Cold Injury

Injuries caused by cold exposure can be systemic (as seen in hypothermia), regional (as in frostbite), or a combination. In this article, the authors discuss the degrees of hypothermia, the classifications of frostbite, and the treatment of both.

Throughout history, cold weather has played a role in the outcome of wars and military actions. It influenced military campaigns, such as those of Alexander of Macedon, and contributed to such notable defeats as Napoleon’s Army in Poland in 1812. World War II saw more than 90,000 U.S. troop casualties from the cold, over 12,000 of these from frostbite. Perhaps the most dramatic cases of freezing occurred in B17 and B24 bombers making high-altitude bombing runs over Germany. When attacked by fighters, the waist-port gunners opened their doors to fire and were exposed to very cold air rushing by at 200 mph. During the winter of 1943, frostbite caused more casualties in these bomber crews than all other sources combined. Cold resulted in over 9,000 casualties during the Korean War, most of these occurring in the winter of 1952.

Civilian cold injury, seen with alcoholism and drug abuse, is more common in the elderly. Hundreds of recreational boaters, merchant mariners, and commercial fishermen also are injured annually. Cold injury can be systemic (as seen in hypothermia), regional (as in frostbite), or a combination. There are several manifestations of regional cold injury that depend upon the temperature and humidity. Chilblain is caused by temperatures above freezing in the presence of high humidity. It is rarely a serious problem because the lesions are limited, superficial, and heal quickly. Trench foot or immersion foot occurs when wet feet are exposed to temperatures of 32° F to 50° F for long periods, usually greater than 12 hours. Frostbite results from tissue exposure to subfreezing temperatures.

Hypothermia

Hypothermia, or systemic cold injury, is a clinical state of subnormal central or “core” temperature. Because some controversy exists as to the best technique for arriving at this “core” temperature, for purposes of this discussion, a rectal temperature below 35° C represents clinical hypothermia. It should be noted that the usual clinical thermometer cannot register a temperature below 34.5° C. Consequently, a laboratory thermometer or an electrical thermistor should be used. When determining rectal temperatures, it is advisable to insert the probe at least 10 cm beyond the anal sphincter to provide an accurate reading of the core.

The first level of hypothermia (mild) occurs with core temperatures between 33° C and 35° C. Under these conditions, there is marked shivering unless hypoglycemia suppresses this response. The body attempts to support the core temperature by peripheral vasoconstriction, tachycardia, and increased cardiac output. Dehydration and an increase in hematocrit may follow an increase in urinary output known as cold diuresis. Individuals feel intense cold but remain conscious. They normally recover spontaneously due to the retained shivering response, and ther-
apeutic intervention for mild hypothermia need not be aggressive.

Moderate hypothermia, with a core temperature between 30°C and 33°C, suppresses the shivering reflex, resulting in muscle and joint stiffness. Central nervous system depression and loss of consciousness can occur when the brain reaches a temperature between 32°C and 30°C. Coordination and peripheral motor and sensory nerve function are impaired. The decreased heart rate, cardiac output, blood pressure, and diminished peripheral circulation make these parameters difficult to measure. Decreased cell metabolism diminishes the oxygen demand and contributes to respiratory slowing. The ECG begins to show bradycardia, increased PQ and QT intervals, broadened QRS complexes, nodal rhythm, second-degree atrioventricular block, and atrial fibrillation. Bowel peristalsis decreases, with frank ileus developing at about 28°C.

A wide gap of up to 20°C can exist between the core and skin temperatures. When the skin temperature reaches 12°C, vasodilatation occurs because of precapillary sphincter paralysis. This further decreases the core temperature, produces a flushing response, and probably is the cause of the “paradoxical undressing” that occurs in this state.

Severe hypothermia exists once the core temperature drops below 30°C. Pronounced bradycardia is accompanied by atrial fibrillation and premature ventricular contractions. Respirations may reach one to two per minute. Hypothermia of this degree shifts the oxyhemoglobin dissociation curve to the left. However, because of the marked acidosis that also develops, the net result is a right shift of the curve. The individual is comatose, and it is very difficult to determine whether he is still alive. Blood pressure and pulses are impossible to detect by the standard indirect methods. Pupils will not respond to light; muscle and joint stiffness may resemble rigor mortis. Because of these physical findings, no patient should be considered dead until rewarming has occurred.

Treatment of Hypothermia

Treatment of hypothermia is the restoration of all lost heat without precipitating additional or fatal side effects. Some authors suggest that hypothermia by itself need not be fatal above a core temperature of 25°C. Below 25°C, ventricular fibrillation may appear spontaneously, although this may not be fatal. There is evidence in dogs that ventricular fibrillation can be tolerated for periods greater than one hour if appropriate rewarming and defibrillation is initiated.

As rewarming occurs, there is a decrease in peripheral vasoconstriction, which may convert compensated hypovolemia into hypovolemic shock. Acidosis may result due to the more efficient removal of acid metabolites from the periphery. The risk of acidosis is increased if heat is applied directly to the peripheral tissue. A return of the tissue temperature to normal levels also increases the tissue oxygen requirements. If the cardiac muscle is cooler than the peripheral tissues, it may be difficult to sustain an adequate cardiac output.

An interesting phenomenon may occur when the patient is removed from the cold environment: the core temperature may decrease further. This “after drop” results from the peripheral tissues continuing to drain heat from the core. The major drop will occur in the first 15 to 20 minutes, and the core temperature will stabilize in about 30 minutes.

Hypoxia, metabolic acidosis, and hypotension should be treated with oxygen, intermittent positive pressure ventilation, sodium bicarbonate, and warm intravenous fluids. Respiratory problems may include aspiration pneumonitis, pulmonary edema, and thickened bronchial secretions. However, suctioning or intubation should be done very cautiously because of the increased risk of cardiac arrhythmias. Cardiac monitoring should...
be in place before any manipulations are initiated. The central venous pressure also should be monitored, but care should be taken to avoid contact of the catheter tip and the myocardium. Because of the hypovolemia, urinary output should be monitored with a catheter. Laboratory tests should include electrolytes.

Prophylactic antibiotics should be considered in view of the potential pulmonary problems as well as other injuries that may be present. The diagnosis of infection is difficult in the acute stage of hypothermia.

Active rewarming should continue until the core temperature reaches the 32°C to 34°C range. At that point, because of the risks of hyperpyrexia, active efforts should cease. In addition, because of the significant metabolic acidosis that can result from shivering during rewarming, phenothiazines should be given.

The actual rewarming techniques are either active (rapid) or passive (slow). No uniformity of opinion exists as to which is the best method, although intermediate rates of rewarming should be avoided. Passive rewarming allows the patient to rewarm himself with his own endogenous heat production alone, usually while at bed rest in a warm room. Rewarming occurs slowly with the core temperature rising at about 1°C per hour. If shivering develops, the rewarming rate may double. With severe hypothermia, it may require 12 to 24 hours to complete the passive rewarming process. Slow rewarming techniques usually avoid rewarming hypotension and "after drop" problems as well as allow for the gradual correction of fluid balance.

Rapid active rewarming attempts to minimize the time the individual is in the hypothermic state, quickly revitalizes a weakened and irritable myocardium, and mitigates the "after drop" by overwhelming the temperature gradients within the body with the application of heat. Active rewarming techniques are divided into surface or core procedures. Surface rewarming includes the use of warm blankets, hot packs, or immersion of the body into water between 40°C and 45°C. Core rewarming techniques include peritoneal dialysis, warm-water enemas, extracorporeal circulation, mediastinal lavage, gastric lavage, diathermy, and breathing warm air. Regardless of the techniques selected, resuscitation should never be terminated until complete rewarming has been achieved.

Frostbite

The basic mechanism by which cold injures tissues can be divided into direct effects on the cells and extracellular fluid and indirect effects on the circulation and organized tissues.

Direct injury results when bare tissue comes in contact with supercool liquid or metal. This causes immediate freezing of the skin and, depending upon the length and intensity of the exposure, the underlying tissue. More gradual freezing allows ice crystals to develop in the extracellular fluid, which then attract water from the cells, creating a hyperosmolar intracellular environment with resulting cell damage. Intracellular ice crystal formation and cold-induced shifts in the cell membrane lipid and phospholipid proportion also can result in the disruption of the cell membrane.

Indirectly, tissue injury can result from vascular stasis and hypoxemia. Cold exposure causes severe vasoconstriction and a decreased peripheral blood flow. The capillaries and arterioles can plug and eventually lead to thrombus formation. This causes vascular occlusion, tissue hypoxia, and ischemic necrosis. Structurally, the damage resembles a burn.

Frostbite Classifications

Two clinical classifications of frostbite exist, but unless the patient is seen before rewarming takes place, any classification can be applied only retrospectively. Washburn's division of frostbite into superficial and deep categories has the bene-
fit of simplicity. Superficial frostbite applies to a part that is white and frozen on the surface but feels soft and resilient when the examiner presses the tissue gently and firmly. Deep frostbite cannot be depressed and feels hard and solid. After the part has thawed, one cannot prognosticate.

The four-part classification can be applied only as the clinical course begins to manifest itself and cannot be used for prognostication. It is difficult to place most patients into a single category. First-degree cold injury consists of hyperemia and edema. Edema develops in three hours and lasts five to ten days. Sloughing of the superficial skin layers begins after about one week and may last one month.

Second-degree cold injury is associated with edema and hyperemia as well as blisters developing on the first day. The blisters eventually dry and form black eschars within two to four weeks. When the eschar sloughs, pink tender skin is left.

Third-degree cold injury consists of full-thickness skin injury with some extension into the subcutaneous tissues. Blisters appear at the edges of the third-degree injury, in areas of second-degree injury. The skin becomes a hard, black eschar, which takes two months to heal. If eschars are circumferential, they often produce a tourniquet effect.

Fourth-degree cold injury involves all tissues, including bone, with total destruction of the affected part. The part becomes black and dry and shrivels with demarcation in one to two months. The mummified part usually autoamputates after two to three months (Figure 1).

The Clinical Picture

Resistance to cold varies with the type of tissue. Although the skin, fascia, and connective tissues are resistant, bone and tendon are the most resistant. Nerves and striated muscles, in contrast, are highly sensitive. The individual affected by frostbite feels uncomfortably cold and later numb. This often is followed by a pleasant feeling of warmth. First the skin becomes red, then pale and waxy white. The initial stage of "frost nip" is reversible and treatment straightforward. If allowed to go unchecked, frostbite is inevitable and adequate treatment in the field impossible. The sudden blanching of the skin must be noticed promptly. Firm, steady pressure of a warm hand on the cold nose, cheek, or ear must continue until the color returns. Cold hands can be placed in the warm axillae. Cold feet can be placed against a warm abdomen or chest sheltered from the wind. Never rub or massage an extremity, and never apply snow.

Temperature, humidity, wind velocity at the initial exposure, and the time elapsed before adequate rewarming all affect the clinical result. The latter is the least important in terms of overall prognosis. It is often advisable to allow the patient to walk for hours on frozen feet if it facilitates his evacuation from a remote area. Once his toes or feet thaw, he becomes a stretcher case, and refreezing severely compromises the injured tissues.

Emergency physicians rarely see the dead, white, hard, brittle stage of frostbite from which some prognostication can be made. Wartime statistics indicate that less than 2% of cold injuries were seen by a medical officer before rewarming.

Previously significant episodes of cold injury, whether affecting the presently involved part or not, increase the risk of further cold injury. Individuals who are undernourished, elderly, or have other injuries resulting in decreased tissue perfusion (such as shock or peripheral vascular disease) are at an increased risk for frostbite.

The wind-chill factor accentuates or accelerates the development of frostbite. Heat loss by convection greatly enhances surface cooling. A temperature of 30°F with a wind speed of 25 mph has the same cooling effect on exposed flesh as a temperature of 0°F without wind.
Figure 1—Frostbite injury to the toes.

Treatment of Frostbite

The patient with frostbite must be removed from the cold as soon as possible. Rapid rewarming is the treatment of choice.\(^3\) It is better to wait to thaw a part than to do so incompletely or risk the chance of refreezing, even if this delays rewarming several hours. Decreased circulation has a significant effect on frostbite, and anything that can contribute to decreased circulation should be avoided, especially smoking. It is better to splint fractures in a well-padded device than to apply traction because the straps applying the force obstruct blood flow. Boots or shoes should be removed from any extremity with a fracture and replaced with several layers of blankets or other suitable material. Alcohol also should be avoided because the increased surface blood flow lowers the core temperature and contributes to hypothermia.

Upon arrival at an adequate facility, the patient should be treated for both frostbite and hypothermia. After all the clothes have been removed from the extremity, the affected part should be rewarmed rapidly by placing it into a large volume of water maintained at 104° F to 108° F (40° C to 42° C). Once the feet or toes thaw, walking should not be permitted. Rewarming can be very painful in the first few minutes and is worse in those with impaired circulation. Therefore, narcotics may be required. Rewarming of an extremity should not take over 20 minutes, and further immersion is of no benefit. Never rub or massage a frozen extremity, even after thawing. Rubbing the extremity with snow or exposing it to an open fire or dry heat has no place in the treatment of frostbite and actually increases the damage.

Although accurate assessment of the
extent of damage must await the full development of tissue reaction, certain features are good prognostic factors. They include early, large, clear blister formation, rapid return of sensation, return to normal temperature to the injured area, rapid capillary filling, and a pink skin color that blanches. In contrast, poor prognostic factors include hard, white, cold, and insensitive areas, cold and cyanotic parts without blisters, complete absence of edema, dark hemorrhagic blebs, lack of skin blanching, and development of systemic symptoms. A history of freezing, thawing, and refreezing of the injured part carries a guarded prognosis, as do lengthy exposures to cold and contact of unprotected skin with metallic or supercool liquid.

Patients with lower-extremity frostbite should be kept at absolute bed rest until all edema subsides and the blisters are completely dry. Upper-extremity injuries should be kept elevated above the heart at all times until a similar stage is reached.

Sterile precautions should be taken for any patient with blister development or worse features. Masks, gloves, gowns, and sterile sheets under the injured part lessen the risk of infection. Prophylactic antibiotics are unnecessary, but tetanus prophylaxis should be administered immediately. A cradle should support the sheets over all leg injuries. Dressings are seldom necessary, but when required, they should be loose, soft, and dry, never greasy or oily. Soft absorbent cotton between the fingers and toes is all that is
needed. Dressings should be changed only when they are very dirty. The room should be kept comfortably warm (72° F to 78° F) in order to maintain body warmth during convalescence.

Physical therapy should provide twice-a-day active range of motion for 20 minutes in a 37° C whirlpool solution of hexachlorophene and water, but passive manipulations should be avoided. These whirlpool treatments provide all the wound debridement necessary. All operative interventions should be avoided except for splitting circumferential eschars that impede circulation. Intact blisters should not be debrided, even if they decompress spontaneously. Any debridement exposes sensitive underlying tissue and increases the risk of infection (Figure 2). If infection develops, broad-spectrum antibiotics should be used until specific sensitivities are available. Smoking is prohibited until healing is complete.

Patience in operative management is very important. Even in cases of obvious deep injury undoubtedly requiring amputation, observation for one to two months frequently shows a considerable reduction in the amount to be removed (Figure 3). The diet should be high in protein with multivitamin supplements. Generally, the results are improved by protection of the extremity during and after rewarming, prevention of infection, and gentle active range-of-motion exercises in a whirlpool bath to maintain function.

Long-term follow-up of Korean War frostbite victims showed that even after 13 years, excessive sweating, pain, cold feet, numbness, abnormal color, and joint symptoms were reported. These symptoms were caused by cold exposure. Patients had persistent tissue loss, scarring, toe and fingernail abnormalities, pigment changes, and radiographic bone and joint changes. Other long-term complications of frostbite include destruction of the epiphyseal plates of the phalanges, stunted growth and mild flexion deformities, and loss of fingernails.27

Figure 3—Amputation should be delayed at least one to two months to allow for further recovery.

References