

Cold Exposure

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Since the homeostatic priority of the human system is to maintain core temperature, drops in ambient temperature will result in vasoconstriction and shunting of blood from the peripheral regions of the circulatory system to the central locations where vital organs reside. In dangerously cold situations, the temperature of peripheral tissues (mainly the extremities, nose, penis, and ears) will fall because heat distribution to these regions is diverted as the body attempts to protect the organs.¹ When the temperature in the peripheral tissues drops to or below freezing, tissue injury will occur.

Peripheral cold injuries are caused by freezing (frostbite and frostnip) or by non-freezing mechanisms (immersion foot). Dry, non-freezing injury also occurs with chilblain (pernio).

Frostbite occurs when tissue temperature falls below freezing. The tissue is injured by direct cellular damage from the formation of ice crystals and indirectly from blood clotting inside small blood vessels that feed the tissue.² Sensation is lost when the temperature in the skin falls below 10°C. As the temperature continues to fall, microvascular vasoconstriction becomes evident and plasma leaks from the blood vessels into surrounding tissue. Radiation and conduction of heat from deeper tissues prevent freezing until skin temperature falls below 0°C.³ As the temperature continues to drop, ice crystals form outside the cells. Water then shifts from inside the cells to the extracellular compartment in order to maintain osmotic equilibrium. Cell death occurs from direct damage to cell membranes by ice crystals and from dehydration and loss of cellular volume.⁴

As the tissue thaws, the red blood cells inside small venules and arterioles start coalescing and impede flow, ultimately leading to formation of thrombi.⁵ Vascular endothelial cellular damage, increased viscosity, and vasospasm of the vessels contribute to thrombosis. Hypoxia from impaired blood flow and arteriovenous shunting at the leading edge tissue damage results in extension of the tissue injury. Increased tissue pressure from plasma leakage exacerbates tissue ischemia and results in tissue necrosis.

Skin damage caused by freezing is partially reversible. Some tissue is revitalized during rewarming. Refreezing during this process will result in additional cellular damage. Further damage to tissues is mediated by chemicals such as thromboxane and prostoglandins released by injured and dying cells into the extracellular milieu.^{6,7} These chemicals promote platelet aggregation, vasoconstriction, and mobilization of the immune system.⁸ Swelling of the affected tissues continues for 2 or 3 days after tissues have thawed. Demarcation of viable and dead tissue becomes apparent as the edema resolves. This phase may last up to 3 months.⁹

In contrast to frostbite, non-freezing neurovascular damage within the tissue causes the injuries associated with immersion foot¹⁰ and from persistent vasospasm and vasculitis are causative in pernio.¹¹

Risk Factors

Multiple predisposing factors influence the extent of tissue damage from cold injury. Certain genetic variables may play a role in the tissue response to cold. For example, a strong reflex in Eskimos and Nordic populations is the “hunting response,”¹² in which periods of vasodilatation interrupt the severe vasoconstriction induced by low temperatures (<10°C) and thus protect peripheral tissues.¹³

A second cold insult may be more damaging than the initial exposure because the tissue's autonomic response has become impaired. Conditions that impair circulation (e.g., diabetes, atherosclerosis, dehydration, and hypotension) create a predisposition to cold injury. Chemicals that cause vasoconstriction, such as nicotine, can exacerbate cold injury.¹⁴ Reduced mobility, constrictive clothing, and dependency of extremities reduce heat distribution to the peripheral tissues.

Psychiatric conditions, medications, and intoxicants that impair judgment predispose people to cold injury.¹⁵ In addition to its intoxicating effects, ethanol causes peripheral vasodilatation, which in turn leads to increased heat loss.¹⁶

Freezing and Non-Freezing Injuries

The most common symptom of frostbite is numbness. There is also diminished sensation to touch, temperature, and pain modalities. Intense vasoconstriction causes ischemic neuropraxia. The distal extremities, nose, ears, and penis are at most risk.¹⁷ Complete anesthesia in a cold and painful extremity is a sign of severe frostbite. The initial presentation of the limb may be deceptively benign. The tissue appears mottled, yellow, or waxy-white. After rapid rewarming, the tissue will become hyperemic and, in most cases, the anesthesia will partially resolve until bullae form in 6 to 24 hours. With partial tissue damage, a dull ache transforms into a severe throbbing sensation within 2 or 3 days and lasts for up to 3 months until the extent of injury is clearly demarcated.⁹ Normal sensation and color and soft pliable skin at presentation indicate superficial frostbite injury, and favorable outcome. Early large, clear bullae formation is associated with a better outcome than late hemorrhagic blebs. The absence of edema within 3 hours

of thawing suggests severe injury. Also, with severe frostbite, early black eschar formation and clear demarcation of injury are possible.¹⁸

Frostbite may be classified according to the depth of tissue injury. First-degree frostbite involves the superficial skin and is characterized by erythema and anesthesia. Clear blebs are associated with second-degree injury. In third-degree frostbite, hemorrhagic vesicles indicate deeper subcutaneous injury. Finally, injury to muscle and bone are seen with fourth-degree frostbite. The extent of injury and accurate classification of frostbite is not typically possible during the initial presentation of the patient and may lead to misleading assumptions; therefore, a simplified two-class system may better suit emergency physicians and prehospital care providers. This classification simply differentiates mild superficial injury (which does not lead to tissue loss) from severe deep injury (which does lead to tissue loss).⁹

Frostnip is characterized by transient numb and tingling sensation, which resolves after warming and does not lead to permanent tissue damage.¹⁹

Pernio is associated with repetitive dry cold exposure of the skin. It is characterized by erythema; itching; and mild swelling on the face and the dorsal surfaces of the hands and feet, which may lead to the development of plaques, nodules, or ulcerative lesions.²⁰ Women with Raynaud's phenomenon or other conditions causing vasospasm are at higher risk for developing pernio. Persistent and refractory cases of pernio may be treated with nifedipine, a calcium channel antagonist.²¹

Immersion foot (trenchfoot) is a non-freezing cold injury that results from repetitive or continuous exposure of tissue to moist cold at temperatures above 0°C. Poor foot hygiene during prolonged expeditions or outings with this type of exposure is a

common cause of trenchfoot. The presenting signs and symptoms include cool, pale, numb feet. The extremity later appears cyanotic and edematous and remains cold. After rewarming, the skin becomes erythematous, dry, and exquisitely tender. When lifted, the extremity turns pale because of injury to the vasomotor apparatus. Vesicles resembling those seen in frostbite may appear. Vesicle formation may progress to ulceration and gangrene.²² Pain while walking and cold sensitivity may last for years after injury. Periodic inspection of the feet and frequent change to dry socks are preventative measures.²³

Stabilizing Treatment for Cold Injury

After addressing possible life-threatening conditions such as systemic hypothermia and concomitant injury, priority is given to rewarming the extremity or body part involved. Time is important because the longer the extremity is frozen, the greater the permanent damage to tissue. Out-of-hospital personnel should not attempt to rewarm the extremity in the field, unless evacuation is significantly delayed and refreezing is preventable. Incomplete thawing or refreezing of a thawed extremity leads to additional tissue injury.²⁴ Even if the circumstances exist for field rewarming, the technique is often not practical. In the field, all constrictive items should be removed, and the affected body part should be kept dry, insulated, immobile, and protected from trauma. Friction massage, an earlier form of recommended treatment, should not be performed because of potential injury to the skin and underlying tissues. The extremity should be kept away from sources of heat, such as kerosene heaters, field stoves, and heating devices in vehicles, which may cause partial thawing.²⁵ If possible, intravenous crystalloid fluids

should be started in the field and a bolus of 1 liter normal saline should be initiated if there are no contraindications.

On arrival at a treatment facility or emergency department, rewarming of the extremity should be initiated immediately by immersing the extremity into a bath of circulating water maintained precisely between 40 and 42°C, for about half an hour or until the extremity becomes pliable and erythematous. The patient may be encouraged to gently move the distal parts of the extremity during the rewarming process. Analgesia will often be required during the rewarming phase and afterward, because of the severe pain associated with reperfusion of tissues. Sensation is blunted with the formation of bullae. Parenteral analgesia should be started before rewarming to prevent premature removal of the limb from the bath as a response to pain.

After rewarming, the extremities should be covered loosely with sterile dressing. Constricting dressings should be avoided. The extremity should be elevated above the level of the heart to reduce edema, and the patient should be encouraged to move the distal extremity to prevent venous stasis; however, weight bearing on the extremity should be avoided until swelling has resolved completely. The patient should abstain from smoking tobacco and using illicit drugs such as cocaine, which can cause severe vasoconstriction.

There is controversy regarding the management of bullae and blisters. Intact blisters can be debrided, aspirated, or left intact. There are advantages to each management strategy, and the technique used should be left to the discretion of the treating physician.⁹ The American College of Surgeons Advanced Trauma Life Support

curriculum advocates leaving uninfected blisters intact for 7 to 10 days to provide a “biological dressing.”²⁶

The team managing the patient with frostbite injury should be on the lookout for compartment syndrome, which is caused by severe swelling within the fascial compartments of an extremity. This complication leads to diminished circulation to the distal extremity as a result of the constrictive pressure within the tensely swollen compartment. If necessary, compartment pressures should be monitored and pressure should be released with fasciotomies (a procedure rarely necessary in the early acute stages of cold injury). Since tetanus has been reported after frostbite, booster or primary vaccination should be provided.²⁷ Although the use of prophylactic antibiotics against staphylococci, streptococci, and *Pseudomonas* species has been advocated by some authors,¹¹ the use of antibiotics in the absence of infection should be avoided.

Demarcation of injury may take weeks in some cases, and the extent of true tissue injury may not be known until then. Surgical debridement or amputation should be postponed until the time of definitive demarcation, unless infection or sepsis is evident.

Many experimental treatment modalities have failed to show benefit in human frostbite injury. Some of the adjuvant treatments that have been studied include sympathetic blockade,²⁸ vasodilators, anticoagulants,²⁹ thrombolytic agents,³⁰ and hyperbaric oxygen therapy.³¹

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