Frostbite is tissue damage caused by localized hypothermia. To be more precise, frostbite is a clinical situation where water molecules freeze and crystallize within biological tissue, causing cellular and tissue death. Frostbite occurs when skin is exposed to a temperature below minus ten degrees Celsius, resulting in vasconstriction. The resultant decrease in blood flow caused by the vasoconstriction does not deliver sufficient heat to the tissues to prevent the formation of ice crystals inside the tissues.

There is a clinical spectrum of tissue injury due to cold. Frostnip is a mild form of cold injury that reverses itself upon warming. Chilblains (sometimes called pernio) is also a milder form of cold injury. Chilblains is a painful, abnormal reaction of small blood vessels in the skin when exposed to cold. Chilblains occurs several hours after exposure to cold in a temperate, humid climate. Frostbite is the most severe condition in the spectrum of cold injury.

Trench foot results from a prolonged exposure to a non-freezing wet and cold environment. Neurovascular damage occurs without ice crystal formation and manifests as pallor, pulselessness and paralysis. Trench foot is reversible and has a better prognosis than frostbite.

Prior to the 1950’s, frostbite was mostly limited to the world’s military. Today, there are new circumstances that make it important for physicians to be aware of the diagnosis, pathophysiology and treatment of frostbite. There has been an increase in the homeless population who experience prolonged exposure to cold temperatures. There has also been an increase in the number of people participating in outdoor activities in cold weather. There are new causes of frostbite. These include ice-pack burns.

**FIGURE 1**

**Characteristics of the Degrees of Frostbite**

- **FIRST DEGREE**
  Anesthetic central white plaque with peripheral erythema

- **SECOND DEGREE**
  Blisters filled with clear or milky fluid, surrounded by erythema and edema, appearing in the first 24 hours

- **THIRD DEGREE**
  Hemorrhagic blisters that result in a hard black eschar seen over the course of 2 weeks

- **FOURTH DEGREE**
  Produces complete necrosis and tissue loss

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recreational use of nitrous oxide, handling of liquid nitrogen, misuse of fluorinated hydrocarbon propellants and working with pressurized liquid ammonia.²

Classifications of Frostbite
Frostbite differs from milder forms of cold injury because frostbite causes tissue destruction. Frostbite is classified by the degree of tissue damage and divided into four categories (Figure 1).²

Superficial vs. Deep Frostbite Injury
Frostbite injury is sometimes classified as being superficial or deep (Figure 2). Superficial injury is equivalent to first degree

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and second degree injury. Superficial injury always precedes deeper injury. There is a white, mottled appearance to the skin. Initial numbness changes to burning and stinging with re-warming. If blisters occur, they are clear. Neurovascular dysfunction is reversible and tissue loss is minimal to non-existent. Deep injury involves skin, subcutaneous tissue, muscle, tendons, and bone. Deep injury is equivalent to third and fourth degree frostbite. The tissues involved remain mottled and pulseless after re-warming. Blisters are infrequent, but when they do occur, they are hemorrhagic. Tissue loss is inevitable. There is a high risk for infection because of the presence of devitalized tissue.

Physiology of Cold Injury

There are four ways that the body loses heat to the environment, through radiation, conduction, convection, or evaporation (Figure 3).3

With radiation, loss of heat to the environment is due to the temperature gradient between the body and the environment.

In conduction, heat loss is due to direct contact between the two objects; it is a molecular transference of heat energy. Water conducts heat away from the body 25 times faster than air because it has a greater density and therefore a greater heat capacity. Getting wet increases the rate of frostbite. Steel conducts heat away from the body even faster than water does.

Convection is a process of conduction where one of the objects is in motion. Molecules on the surface of the skin are heated, move away, and are replaced by new molecules that are warmed. The rate of convective heat loss depends on the density of the moving substance and the velocity of the moving substance. Wind chill is an example of air convection.

In evaporation, heat is lost from converting water from a liquid to a gas. Perspiration is the evaporation of water to remove excessive heat.

Fluid Loss

It is important to recognize that there is a strong connection between fluid loss, fluid levels and heat loss. As body moisture is lost, circulatory volume is reduced and dehydration results. Decreased fluid levels make the body more susceptible to hypothermia and cold injuries.4

Heat is required and produced at the cellular level. The hypothalamus is the major center of the brain for regulating body temperature. It is sensitive to temperature changes in blood and reacts to nerve impulses from nerve endings in the skin. The optimal temperature for chemical reactions to occur in the body is 98.6 degrees F. Above 105 degrees F, enzymes become denatured. Below 98.6 degrees F chemical reactions slow down, causing complications that can lead to cell death.

The core temperature (core = internal organs) is the temperature that is essential to the overall metabolic rate of the body. The temperature of the periphery (appendages, skin and muscle tissue) is not important.

Hypothermia

Hypothermia is a decrease in the body core temperature

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to a level where normal muscular and cerebral functions are impaired. Mild hypothermia occurs at core temperatures of 98.6-96 degrees F., moderate hypothermia at core temperatures of 95-93 degrees F., and severe hypothermia at core temperatures from 92-86 degrees F. and below.1

Symptoms of mild hypothermia are shivering, and inability to perform complex motor functions; there is vasoconstriction to the periphery. In moderate hypothermia there is dazed consciousness, loss of fine motor coordination, slurred speech, violent shivering, and irrational behavior. Severe hypothermia is life-threatening, with violent shivering in waves that stops when muscle glycogen is depleted, the person falls to the ground in a fetal position to conserve heat. Muscle rigidity develops with pale skin and dilated pupils, and pulse rate decreases. At 86 degrees F., the body tries to hibernate, shutting down peripheral blood flow, reducing the breathing and heart rates. The person looks dead but is still alive. Cardiac ar-

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rhythmias develop, ventricular fibrillation follows, the heart stops, and the body dies.3

Cutaneous Circulation

Cutaneous circulation is a significant influence in the thermal homeostasis in humans. Humans acclimate better to heat than to cold because the skin loses heat more easily than gaining heat. Shunting of blood preserves core temperature at the expense of peripheral tissue circulation. Some structures, such as fingers, toes, ears, and nose, contain multiple arteriovenous anastomosis that allows shunting of blood. These structures are most at risk for frostbite.4

In addition to the skin's natural heat conductivity and loss of heat from the evaporation of sweat, convection from cutaneous vessels is a vital component of thermoregulation. Cutaneous blood flow is 10-20 times that required for essential oxygenation and metabolism. Large amounts of heat can be exchanged through the regulation of cutaneous blood flow. The thermoregulatory center in the hypothalamus controls vasoconstriction and vasodilatation of cutaneous vessels through the sympathetic nervous system.3

Normal cutaneous blood flow is 200-250 mL/min (Figure 4). At 15 degrees centigrade, maximum vasoconstriction has been reached and the pattern changes into the “hunting reaction.” In the hunting reaction, vasoconstriction is interrupted by rhythmic bursts of vasodilatation that occur 3-5 times per hour and last 5-10 minutes. In persons acclimatized to cold, these bursts are more frequent and last longer, giving acclimatized individuals an increased resistance to frostbite injury. At 10 degrees centigrade, neuropraxia occurs, which is a loss of cutaneous sensation. Below 0 degrees centigrade, there is negligible cutaneous blood flow and the skin freezes. Smaller blood vessels begin to freeze before larger ones, and the venous system, having a lower flow rate, freezes before the arterial system.3

Pathophysiology

There are three distinct processes that occur in frostbite injury. There is extracellular and intracellular ice crystallization, intracellular dehydration and a vascular process of arterial insufficiency with intermittent spasm.1

The initial injury to the tissues is caused by formation of extracellular ice crystals, which damage the cell membranes, initiating a cascade of events that lead to cell death. As the tissues continue to freeze, osmolarity changes due to water shifting out of the cells into the extracellular spaces. The cells dehydrate and intracellular crystals are formed. These intracellular crystals expand and mechanically damage the cell, resulting in irreversible changes.

When the hunting reaction stops as the temperature gets colder, vasoconstriction persists. This leads to hypoxia, acidosis, arteriolar and venular thrombosis and ischemic necrosis. There is a difference between quick freezing and slow freezing. Rapid freezing increases intracellular ice formation.

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Slow freezing causes equal extracellular and intracellular freezing, resulting in more extensive cellular damage. Extracellular freezing progresses more rapidly than intracellular freezing.¹

The last process of cold injury is the vascular process, which consists of atrophic shunting, coagulation, thrombus formation, hypoxia, anaerobic metabolism, and tissue death.²

The Three Zones of Frostbite

The tissue injury of frostbite is divided into three zones (Figure 5).³

Zone 1: Zone of Coagulation. This is the area of most severe tissue damage. It is the most distal region of damage. There is irreversible tissue death.

Zone 2: Zone of Stasis: This is the middle region. It is characterized by severe tissue damage that may be reversible.

Zone 3: Zone of Hyperemia: This is the most proximal zone with the least amount of tissue damage. Recovery of this region is expected in ten days.

Slow vs. Rapid Re-warming

When external warmth is applied, perfusion from deep vessels returns slowly. The re-warmed tissue has an accelerated oxygen demand. In slow re-warming, ischemia results because perfusion from deep vessels returns too slowly relative to the increased oxygen demand of the re-warmed tissue. Rapid re-warming minimized this discrepancy and is therefore preferred to slow re-warming of frostbitten tissue. Re-freezing and re-thawing of frozen tissue causes great ischemic injury for the same reasons as slow re-warming.¹

Post-Re-warming Injury

Re-warming edema appears within three hours of applying heat and lasts for one week. Large, clear blebs occur within 6-24 hours with superficial injuries, and small hemorrhagic blebs appear 24 hours after deep injury. An eschar forms in 9-15 days. This is described as a “shrunken black carapace shell” covering the wound.¹ If the frostbite was superficial, new skin will appear below this carapace. If the injury is deep, the area will self-amputate. In three to six weeks mummification occurs and causes a demarcation line between live and dead tissue.

Medical Therapy

Lab Studies

Frostbite is a clinical diagnosis.³ Laboratory studies are done to identify infection and systemic complications.

Field Management

According to U.S. army protocol, transporting the patient to a burn or trauma center is a priority. Field re-warming should be started only if it will take more than two hours to transport the patient to a definitive treatment center.² The extremity should be dressed in a way to avoid mechanical trauma. Re-warming should be avoided if it cannot be maintained. The freeze-thaw-freeze cycle causes an increase in tissue damage.

Any systemic hypothermia must be corrected to a core temperature of 34 degrees C. before treating the frostbite.³

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**Re-warming**

The extremity should be rapidly re-warmed in circulating warm water at 40-42 degrees C. (104-108 degrees F.) for 15-30 minutes or until thawing is complete by physical assessment. With circulating water, constant temperature is applied to the entire affected area. This ensures that maximum viable tissue is preserved. Other methods of re-warming can cause greater tissue damage. Massaging, rubbing with ice or by hand causes mechanical trauma to the frozen tissues. Re-warming at higher temperatures or for longer periods of time also causes more tissue damage. Direct heating with a heater can cause burns secondary to the loss of temperature sensation caused by the cold injury. Partial thawing and refreezing (called refreezing injury) generates more damage than prolonged freezing. Thawing takes about 20-40 minutes for superficial cold injuries, and can take up to one hour for deeper injuries. Treatment is complete when the distal area of the extremity is flushed, soft and pliable. Encourage active, gentle motion of the frostbitten

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area during re-warming. A common error is premature termination of the re-warming process.

After Re-warming
1) Aspirate or debride white or clear blisters and use topical aloe vera every six hours.
2) Leave hemorrhagic blisters intact. This helps avoid dessication and infection of deep layers.
3) Elevate affected parts.
4) Appropriate Antitetanus prophylaxis
5) Ibuprofen 400 mg PO every 12 hours
6) Appropriate antibiotics
7) Daily hydrotherapy for 30-45 min at 40 degrees C.
8) Serial photographs
9) Prohibit tobacco
10) Manage fractures or dislocations conservatively until thawing is complete

Investigational Therapy
There are many ancillary modalities, but there are no controlled human studies to justify their use.

Conclusion
A thorough understanding of the physiology of cold injury and frostbite will help the medical practitioner in making treatment decisions. Frostbite requires special care. Standard first aid may cause more damage than help.

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