who was admitted to the emergency department with a core temperature of 18.4° C.[14] After rewarming on cardiopulmonary bypass and restitution of her circulation, respiratory failure resistant to conventional respiratory therapy prohibited weaning from cardiopulmonary bypass. Consequently, the medical team instituted ECMO. Fifteen hours later, the patient could be weaned from ECMO but required assisted ventilation for 12 days. Twenty months later, no neurological deficits were in evidence.[14]

Cardiopulmonary bypass and ECMO are not available in hospitals without cardiopulmonary bypass capabilities. Winegard reported an instance in which closed thoracic cavity lavage was used successfully in the treatment of severe hypothermia.[15] In Winegard's patient, closed thoracic cavity lavage was initiated through a 28-mL straight chest tube inserted into the left pleural cavity. Closed thoracic cavity lavage was performed with a saline solution at a temperature of 40°C. This lavage was continued after the patient began to make spontaneous respirations and was successfully defibrillated. The patient was discharged 10 days after admission. Follow-up neurological examination revealed relatively minor neurological sequelae. Two months after the accident, the victim had minor numbness, mostly in his feet. This was considered a cold neuropathy rather than a pressure palsy neuropathy.[15]

Because a large proportion of hypothermic patients are thiamine-depleted and alcoholic, they should be given thiamine at 100 mg intramuscularly, followed by 50-100 mL of 50% dextrose. Administration of antibiotics, steroids, and thyroid hormones must be considered. Very cold patients are immunosuppressed, and antibiotics are usually withheld until a definite infection is evident. Hydrocortisone should be administered to patients with a history of adrenal suppression or insufficiency and to hypothermic patients with cachexia and/or generalized weight loss. Levothyroxine (Synthroid) is necessary for patients in a myxedema coma and may be helpful in elderly persons.

Peripheral Cold Injuries

The mechanisms of peripheral cold injuries can be divided into phenomena that affect cells and extracellular fluids (direct effects) and those that disrupt the function of the organized tissue and the integrity of the circulation (indirect effects).[16] Generally, no serious damage is seen until tissue freezing occurs. During frostbite, ice crystals many times the size of individual cells form from the available extracellular compartment, producing intracellular dehydration. The cell content becomes hyperosmolar, and toxic concentrations of electrolytes may cause cell death.[17] Usually, no gross rupture of the cell membrane is evident. A reversal of this process probably occurs during thawing of frozen tissues. After tissue thawing, vasodilation and leakage from capillaries occur, causing tissue edema. Alternating freeze-thaw cycles potentiate the vascular injury and lead to ischemic infarction.

The indirect effects of frostbite, a fulminating vascular reaction and stasis, are associated with the release of prostaglandins that have been implicated in progressive dermal ischemia. Both prostaglandin F2 and thromboxane A2 cause platelet aggregation and vasoconstriction. Therapy with antiprostaglandin agents and thromboxane inhibitors has been shown in experimental and clinical studies to increase tissue survival.

Frostnip

The mildest form of peripheral cold injury is frostnip, which tends to occur in apical structures (nose, ears, hands, feet), where blood flow is most variable because of the richly innervated arteriovenous anastomoses. Frostnip most often occurs in skiers exposed to fast-moving, very cold air. Simple warming either by pressure of a warm hand or by placing the hand in the axilla is sufficient treatment. More consequential local cold injuries may be divided into freezing (frostbite) and nonfreezing (chilblains and immersion foot) injuries. The diagnosis of freezing and nonfreezing injuries can generally be made on the basis of history and clinical manifestations.

Chilblain (Perniones)

Chilblain represents a more severe form of cold injury than frostnip and occurs after exposure to nonfreezing temperatures and damp conditions. This condition is characterized by a chronic, recurrent vasculitis manifested by red-to-violaceous raised lesions in unprotected extremities, such as the hands, feet, and face. Blisters, erosions, or ulcers are sometimes seen. It commonly affects young and middle-aged women and is associated with a variety of chronic disorders, especially Raynaud phenomenon.

The lesions usually resolve spontaneously in 1-3 weeks, but they may recur in some individuals. Management involves local heat, gentle massage, and lubricants to keep the skin supple. Nifedipine (Procardia) at 20-60 mg/d may be used to reduce the pain and speed the resolution of the lesions.

Immersion (trench) foot

Immersion foot, or trench foot, a disease of the sympathetic nerves and blood vessels in the feet, is observed in shipwreck survivors or in soldiers whose feet have been wet, but not freezing, for prolonged periods.[18] It may occur at ambient temperatures near or slightly above freezing and is usually associated with dependency and immobilization of the lower extremities with constriction of the limb by shoes and clothing. Immediate symptoms include numbness and tingling pain with itching, progressing to leg cramps and complete numbness. Initially, the skin is red; later, it becomes progressively pale and mottled and then gray and blue. The soles of the feet are wrinkled and very tender to palpation.

The progression of this cold injury has 3 stages. The first is a prehyperemic phase, lasting for a few hours to a few days, in which the limb is cold, slightly swollen, discolored, and possibly numb. Major pulses are barely palpable. The second, or hyperemic phase, lasts 2-6 weeks. It is characterized by bounding, pulsatile circulation in a red, swollen foot. The third, or posthyperemic, phase lasts for weeks or months. The limb may be warm, with increased sensitivity to cold. The injury often produces a superficial, moist, liquefaction gangrene quite dissimilar to the dry, mummification gangrene that occurs with severe frostbite.

Management of this injury entails careful washing and air-drying of the feet, gentle rewarming, bed rest, and slight elevation of the extremity. Improvement occurs within 24-48 hours, while the injury completely resolves in 1-2 weeks. Early physical therapy is essential. The patient should be warned that subsequent chilling will preferentially affect the previously injured area. Key to prevention of immersion foot injury is keeping the feet dry for at least 8 h/d.

Frostbite

Patients with frostbite frequently present with multisystem injuries (eg, systemic hypothermia, blunt trauma, substance abuse). The health professional must detect these multisystem injuries and appropriately triage care

according to the most life-threatening injuries.

Several days after the injury, frostbite can be classified into 4 degrees of severity. In first-degree frostbite, hyperemia and edema are evident. Second-degree frostbite is characterized by hyperemia and edema, with large, clear blisters that may extend the entire length of the limb, digit, or facial feature. Third-degree frostbite is characterized by hyperemia, edema, and vesicles filled with hemorrhagic fluid that are usually smaller than those of second-degree frostbite and do not extend to the tip of the involved digit. Fourth-degree frostbite, the most severe type, involves complete necrosis with gangrene and loss of the affected part.

The classification of frostbite into 4 degrees of severity is not favored by clinicians, who find assessing the full extent of tissue injury difficult in the acute setting. A simpler classification divides frostbite injury into 2 types: superficial or deep. Superficial frostbite (first- and second-degree frostbite) involves the skin and subcutaneous tissues. The skin is cold, waxy white, and nonblanching. The frozen part is anesthetic but becomes painful and flushed with thawing. Edema develops and clear bullae filled with serous fluid appear within the first 24 hours. Deep frostbite (third- and fourth-degree frostbite) involves the muscle, tendons, neurovascular structures, and bone, in addition to the skin and subcutaneous tissues. The frozen part is hard, woodlike, and anesthetic. It appears ashen-gray, cyanotic, or mottled and may remain unchanged even after rewarming. Edema develops, but bullae may be absent or delayed. Bullae, if present, are filled with hemorrhagic fluid.

Initially, as the tissue is freezing, the patient experiences discomfort or pain. This progresses to numbness and loss of sensation. Upon examination, the frozen tissue is white and anesthetic, owing to intense vasoconstriction. Tissues that remain frozen can appear mottled, violaceous, pale yellow, or waxy. Favorable signs include warmth, normal color, and some sensation. Several factors predispose to this cold injury. Clinical experience suggests that frostbite occurs at higher temperatures in patients with preexisting arterial disease.[19] In addition, a physiologic basis seems to exist for the reported susceptibility of black persons to frostbite. Finally, it has been demonstrated repeatedly that a person who previously suffered frostbite is more prone to develop this cold injury in the same body part than an individual with no history of such a cold injury.

Several principles of frostbite treatment are universally accepted. The patient must be removed from the cold environment. Treatment should not be attempted in the field if a hospital is available within a short distance or if a risk exists that the extremity will be refrozen. Once the rewarming process has begun, weight-bearing on the affected part is almost certain to result in additional injury. Rubbing the frostbitten part with snow or exercising it in an attempt to hasten rewarming is absolutely contraindicated. Contrary to popular belief, walking some distance on frostbitten feet can result in tissue fracture. Consequently, this ambulation should be avoided.

Upon arrival at the emergency department, normal body temperature should be restored before treating the local injury. The preferred initial treatment for frostbite is rapid rewarming in a water bath at a temperature of 39-42°C (102.2-107.6°F). Strict aseptic technique (eg, mask, powder-free gloves) should be used by all personnel during the warming procedure and during subsequent wound treatments. The rewarming bath should be large enough so that the frostbitten part does not rapidly reduce the temperature of the water. The temperature of the bath is monitored carefully as the bath cools. Additional hot water is added to the bath only after the extremity is removed from it. After hot water is added, the bath is stirred and the temperature retested before the extremity is reintroduced.

Rewarming is continued until the frostbitten tissue has a flushed appearance, demonstrating that circulation is reestablished. This rewarming procedure usually lasts 30-45 minutes. Because rewarming is painful, narcotics are often required. After rewarming, the skin is washed gently with a fine-pore cell sponge soaked in poloxamer 188 (Shur-Clens) to remove any residual dirt. The skin is then carefully dried with fine-pore cell sponges without surfactant. Tetanus prophylaxis is indicated with a tetanus vaccine (Td) without thimerosal because frostbite injuries are considered tetanus-prone wounds.[20] A vaccine information statement, which outlines the adverse effects of the vaccine, should be given to the patient or a family member, along with the National Vaccine Information Compensation Program.[21] The benefit of prophylactic antibiotics continues to be debated, and the authors reserve them for specific infectious complications.

Heggers et al recommended a therapeutic approach devised to prevent the progressive dermal ischemia of frostbite.[22] The combination of the systemic prostaglandin inhibitor ibuprofen (Motrin) and the topical antithromboxane agent aloe vera was used to inhibit localized thromboxane production, which had been implicated as the cause of dermal ischemia.[22]

Severe frostbite can have devastating consequences, including the loss of limbs and digits. One of the mechanisms of cold injury to human tissue is vascular thrombosis. The effect of tissue plasminogen activator and heparin in limb and digit preservation has been recently demonstrated by Twomey et al.[23] Patients with severe frostbite were divided into 2 groups. A group of 6 patients was treated with intra-arterial tissue plasminogen activator (TPA) and intravenous heparin. A group of 13 patients was treated with intravenous TPA and intravenous heparin.

Patients eligible for this study included all patients with severe frostbite between January 1, 1989 and February 1, 2003 whose symptoms were not improved by rapid rewarming, who had absent Doppler pulses in distal limb or digits, who did not have perfusion by technetium-99m three-phase bone scan, and who had no contraindications to TPA use.[23] Efficacy was assessed on the basis of predicted digit amputation before therapy, given the clinical and technetium-99m scan results versus partial or complete digits removed. Fortunately, no serious complications with intravenous TPA occurred. Two patients with intra-arterial tissue plasminogen activator experienced bleeding complications. On the basis of historical technetium-99m scan data, the investigators predicted which digits were at risk for amputation. In their study, 174 digits were at risk in 18 patients, and only 33 were amputated.

In 2007, Bruen et al further confirmed the reduction of the incidence of amputation in frostbite injury with thrombolytic therapy.[24] From 2001-2006, their patients with severe frostbite within 24 hours of injury underwent digital angiography and treatment with intra-arterial tissue plasminogen activator (tPA) if abnormal perfusion was documented. These patients were compared with those treated from 1995-2006 who did not receive tPA. In their study, 32 patients with digital involvement were identified. Seven patients received tPA (6 within 24 h of injury). The incidence of digital amputation in patients who did not receive tPA was 41%. In those patients who received tPA

within 24 hours of injury, the incidence of amputation was decreased to 10%. The authors concluded that the use of tPA represents the first clinically significant advancement in the treatment of frostbite in more than 25 years.

The clear blisters are debrided immediately, and the aloe vera (Dermaide Aloe) is applied directly to the debrided wound. In contrast, the hemorrhagic blisters are left intact and treated with aloe vera. The aloe vera is reapplied to the frostbitten wounds every 6 hours. When the hemorrhagic blisters rupture, the blisters are debrided, facilitating application of the aloe vera to the wound. Unless contraindicated by medical history (eg, aspirin allergy, peptic ulcer disease), ibuprofen (12 mg/kg/d for 1 wk) is administered orally to counteract the deleterious effects of increased thromboxane production. These treatments should reduce the length of hospital stay and the morbidity of patients with frostbite.

The affected part should be protected from trauma and infection, and it should be elevated above the patient's heart to minimize edema. A protective cradle should cover frostbitten lower extremities to prevent trauma. An environmental temperature of 21-26°C (69.8-78.8°F) in the hospital room is usually comfortable for the patient. Tobacco should be avoided because of its vasoconstrictive effect.

Patients with first- or second-degree frostbite of the feet should continue bed rest until the edema has receded and the vesicles and bullae have dried, which usually takes 2 weeks. Patients with more severe frostbite should remain in bed until wound repair is complete. Whirlpool treatments at 37°C (98.6°F) are instituted daily and should include active movement of all joints to maintain range of motion. Avoidance of joint stiffness and wound contraction is an essential goal of the rehabilitation program. The intrinsic muscles of the hands are particularly sensitive to frostbite injury. Night splinting of the hand in the intrinsic-plus position is recommended. Suppurative or loose necrotic debris should be removed by gently wiping the wound with the fine-pore cell sponge soaked in poloxamer 188.

The difficulty in determining the depth of tissue destruction has led to a conservative approach to the care of local cold injuries. As a general rule, amputation and surgical debridement should be delayed for 60-90 days unless severe infection with sepsis develops. The natural history of most injuries is one of gradual demarcation of the injured area, followed by dry gangrene or mummification of the area, with later sloughing of necrotic tissue, resulting in a viable, but shortened, extremity beneath the eschar.

Emergency surgery is occasionally required for patients with a frostbitten extremity. Open amputations are indicated in patients with persistent infection with sepsis that is refractory to debridement and antibiotics. Compartment syndrome may be encountered in a frostbitten extremity, which mandates fasciotomy.

The intense vasoconstrictive effects of increased sympathetic tone in cold injuries have attracted attention for many years. The theoretical benefit from sympathetic blockade is the release of vasospasm that may precipitate thrombosis in injured vessels. The vasospastic effects may, therefore, be counteracted by intra-arterial drugs, such as tolazoline, or by surgical sympathectomy. However, a controlled clinical study by Bouwman et al demonstrated no subsequent differences in the natural history of the acute frostbite injury between patients who underwent early operative sympathectomy within 20 hours of hospitalization and those who underwent intra-arterial drug infusion within the first hour of hospitalization followed by operative sympathectomy.[25] Apparently, the injury was well established by the time the patient sought help, so therapeutic manipulation of the microcirculation was too late to be of benefit.

Adjunctive Treatment

Multiple experimental therapies have been proposed for the treatment of frostbite. Antithrombotic agents such as heparin, dextran, and dipyridamole (Persantine), thrombolytic agents, calcium channel blockers, steroids, and hyperbaric oxygen have not been shown to enhance tissue salvage. Pentoxifylline (Trental), 400 mg every 8 hours, may aid small vessel perfusion. Phenoxybenzamine (Dibenzyline), 10-60 mg/d, may reduce refractory vasospasm in some patients.

Despite the lack of effectiveness in the acute phase, sympathectomy does appear to provide prophylaxis against the deleterious effects of subsequent cold exposure. This observation may have clinical implications in patients who need such prophylaxis for occupational reasons, such as professional skiers with cold sensitivity. In addition, the results of digital sympathectomy in the management of chronic vasospastic frostbite sequelae, such as vasospastic pain, are encouraging.

A body part that suffers frostbite seldom recovers completely. Some degree of cold sensitivity and hyperhidrosis are common. Neuropathies, decreased nail and hair growth, lymphedema, ulcerations, and persistent Raynaud phenomenon in the affected part are other residua of the injury. Permanent tissue damage, such as subcutaneous tissue atrophy, bony defects on x-ray examination, and abnormal epiphyseal growth, may occur. In children, the healed frostbitten hand frequently develops shortening of the digits, skin redundancy, joint laxity, and distal interphalangeal joint radial deviation. Surgical management options for the sequelae of frostbite of the juvenile hand include epiphyseal arrest, arthrodesis, and angular osteotomy.

Ophthalmic Injuries

Freezing of the corneas has been reported to occur in individuals who have kept their eyes open in high-windchill situations without protective goggles (eg, snowmobilers, cross-country skiers). Initial corneal flare and pain during rewarming are signs of this injury. Keratitis and corneal opacification may require corneal transplantation.

Snow blindness is produced by ultraviolet (UV) solar radiation reflected from snow, ice, or water. It tends to be more common at high altitudes, where the air filtration of UV radiation is diminished. Excess radiation produces corneal pitting and disruption of the epithelium. Retinal damage may also occur. Symptoms develop 4-12 hours after exposure and include a painful eye and excessive tearing. The lids and corneas of the eyes may be swollen and red. Treatment includes induced cycloplegia, mydriasis, and eyelid closure with a dressing. Artificial tears and antibiotics are indicated early in the treatment.

Scientific Basis for the Selection of Medical Gloves

When treating patients with cold injuries, the emergency medical technician or paramedic should wear powder-free emergency medical examination gloves with a glove hole leakage rate of 1%. In a hospital setting, the Food and Drug Administration (FDA) requires that examination gloves have a 4% glove hole leakage rate.[26]

In 2008, 13 health professionals filed a citizen's petition with the FDA to ban cornstarch powder on all medical gloves.[27] Cornstarch has been documented as promoting wound infection and causing serious peritoneal adhesions and granulomatous peritonitis, and it is a well documented vector of the latex allergy epidemic.

The FDA allows 1.5% of surgical gloves to have holes. These holes allow blood with deadly borne viral infections to be transmitted between the patient and the surgeon. Consequently, the Biogel double-glove hole indication system should be used to detect the location and presence of holes in the gloves, allowing the surgeon to change the gloves when a hole is detected.[28] After surgery has begun, one of the major causes of glove holes is surgical needle penetration, which can be detected by the double-glove hole indication system.

In collaboration with Dr. Robert Zura of Duke Medical Center, the authors have developed a poster entitled: A Demonstration of How the Biogel Patented Puncture Indication System Works When Immersed in Water or Saline.

Poster for the Biogel puncture indication system.

This poster can be framed and placed in the operating room as a guide to the appropriate use of the double-hole indication system during surgery. Copies of the poster can be obtained by contacting Mr. Milt Hinsch at Milt.Hinsch@biogelusa.com.

Multimedia

Media file 1: Poster for the Biogel puncture indication system.

(Enlarge Image)

Keywords

cold injury, frost bite, thermal injury, thermal injuries, cold exposure, peripheral cold injuries, systemic hypothermia, hypothermia, frost bite, frost nip, frostbite, frostnip, chilblains, immersion foot, trench foot, corneal freezing, frozen cornea, snow blind, snow blindness, transceivers, tetanus immunization, vaccine information statement, vaccine injury compensation program, thimerosal, extracorporeal rewarming, extracorporeal membrane oxygenator, cardiopulmonary bypass, closed thoracic cavity lavage, tissue plasminogen activator, heparin, technetium-99m three-phase bone scan, three phase bone scan, amputation of limbs and digits, amputation, limb amputation, digit amputation, finger amputation, toe amputation, foot amputation, powder-free double-glove hole indication system, double glove system

Acknowledgments

In trauma surgery, we are continually inspired by our beloved patients, such as Jamie Hunter, as well as her courageous parents John and Wendy Hunter, of Seattle, Washington, who have acknowledged to us and our country the importance of having an organized approach to trauma care in our nation that will save lives of those who are critically injured. This is our quest.

More on Cold Injuries

References