AMERICAN HEALTH CONSULTANTS® A Medical Economics Company

PEDIATRIC

Emergency Medicine Reports™

The Practical Journal of Pediatric Emergency Medicine

Volume 4, Number 1 January 1999

Frostbite injuries are uncommon in pediatrics, but have the potential to be devastating. Once a child develops frostbite, it is critical to maximize tissue preservation in the extremity. The adage "do no harm" applies well to this clinical situation where "well meaning" therapies may further exacerbate tissue loss. The emergency department (ED) physician must be well versed in therapies to be avoided, as well as in therapies that must be initiated in a timely fashion.

—The Editor

Local Cold Injuries in Children: Diagnosis, Management, and Prevention

Author: **Charles Stewart, MD, FACEP,** Emergency Medicine, Colorado Springs, CO.

Peer Reviewer: **Steven Winograd, MD, FACEP,** Attending Physician, Department of Emergency Medicine, Lakeland Regional Health System, St. Joseph, MI.

Introduction

Frostbite is thought to be uncommon in civilian pediatric practice.[1] These injuries are not often reported and may rarely be seen by an emergency physician. There are no available statistics that document the multitudes of minor cold injuries. There is little in the literature that specifically addresses the sequelae of these injuries in children.

The popular image of the cold injury amputee as the intrepid explorer, cross-country trekker, or climber caught in a blizzard is certainly not the case in urban environs.[2] Children may be exposed to cold weather by parents who exhort them to perform in cold weather sports.[3-5] Local cold injuries can occur at temperatures both above and below freezing and can cause permanent disability and disfigurement. Urban frostbite victims may have significant underlying psychosocial pathology that can interfere with rehabilitation efforts. These diseases range from alcoholism to drug dependence to suicidal ideation. Other (often well meaning but uninformed) parents expose children in carriers, sleds, and snowmobiles . . . often without appropriate protective clothing.

The kind of injury depends upon the degree, nature, and the duration of exposure to the cold. Frostbite is not the only cold-related injury. For practical purposes, cold injuries may be divided into "freezing" and "non-freezing" types. Frostbite, either superficial or deep, is an example of the former. The non-freezing types include chilblain, trench foot, and immersion foot.

Non-Freezing Cold Injuries

Chilblain (Pernio). Chilblain is a neurocirculatory skin disturbance that is primarily seen on the face, feet, fingers, and ears. It is most common in young women who spend long periods outdoors in cold weather. Chilblain (pernio) is the mildest form of cold injury, and may be divided into acute and chronic forms.

This injury occurs with repeated exposure of bare skin to cold, wet, and windy weather ranging from 0-15.5°C (32-60°F). Chilblain is more commonly recognized in England than in the United States.[6]

In the acute case, there is usually moderate to severe pruritis with red, dry, and roughened skin. Erythema, cyanosis, and edematous patches are seen 12-24 hours after exposure to the cold. The lesions are usually bilateral and symmetrical, although they may be single or multiple. Patients frequently describe a burning sensation, which is particularly noticeable upon rewarming the areas. After rewarming, tender bluish nodules may develop.

These nodules may persist for 10-14 days. This condition is frequently seasonal, starting with the onset of cold weather and clearing up when warm weather returns.

Chronic pernio is not usually a child's disease. It appears to occur more frequently in middle age, and may evolve because of a continued exposure to the cold in patients with acute chilblain. Symptomatic complaints are the same as in acute pernio. Numerous clinical variants of chronic pernio have been described. Lesions may be annular, papular, or, rarely, hemorrhagic bullae and pustules. Scarring and postinflammatory pigmentation changes are common. Healing occurs during the summer months.

Histologically, chilblain is characterized by edema of the papillary dermis and a mild vasculitis of both the superficial and deep dermal vessels. Histological changes strongly favor vascular damage as the primary etiologic factor in the formation of the perniotic lesions.[7] It is associated with increased vascular tone, which results in prolonged vasospasm even after initial warming is begun. Patients with Raynaud's phenomenon and those who take sulindac appear to be predisposed to chilblain.[8] A differential diagnosis for pernio or chilblain may include Raynaud's disease, lupus erythematosis, cellulitis, frostbite, herpes, traumatic injury, chronic myelocytic leukemia, vasculitis, and vascular thrombosis.[9,10]

To treat chilblain, the child should wear protective clothing and the parents should apply a soothing topical lotion to the affected areas. For severe cases, avoidance of cold weather areas may be needed. Acute chilblain is self-limiting, and the lesions do not recur unless re-exposure to cold occurs. Severe cases may demonstrate vesicles, bullae, petechiae, hemorrhage, and ulcers (although this presentation is rare).

Trench or Immersion Foot. The term "immersion foot" was coined during World War I as a description of those patients who were shipwrecked and isolated in a lifeboat for a prolonged period of time. The patients would have their feet in a dependent position in the bilge water. The water temperatures were usually between 0°C and 10°C (32°F and 50°F), and the exposure often lasted many days. The term "trench foot" was applied to a similar situation when the infantry would be pinned down in trenches filled with cold mud and water.

Although once thought to be different entities, trench foot and immersion foot are indistinguishable. They both occur after prolonged exposure to wet and cold. The ambient temperature usually ranges from just above freezing to about 50°F.[11] The duration of exposure to the wet and cold usually exceeds 10-12 hours. If the extremity is kept in an immobile and dependent position, the depth and extent of injury will be exacerbated. These are impressive conditions in children, and immersion injuries are quite unusual in the civilian pediatric population. Only conditions such as springtime floods, waterfowl hunting, refugee situations, and shipwreck would bring this disease to the typical pediatric population.

There are three phases to this type of injury.

First, the tissue becomes vasospastic and appears cold, swollen, white, or cyanotic. Pulses and sensation in the extremity are decreased. This initial phase results in tissue hypoxia leading to increased capillary permeability and edema.

Warming the extremity initiates the second phase, a hyperemic response where the extremity appears hot, dry, and reddened with bounding pulses and extreme pain. Bullae are also common. This phase may last from 4 to 10 days. Severe or repeated exposures can result in gangrene and extensive tissue loss. In many cases, after the patient has been rewarmed, it is difficult to distinguish the effects of trench foot from frostbite.

The third and last phase is one of recovery. It is characterized

Table. Prognostic Signs

Favorable	Unfavorable
Sensation intact	No sensation
Normal color	Cold, cyanotic appearance
Warm tissues	White "frozen" appearance
Early appearance of clear bullae	Late appearance of dark bullae
Edema	Absence of edema

Pediatric Emergency Medicine Reports™ (ISSN 1082-3344) is published monthly by American Health Consultants, 3525 Piedmont Road, N.E., Six Piedmont Center, Suite 400, Atlanta, GA 30305. Telephone: (800) 688-2421 or (404) 262-7436.

Publisher: Brenda Mooney
Executive Editor: Park Morgan
Managing Editor: David Davenport
Copy Editor: Suzanne Zunic
Marketing Manager: Debra Zelnio

GST Registration No.: R128870672

Periodical Postage Pending at Atlanta, GA 30304.

POSTMASTER: Send address changes to ***Emergency Medicine Reports***, P.O. Box 740059, Atlanta, GA 30374.

Back issues: $23.
Missing issues will be fulfilled by customer service free of charge when contacted within one month of the missing issue's date.

Subscriber Information

Customer Service: 1-800-688-2421
Customer Service E-Mail Address: custserv@ahcpub.com
Editorial E-Mail Address: david_davenport@medec.com
World-Wide Web page: http://www.ahcpub.com

Subscription Prices

1 year *with* 20 ACEP, AMA, or AAP
Category 1 credits: $289;
1 year *without* credit: $239;

2 years *with* ACEP, AMA, or AAP
Category 1 credits: $578;
2 years *without* credit: $478;

3 years *with* ACEP, AMA, or AAP
Category 1 credits: $867;
3 years *without* credit: $717.

Multiple copies:
One to nine additional copies: **$75 each;**
10 or more additional copies: **$50 each.**

All prices U.S. only. U.S. possessions and Canada, add $30 postage plus applicable GST.
Other international orders, add $30.

Accreditation

Pediatric Emergency Medicine Reports'™ continuing education materials are sponsored and supervised by American Health Consultants. American Health Consultants designates this continuing education activity as meeting the criteria for 20 credit hours in Category 1 for Education Materials for the Physician's Recognition Award of the American Medical Association, provided it has been completed according to instructions. This CME activity was planned and produced in accordance with the ACCME Essentials. ***Pediatric Emergency Medicine Reports*** is also approved by the American College of Emergency Physicians for 20 hours of ACEP Category 1 credit. This continuing medical education activity has been reviewed by the American Academy of Pediatrics and is acceptable for 20 AAP Credit hours. These credits can be applied toward the PREP Education Award available to Fellows and Candidate Fellows of the American Academy of Pediatrics.

American Health Consultants is accredited by the Accreditation Council for Continuing Medical Education to sponsor continuing medical education for physicians.

Questions & Comments

Please call **David Davenport,** Managing Editor, at (404) 262-5475 between 8:30 a.m. and 4:30 p.m. ET, Monday-Friday.

Statement of Financial Disclosure

American Health Consultants does not receive material commercial support for any of its continuing medical education publications. In order to reveal any potential bias in this publication, and in accordance with Accreditation Council for Continuing Medical Education guidelines, we disclose that Dr. Stewart (author) and Dr. Winograd (peer reviewer) report no consultant, stockholder, speaker's bureau, research, or other financial relationships with companies having ties to this field of study.

by decreasing edema and the return of normal pulses in a patient who has no complications. In severe cases, healing is hampered by the extensive edema and pain. Associated with the recovery phase may be skin depigmentation, hyperhidrosis, cold sensitivity, and pain on weight bearing. This phase may last for years after the initial insult. Late problems may include intermittent local ulcerations, painful cutaneous fissuring, and chronic infections.

Frostbite

Pathophysiology. Frostbite occurs when tissue freezes. Since tissue is not composed of water alone, it will not freeze until it has been cooled to -3°C to -4°C. For the tissue temperature to drop this low requires the ambient temperature to be somewhat less than -6°C. The tissue temperature is influenced by both external cold stress and endogenous internal heat production.

Because the extremities are more than 50% of the body surface area, they are vital in thermoregulation. Under normal climatic conditions, the blood flow to the extremities is abundant. Arteriovenous anastomoses shunt warm arterial blood to the superficial veins and warm the skin. If the core body temperature drops, these distal arteriovenous anastomoses are shut down and the blood is rerouted away from the skin. This minimizes core body heat loss. Unfortunately, it also deprives the extremities of warmth and predisposes the human extremities to cold injury. More than 89% of frostbite injury occurs in the extremities.

Experimental studies show two possible mechanisms of tissue injury in frostbite—ice crystal formation in the extracellular fluid compartment or microvascular aggregates and emboli formation with subsequent vascular stasis. There are proponents of both theories in the literature, but years of research have not shown either to be predominant. It is likely that we will find that both mechanisms contribute in some way to tissue injury. Better understanding of the pathophysiology of freezing and subsequent tissue damage may improve the treatment of this condition.

Ice Crystal Formation. As tissue freezes, ice will eventually form in the extracellular fluid compartment. Two pathological results happen. As fluids are drawn from the intracellular space in an attempt to maintain osmotic equilibrium, the colloid osmotic pressure in the extracellular region decreases. The cell membranes are ruptured by resultant cellular dehydration and hypertonicity. Cellular membranes may also be ruptured by formation of sharp spicules of intracellular and extracellular ice crystals. The extracellular crystallization also causes pressure on the surrounding tissues including the vascular structures and the cell membranes. These mechanisms lead to cell rupture and subsequent death if they are severe enough or of long enough duration.

Microvascular Changes. After sufficient cooling time, early signs of tissue damage occur at the endothelial level. Subsequent endothelial damage is prominent in animal studies of even minimal frostbite. This endothelial damage may be the starting point of a cascade of subsequent pathology and the initiation of a vicious feedback cycle.

Associated with rewarming are red-cell sludging, platelet aggregation, and microthrombus formation, which impedes distal blood flow.[12] These microvascular aggregates form within 1-2 hours after the tissues are thawed. The initial insult is followed by increased vascular permeability, edema formation, and decreased circulation.[13] There is a proliferation of adventitial cells and mural hemorrhage in both plugged and patent vessels.

It is felt that injury to the microvasculature system is a major cause of tissue destruction in frostbite. Weatherly-White and associates demonstrated the magnitude of this vasculature insult when they showed that normal skin transplanted to frostbitten tissue beds will slough, whereas frostbitten skin transplanted to a normal tissue bed will have a significant chance of survival.[14] Also, the decreased circulation can be detected by RISA studies and by serial angiographs of the affected extremity.

The causes of the resulting microvascular aggregates, intravascular sludging, and tissue damage are not well understood but may include the increased viscosity of fluids at low temperature, capillary membrane damage, an increase in the activities of clotting factors, and local vasospasm with hypoxia. Recent work has also implicated arachidonic acid, released from the damaged epithelial cells, as a direct cause of tissue damage.[15,16]

The major pathophysiologic problem after the vessel damage seems to be hypoxemia and microvascular compromise.[17] Local tissue hypoxia and death occur as a result of the decrease in circulation. Any factor that decreases oxygen transport will exacerbate the injury and lead to further damage. The surrounding cells then convert to anaerobic metabolism, producing lactic acid. This leads to impaired enzyme function and further disruption and death of cells in an already compromised cellular milieu.

It is important to remember that this process expands outward as the injury progresses. The peripheral region of injury is in a state of reversible damage, not death; therefore, proper care is important to limit the final tissue loss. Partial rewarming allows the circumferential tissue to temporarily revitalize, but if the tissue is refrozen, it will result in increased tissue loss. This may be caused by local blood trapping, with more local fluid crystallization and further tissue destruction. This seems to be the basis of "freeze-thaw-freeze" injuries.

Tissues vary in their susceptibility to cold injuries. Those most sensitive include the nerves, blood vessels, and striated muscle. Skin, fascia, and connective tissue are relatively resistant to injury by cold, while bone and tendon are highly resistant. An exception appears to be juvenile bony epiphyses, which appear to be more sensitive to cold than other bony surfaces.

One problem in researching frostbite is that there are no completely satisfactory animal models to study.[18] A major obstacle in the analysis of frostbite is the difference between slow onset of frostbite and variable rewarming techniques found in humans and the entirely controlled techniques found in animal experiments. Current animal models include the rabbit (ears and hind feet), hairless mice (ears), and pig (back and abdominal skin).

Risk Factors. *Ambient temperature and climatic factors.* Needless to say, the predominant risk factor in frostbite is exposure to cold. Cold exposure is directly related to the air temperature and the duration of the exposure. It also is related to the relative humidity and the local wind conditions. Thermal losses increase with decreased temperature, increased winds, increased humidity, and increased duration of exposure. Conditions that increase the body's rate of heat loss lead to a higher chance of cold injury. In sufficiently cold ambient temperatures with inadequate insulation, even penile frostbite has occurred.[19]

Sensory nerves are very sensitive to cold. Nerve conduction is impaired at 10°C and below. This means that the frostbite victim is unable to feel the changes that occur in the affected part. Indeed, a relatively reliable symptom of frostbite is the complete cessation of discomfort in the involved area.

Perhaps the most dramatic recorded cases of frostbite

occurred in World War II. In the B-17 and B-24 bombers, the waist port gunners had to open their doors to fire their weapons. They were exposed to very cold air rushing by at more than 200 miles per hour. During the winter of 1943, frostbite caused more casualties in these bomber crews than all other sources combined, including enemy fire. In the civilian population, 90% of frostbite occurs at temperatures below -6.6°C (20°F) after 7-10 hours of exposure.

Increased heat loss is also seen when tissue is in contact with volatile liquids, such as kerosene or gasoline. Wet clothing or direct metal contact to skin dramatically decreases the time needed for frostbite to occur. The wetness enhances the skin's conductive and evaporative heat losses. Exposure to very cold chemicals, such as liquid oxygen or liquid nitrogen, produces "instant" frostbite.[20] This quick-freezing phenomenon may also be seen when the skin is in direct contact with ice or "dry ice."[21,22] The "instant" frostbite of liquid gases has the potential to produce not only cutaneous but deep and severe tissue injury.[23,24]

The direct contact of living tissue with cold objects will result in a quicker and more significant cold injury than mere exposure to cold air. The amount of tissue exposed plays a role in the overall recovery from the cold injury. The larger the surface area exposed, the greater the total heat loss. This may be particularly notable in children who have been injured or are comatose and sustain cold injuries. Those body parts in contact with the snowbank or ground will show a deeper cold injury. Heat is more easily lost by conduction (direct conduct) across a wet surface.

Nutritional status. Because of the borderline metabolic status of the cells, severe malnutrition predisposes a person to cold injury. The cells have little reserve against injury, and the cold injury is deeper and more severe than in a person with normal metabolic reserves.

Race and Heredity. Military studies have demonstrated that blacks are from 2.8 to 6 times more susceptible to a cold injury.[25] In Korea, blacks suffered 52% of the total cases of frostbite but numbered only 9-10% of the exposed population. In addition, their injuries appear to be both deeper and more severe than those of other races.[26,27] This susceptibility is a racially mediated decrease in periodic vasodilation despite cold stress (the Hunting reflex).

Caucasian southerners are more than 3.7 times more susceptible to cold injuries than those from the northern-most states. It is not known whether this is related to birth, training, or acclimation.[28]

Acclimation. Exposure to cold will evoke acclimation and result in increased tolerance to both generalized and local cold exposure. Massey found that people in their second year in the Antarctic showed greater immunity to frostbite than newcomers. His study demonstrated that 29% of cold exposures resulted in frostbite in adapted researchers vs. 74% in newcomers.[29] Savourey found that acclimation to cold can occur in as little as two weeks of exposure.[30]

Previous cold injury. A history of previous cold injury increases the risk of reinjury. Residual microvascular changes can increase the morbidity of any additional cold insult. Fully resolved minor superficial cold injuries probably do not cause an increased risk.

Constricting clothes, shock, and blood loss. Constricting clothes, shock, and hypovolemia may markedly decrease the peripheral blood flow. As the peripheral blood flow is decreased, internal heat is moved to the extremities and oxygen supply is decreased. Heat flow into the extremity can also be hampered by a cramped position or direct pressure. These factors increase the chance that frostbite will occur in the affected tissues and may increase the severity of the subsequent injury.

Trauma sustained in freezing and subfreezing conditions poses a special problem in management. The patient who is immobilized, has shock due to hypovolemia, and has possible vascular compromise due to fractures is at high risk for frostbite and generalized hypothermia. Large amounts of heat can be rapidly lost through contact with the ground or snow. It is extremely important to insulate the patient from the ground to minimize these losses.

Drugs and Medications. Any drug or medication that impairs the ability to get out of the cold, decreases the peripheral circulation, or alters the reflex arcs will increase the risk of cold injuries. Vasoconstrictive drugs, such as nicotine, can cause hypoxia and rapid heat loss in the extremities. This increases the extent of the frostbite and shortens the freezing times.

Vasodilating drugs, such as alcohol, cause increased blood flow to the cold extremities. The increased cooling of the blood increases the risk of hypothermia and obtundation. Cerebral functions are impaired as the body temperature drops, and it becomes difficult to solve more complex problems, such as how to get to someplace warmer.

Some drugs, such as alcohol, narcotics, and barbiturates, decrease baseline physical activity and central heat production. This generalized cooling will increase the chance of frostbite in all exposed areas. If mental function is impaired due to pharmacologic or psychiatric reasons, the victim may neglect to select proper clothing and shelter. This further increases the risk of exposure.

Altitude and hypoxia. If all other factors are equal, a frostbite injury at high altitude is more profound and deeper than at sea level.[31] The decreased ambient oxygen tension found at high altitude causes hypoxia, which increases the severity of the injury.

Activity. Activity and the subsequent increased heat production can be protective. The increase in internal heat can delay the onset of a cold injury. But a child who has been exercising in sufficiently cold weather, with inadequate insulation or with exposed tissues, may develop frostbite. There is a higher chance of injury occurring if the child has been inactive or rests with the extremities in a dependent position.

At least one author has proposed that anxiety and fear also predispose to cold injuries.[32] Sampson notes that behavioral immobility, disorganization, and carelessness were commonly associated with anxiety in combat. He further notes that anxiety and fear provokes sweating and vasomotor constriction in the extremities. If a child trapped by cold weather becomes overwhelmed by anxiety, he or she may be unable to make appropriate decisions to protect himself or herself from the cold. His or her body may be unconsciously responding to the anxiety in a manner that makes a cold injury more likely.

Clinical Course

During Freezing. As frostbite begins, the child is often unaware that it is occurring. As noted earlier, when tissue tem-

peratures drop to less than 10°C (50°F), afferent sensory impulses no longer warn of the impending danger. The initial symptoms are variable, but most patients describe a gradual subsidence of painful cold sensations followed by tingling and then numbness. Frequently, this numbness is likened to "a block of wood" or "a club."

The parents or bystanders may note that the child's skin is initially red followed by a pale or waxy white appearance. This stage is reversible, but if not stopped, will progress to deep frostbite. The tissues first blanch, and then progress through a doughy stage to "rock hard" indicating complete freezing. Frequently a frosty rime will appear on the tissues after freezing.

After Thawing. After the extremity has been thawed, a throbbing pain is common and lasts 2-4 days. It may last as long as several weeks in severe cases. Shortly after thawing, edema formation begins. With more severe injuries, blisters form within 24 to 48 hours.

In several days, edema and blisters begin to resolve. A dark black, dry eschar will form over the course of 2-3 weeks. Four or more weeks after the injury, the eschar will begin to gradually slough, and a demarcation will form between the viable and the nonviable tissues. Over a period of days, weeks or months, demarcation between healthy and dead tissue becomes more pronounced, and the viable tissues separate from the mummified until spontaneous amputation occurs.

Late Findings. Some patients describe a long-lasting tingling sensation. This may be either mild or particularly unpleasant and similar to an electric current sensation. The feeling is more noticeable at night. Other patients have described a burning sensation that slowly subsides over a three- to four-week period. This burning sensation may be intensified if the person places the affected part in a dependent position, decreases activity, or wears tight clothing or boots. It does not occur in patients who have no tissue loss. All frostbite victims will lose some sensation to touch, pain, and/or temperature. In extreme cases, the loss of sensation may extend as much as 4 cm proximal to the demarcation. This sensory deficit may persist indefinitely.

Physical Findings

Degrees of Frostbite. In the past, the extent of the frostbite injury was described by degrees, as in thermal burns.[33] The authors who described frostbite in this manner were impressed by similarities between thermal burns and cold injuries.

First-Degree Frostbite. First-degree frostbite is characterized by hyperemia and edema. After rewarming, the tissue becomes mottled, cyanotic, and painful, with intense itching or burning sensations. There may be desquamation of superficial tissue 5-10 days after the injury.

Second-Degree Frostbite. In a second-degree injury, there is hyperemia and vesicle formation. After rewarming, the skin becomes deep red and feels hot and dry to the touch. Within 2-3 hours after rewarming, the injured area begins to swell. Blebs, usually containing a clear fluid, form after 6-12 hours.

Third-Degree Frostbite. Third-degree frostbite is associated with necrosis of the skin and cutaneous tissues. Again, bullae form, but these frequently appear violaceous or hemorrhagic. Within six days after rewarming, the entire region becomes edematous. This "degree" is frequently associated with early anesthesia followed by severe, deep aching or throbbing in 1-2 weeks.

Fourth-Degree Frostbite. A fourth-degree injury is characterized by complete necrosis and loss of tissue. In this stage, the tissue, including the bone, is destroyed. After rewarming, the part becomes deep red, cyanotic, or mottled. There is anesthesia in the involved area, and after 6-12 hours, the proximal area begins to swell. The injured area does not become edematous. Dry gangrene and mummification rapidly develop.

Depth of Frostbite. The idea of "degrees" of frostbite is cumbersome and often inaccurate. In the ED, after rewarming, it is often difficult to differentiate frostbite from trench foot, or to determine the level of tissue destruction. Even after days of observation, a classification by degrees may be incorrect and misleading in prognostic value. The common old expression "frozen in January, amputate in July" illustrates the difficulty of determining the depth of the injury on the basis of clinical signs and symptoms alone. Classifying the injury in degrees is being replaced in EDs by simple differentiation into superficial and deep cold injury.[34]

Superficial Frostbite. Superficial frostbite describes a sudden blanching of the skin which is followed by a white, waxy, frozen appearance. Beneath the superficial injury, the tissues are resilient. This injury is often also called frostnip. It represents a reversible and superficial injury.

Deep Frostbite. Deep frostbite occurs when the tissues are icy hard, without deep tissue resilience. The area resembles a piece of frozen meat. There is no sensation, and the patient frequently describes an extremity as "feeling like a block of wood." If the patient presents after the extremity has been thawed, these signs will not be present, and it will be difficult to classify the injury. Historical data from the patient may help in the classification process.

Prognostic Factors. Damage is commonly overestimated in the early stages of frostbite, even by relatively experienced clinicians. An important part of management is to allow time for the resolution of the injuries before giving firm prognostic predictions.

Blebs. Within 24 hours after thawing, blisters or blebs of various sizes usually develop in more severe cases of frostbite. These blebs result from the death of the superficial layers of skin. If blebs develop, a relatively favorable sign is the presence of larger blebs, filled with pinkish fluid, that extend to the ends of the affected digits. Blebs that are small, filled with purple or hemorrhagic fluid, and extend only partway to the ends of the digits are a poor prognostic sign.

In the most severe cases, the extremity is completely anesthetic, cold, bloodless, and without blisters or edema. Mummification will often begin within a few days.

Freeze-Thaw-Freeze Injuries

Under no circumstances should the extremity be allowed to refreeze. The cycle of freeze-thaw-refreeze leads to a marked increase in tissue destruction. Indeed, this was the very point that Baron Larey had noted in his famous clinical observations that were quoted previously.

It should be noted that keeping the frozen part frozen without increasing the amount of frozen tissue is not an easy task. The extremity should be insulated from further cold exposure, while not so insulated that internal body heat provides a slow rewarming. Transport in heated ambulances or "snow cats" is frequently associated with slow rewarming over long transports.

If the transport vehicle subsequently breaks down or becomes mired, the patient is at risk of the freeze-thaw-refreeze phenomenon. Determining the best time in a winter storm to evacuate a patient with severe cold injuries may require both expert judgment and experience.

Adjunctive Diagnostic Procedures

The unpredictable course and the time before demarcation is completed in a cold injury presents a dilemma to the physician. The desire to preserve as much extremity length as possible is offset by the duration of therapy required before definitive characteristics of the problem occurs. Few of the proposed prediction or classification schemes have succeeded in accurately predicting the clinical course of an individual injury.[35]

X-ray. Plain film x-rays are relatively insensitive in assessing the tissue damage in frostbite. Early indicators of soft tissue freezing are essentially nonexistent on x-ray.[36] Late indicators are further discussed in the section on sequelae of frostbite and are not useful in diagnosis or prognosis of the disease.

Angiography. In frostbite, it appears that the vascular injury with subsequent interruption of blood flow is the major cause of tissue death. Flow studies of the region affected by the frostbite may be a useful indicator of tissue viability. Arteriography has been used to assess tissue viability by demonstrating perfusion of the vessels in the distal part of the involved extremity. Routine angiography is relatively crude and inadequately demonstrates the smaller vessels or the microvasculature needed for continued cell life. Angiography catheters can also serve to instill vasodilating medications into the selected arterial perfusion tree.[37] Serial microangiography has been used to show the changes in perfusion in the rabbit's ear, but no studies have been done with this technique in humans.[38]

Magnetic resonance angiography (MRA) has been anecdotally used to outline vessel flow patterns in affected limbs.[39] MRA/magnetic resonance imaging (MRI) is more expensive than nuclear flow scanning, but may be useful earlier and certainly gives better resolution. The utility of MRI/MRA has yet to be determined.

Nuclear Scanning. Routine bone scans using technetium are not a good prognostic indicator of the extent of tissue loss until at least five days and possibly three weeks have elapsed.[40] Methods that have been tried include bone scanning with technetium methylene diphosphonate (^{99}mTcMDP) and ^{99}mTc pertechnetate imaging.[41-44] ^{99}mTc pertechnetate appears to be both most readily available and useful. In many cases, it is the soft tissue injury that determines the extent of resection not the bony injury that is seen on the scan.

The search for quicker diagnostic classification techniques has yielded one fruitful technique—nuclear flow scanning. By combining the techniques of radionuclide scans and angiography, a good, early method for eliciting the depth and level of the frostbite injury has been found. Tc^{99}m pertechnetate angiographic imaging within 24-48 hours after the injury is the current method of choice.[45] In multiple cases, there has been a good correlation between the flow scintigraphic findings and the extent of deep tissue that ultimately required surgical resection. The nonviable tissue appears as a perfusion defect on the radionuclide angiogram and the subsequent static images. Persistent perfusion defects on follow-up scans 7-10 days after the injury show the extent of nonviable tissue and that need surgical therapy. This technique has proved more accurate than Doppler flow studies or routine angiography.

Other Studies. Thermal clearance, ^{31}P-NMRS, thermography, and ^{131}Xe-muscle blood flow estimates have been used anecdotally to give flow patterns in frostbitten extremities. The use of these studies has not been shown in any controlled study. In the single 19-year-old patient studied, they corresponded with older studies such as bone scan.[46] Early signs of severity were found to be the low temperature measured with thermography and the results of the ^{31}P-NMRS on the first and fourth day.

^{31}P-NMRS was thought to be particularly useful and demonstrated decreased concentrations of high-energy compounds and increased concentration of free inorganic phosphate. This was hypothesized to be due to decreased oxygen supply caused by the post-warming thrombosis.

Thermography assesses the temperature of the deeper tissues affected. It was originally thought that this technique would be able to predict the clinical outcome and severity of frostbite at an early stage in the disease.[47] Thermography can quite accurately distinguish superficial frostbite from deep frostbite. It has been less useful in staging tissue loss.

Treatment

The treatment of a severe cold injury should begin in the field with proper management. It continues with an evaluation of the situation that caused the cold injury, and then includes rapid rewarming of the lesions in specific and the patient in general. After rewarming, there is a predictably long course of rehabilitation and demarcation of the lesions.

Field Considerations Prior to Rewarming. *Treatment of Hypothermia.* With very rare exceptions, children who have sustained frostbite should be considered to have had such significant exposure to cold that they are at risk of generalized hypothermia. A rectal or other core temperature should be obtained on any child who has sustained frostbite or immersion foot. Oral temperatures are usually not adequate, because some well meaning soul has frequently pressed a cup of hot drink into the victim's hands prior to recording the temperature. If the core temperature is less than 35°C (95°F), hypothermia needs to be treated before the frostbite or immersion foot.

Frostbite may easily affect more than one body part, so the emergency provider should examine other likely areas as well as the obvious injuries. Frequently, children who are complaining about frostbite of upper extremities will have ear or foot damage also.

The patient should have a thorough secondary assessment, with special attention to the presence of complicating conditions. Look beyond the frostbite. Other conditions that should be considered in children include dehydration or hypovolemia, anoxia, hypoglycemia, carbon monoxide poisoning from faulty heaters or cars, and metabolic acidosis. In older children and adolescents, frostbite may be attributable in part to trauma or the effects of alcohol or drug use.

Mechanical Damage. Frozen tissues must be treated in a gentle fashion. Massage, rubbing with snow or ice, or ambulation are to be avoided. Patients with frostbite of the lower extremities should be moved on a litter, if at all possible.

Field Rewarming. If there is any danger that the extremity may be refrozen, the child should not be rewarmed in the field. Remember that in temperate climates, where temperatures often fluctuate daily, thawing is likely to occur spontaneously. An extremity that has already thawed should *not* be rapidly rewarmed. This serves no useful purpose and may damage the extremity further.

It is virtually impossible to transport a patient in a warm ambulance for any distance without some thawing of frozen parts. When transporting the patient with unthawed frostbite, the patient compartment or cabin should be kept cool and the extremity kept away from the heater. Unfortunately, this is a premise that is often violated—for the creature comforts of the transportation crew.

If the weather causes additional delay or breakdown, the patient risks a freeze-thaw-refreeze cycle. Transportation times, modes, and routes should be carefully planned to avoid this possibility.

A cold-injured part should never be rubbed with snow or massaged. This can lead to irreversible mechanical trauma. The part should not be massaged during rewarming.

Avoidance of Ambulation. The child with a cold injury to the lower extremities should be evacuated if at all possible. Field transportation should be by litter unless only ears, face, or upper extremities are involved. It is considered to be less damaging to walk on frozen feet to shelter than it is to stay out in the cold or to walk on feet that have been thawed and then allowed to refreeze.[48]

Field providers should be reminded that injured extremities are at very high risk for cold injury, particularly if they are splinted. Be certain to frequently check the neurovascular status of a splinted extremity and the tightness of the splint. Ensure that the extremity, if not already frostbitten, is well protected.

Rewarming

The ideal method of rewarming the frozen extremity has not been found. Most of the tissue damage occurs during rewarming, not freezing. Although sperm, bone, and arterial grafts have been frozen and rewarmed, more complex tissues have uniformly been damaged during the rewarming process.

Warm Water. The Alaskan frontier and winter military campaigns provided good testing grounds for various treatments of cold injuries. In 1956, studies were done to test the use of warm water for rapid rewarming of the injured area. Water between 32°C and 44°C (90°F and 108°F) was used to bathe the affected region for 20-40 minutes.[49,50] This temperature range has seemed to provide the least amount of residual tissue damage and death.

Rapid rewarming by external means appears to provide better results than slow thawing but does not always give protection from tissue losses, particularly in deep or long duration freezing injuries.

The current preferred initial treatment for deep frostbite is rapid rewarming in a water bath at temperatures of 32-44°C (90-108°F). The temperature of the water is monitored closely. Additional hot water should be added to the bath only after the extremity has been temporarily removed. The hot water should be mixed with the cooler water and the temperature checked before replacing the extremity in the water bath.

Rewarming should be continued until a hyperemic flush extends completely to the ends of the extremities. This often takes 30-40 minutes. If the part has been quite deeply frozen, the flush may not extend completely to the ends of the digits. To encourage vasodilation, warm blankets and warm drinks should be offered to the patient if medically appropriate.

Rewarming is quite painful, and the patient often requires narcotic analgesia during the initial thawing phase. Intravenous narcotics are suggested for both speed of action and surety of effect. Children may require sedation during rewarming. If the child has severe vasoconstriction, a central line may be required for hydration and administration of analgesia.

The patient's extremities should not be massaged during the rewarming phase. Debridement of tissue by the action of a Jacuzzi or similar device is appropriate, but scrubbing or rubbing will only damage tissues further.

Non-Recommended Forms of Rewarming

Spontaneous Rewarming. Spontaneous rewarming permits variable results that appear to be determined by the depth of injury, duration of the freezing injury, and the patient's activity during the rescue and thawing phases. Results of gradual rewarming at room temperature seem satisfactory for treatment of children with superficial frostbite.

Dry Heat. Excessive heat should be avoided in the rewarming phase. Numerous anecdotal and published accounts have shown that excessive heat can lead to poor recovery.

Because dry heat temperatures are too difficult to control and the frozen part is too insensitive, the use of dry heat may cause further damage. Avoid sources of dry radiant heat at temperatures greater than 150°F (66°C) (such as found with wood fires, hair dryers, or radiant heaters).[51] Neither auto heaters nor engine exhaust should be used to rewarm a frostbitten area for the same reasons.

Miscellaneous Rewarming Techniques. Many forms of treatment for frostbite have been tried, including the following: thawing the tissue in water mixed with melting ice, rabbit or other animal fats, cods' heads, and hot oil. The results of these methods have been uniformly disastrous. They increase the incidence of gangrene, high-degree amputation, infection, and death from sepsis.

Late Care

Unfortunately, most frostbite is not seen by physicians while still frozen. Indeed, wartime statistics revealed that less than 2% of the cold injuries were seen by a medical officer prior to rewarming.[22] In civilian practice, many of the children with frostbite had parents try a home remedy prior to seeking medical care. These do-it-yourself treatments can result in serious complications. Other patients will not seek care until bullae formation, marked cyanosis, or even gangrene of the extremities has occurred. It should be noted that Alaskans have had good experience with a telephone treatment protocol for home therapy of frostbite. It is used when weather does not permit transportation to a medical facility. This telephone protocol follows the steps outlined for hospital treatment of cold injuries.

After the patient has been rewarmed, it is frequently impossible to adequately stage the illness. The patient's history may aid

in classification, but prior inappropriate therapy may make any classification scheme unusable. These patients must be managed as is appropriate for the presenting stage of their disease.

Post-thaw Care

In addition to rewarming the frozen extremity, hospital based treatment includes prevention of infection, promotion of healing, and preservation of function. Since much of the underlying damage of frostbite is related to the vasospasm and intravascular microemboli, substantial efforts have been directed toward the remedy of these problems. All children should be admitted to the hospital who have more than a superficial injury.

Wound Care

General. After thawing, the digits should be separated with cotton pledgets or lambs wool and the limbs elevated to minimize swelling. Most authorities agree that treatment of the cold injured extremity should be open, although there is some disagreement about this precept.[52] Occlusive dressings and wet dressings should be assiduously avoided. The injured tissue should be kept protected, clean, and dry. Children with frostbite of lower extremities should be restricted to complete bedrest until edema in the foot has resolved and any blisters are completely dry.

Reverse isolation is recommended in severe cases until the blebs rupture and dry. When the injury is severe, the extremities should be protected by sheet cradles to avoid trauma and pressure. Feet should be elevated and may be covered with soft wrappings. This is not necessary for upper extremity injuries that may be laid over the chest and trunk upon sterile bed sheets. Hands may be protected with plastic bags for routine self care, such as shaving and use during toilet functions.

Antibiotics. In general, neither topical or systemic antibiotic medications are required. Gentle debridement of superficial tissue can be adequately performed by the twice daily use of a whirlpool using a Betadine solution at a temperature of 30°C (90°F). This is followed by a clear rinse and air drying. Some clinicians disagree with this approach and start all nonallergic patients on penicillin until the edema resolves.[13] There are no controlled studies that validate this practice.

Ambulation. If the lower extremities are involved, ambulation should not be permitted. Crutches or a wheelchair should be used for the first 1-2 weeks. If eschar limits a full range of motion or circulation, escharotomy may be performed.

Surgical Debridement. The ultimate debridement is amputation. This should be done only after clear demarcation of the injury and the surgeon needs to be aware that internal injury may extend above the 'line of demarcation' of the wound.

Debridement of Blebs. Current controversy reigns about the management of blebs in frostbite. One group feels that blebs contain prostaglandin and thromboxane. According to this group, removal of blebs will decrease tissue damage and speed healing. Recent studies have shown that thromboxane, a breakdown product of arachidonic acid, and prostaglandins may increase dermal ischemia.[12] The presence of these products in the fluid obtained from the frostbite blebs of an experimental rabbit-ear model, and the quicker healing after removal lead to this conclusion.

An older group feels that the blebs are protective against infection and leave blebs intact until they spontaneously rupture. Already ruptured blebs are gently debrided.

All authorities stress the absolute need to watch and wait before other surgical debridement is initiated. If given enough time (1-6 months), the dead tissues will demarcate and mummify—clearly delineating between dead and viable tissue. If one intervenes too early, this delineation will not be apparent and excessive tissue may be surgically excised. Splitting or bivalving of the eschar may be needed to promote joint movement and prevent stiffness.

Whirlpool Debridement. Whirlpool baths for 20-30 minutes at a time in water temperatures between 32°C and 35°C (90°F and 95°F) are taken twice daily. The whirlpool cleans the debris and superficial bacteria from the extremity. The gentle debridement of the whirlpool is the only surgical debridement until the demarcation is complete.

Compartment Syndrome. If the extremity has remained frozen for a prolonged period of time or if significant trauma has been associated with the freezing injury, a compartment syndrome may be noted. This condition is usually found in the lower extremities, although it may occasionally be seen in the upper extremities. The diagnosis may be determined by measuring the compartment pressures; in serious cases, continuous compartmental pressure monitoring may be indicated. Fasciotomy may be required for resolution of this problem. Delay in performance of fasciotomy in the face of high compartment pressures may lead to the loss of the limb.

Smoking. Nicotine should be avoided due to its vasoconstriction effects. Because it is vasodilating, alcohol should be avoided in the early phase of rewarming. Vasodilation of cold and frozen extremities may send a rush of cold blood to the heart. Combine this with a general hypothermia and the likelihood of fatal arrhythmias increases.

Adjunctive Therapy

Numerous agents and therapies have been proposed to decrease the ravages of frostbite. None of these agents or therapies has been unequivocally shown to decrease tissue loss or increase limb survival. They are listed so that the emergency physician has an increased understanding of the follow-up therapy that may be proposed for the patient with frostbite.

Calcium Channel Blocking Agents. Calcium channel blocking agents provide a vasodilating effect. These decrease the vasospasm and increase the blood flow to the injured area, often in a more controlled fashion than other agents. If systemic drugs, such as nifedipine, are administered, the patient needs to be well hydrated to prevent orthostatic hypotension. The patient should also be protected from the cold to prevent generalized hypothermia.

Dextran. Experimental animal studies have shown that low molecular weight dextran, administered as soon as possible after a frostbite injury, helps to decrease red blood cell aggregation and microemboli formation.[53] These experimental results conflict with clinical studies that have failed to confirm any clear benefit in humans.[54]

Anticoagulant and thrombolytic agents. Heparin and warfarin (Coumadin) have shown, when administered early, to improve the outcome of frostbite injuries in animal models. Tissue survival was not improved when these drugs were given to humans during studies in Korea. The cold damaged areas were not rewarmed under controlled conditions so the conclusions of human studies may be subject to considerable error.

Streptokinase and tissue plasminogen activator have been proposed as adjunctive agents in the treatment of frostbite. Animal studies have been done, but no human clinical studies yet have been accomplished.[55] (Unpublished communications with Genentech, Inc.) The idea of lysis of the microvascular emboli is appealing and may offer significant relief from the local hypoxic effects. Animal and human clinical studies are needed to explore this concept. Both of these agents have significant side-effects.

Thromboxane Inhibitors and Antiprostaglandin Agents. Aloe vera cream (Dermaid) has been used in humans as a topical thromboxane inhibitor, along with early surgical debridement of the blebs by Robson et al.[56,57] This study reported good results in the protocol group. Unfortunately, these studies were poorly controlled, showed selection bias in favor of less severe frostbite, and are essentially anecdotal in nature. The same surgeons did not even treat both groups of patients. Some local perfusion benefit was noted, but the study did not show increase in tissue survival. The quality of the healed tissue may have been improved. In this same study group, systemic aspirin or ibuprofen was given to inhibit prostaglandin production. Again, the results were encouraging, but are still open to some question because of the nature of the study.

When thromboxane inhibitors alone were studied in experimental animals, the results were mixed.[58,59] In the study by Goldberg et al, no support was found for thromboxane inhibition with methimazole in improving tissue survival This was true for both immediate treatment and delayed administration (4 hours) of the treatment. Raine et al, in an earlier and similar study, found that treatment with steroids, aloe vera, methimazole, and aspirin all yielded better survival than controls. Further investigation using these and other similar agents is needed.

Since aspirin, ibuprofen, and topical aloe vera are relatively free of noxious side effects and are easily tolerated by most patients, even modest benefits in healing would justify routine use in cases of frostbite. These drugs deserve a well-controlled human study to establish their efficacy and to determine which agents are most effective.

Hyperbaric Oxygen. Hyperbaric oxygen has been used by some centers as an adjunctive therapy for frostbite. If any part of the ultimate gangrene is due to a reversible vascular factor, it is felt that measures designed to relieve local tissue anoxia will be helpful during healing of the vascular damage. It is further postulated that the administration of hyperbaric oxygen will increase the oxygen content of the plasma up to 20 times, enhancing the oxygenation of the marginal tissues. Anecdotal reports have been equivocal.[60,61] Animal studies have been contradictory, and there are no controlled studies in humans.[62,63]

Non-Recommended Adjuvant Therapy

Surgical Sympathectomy. Surgical sympathectomy has been recommended for treating cold injuries severe enough to produce necrosis. Performed from 36 to 72 hours after thawing, it is thought to hasten the resorption of edema and reduce the spontaneous pain and hypersensitivity; it does not appear to increase the amount of tissue salvaged.[64,65] If a lumbar surgical sympathectomy is done, male patients must be counseled that a possible complication is permanent impotence. Sympathectomy does appear to confer some protection against subsequent cold injury.[66] The marginal improvement obtained does not outweigh the risks of this additional operative procedure, particularly in pediatric patients.

Medical Sympathectomy. Medical sympathectomies can be accomplished using regional intra-arterial guanethidine or reserpine.[67,68] Some studies have found that these agents cause a more rapid demarcation between viable and nonviable tissue. Onset of action of intraarterial reserpine is 3-24 hours after the injection, with an effective duration of 14-21 days.[69,70] Studies of patients using either intraarterial reserpine or tolazoline show some decreased tissue loss if rewarming has been slow. Unfortunately, neither agent is readily available in the United States.

Ganglionic-Blocking Agents. Ganglionic-blocking agents, such as Dibenzyline, have been used in the past to produce a systemic medical sympathectomy. They decrease vasospasm and increase the blood flow to the injured area. These agents are no longer in general use.

Sequelae. In general, the degree of long-term disability and residual damage is directly related to the extent and severity of the original injury. It is important to remember that tissue damage is in proportion to the depth of the frostbite. After suffering even from superficial frostbite, a person may have long-term problems, such as small muscle atrophy, fatty tissue loss, and cold sensitivity. Amputation may be needed for extensive and deep injuries. The muscle damage may partially resolve after blood flow returns to normal.

Miscellaneous Late Sequelae. Late symptoms occur in as many as 80% of frostbite victims and may last as long as four years.[71] These symptoms include sensation of cold feet, excessive sweating (hyperhidrosis), and causalgia-like pain or joint discomfort.[72] The symptoms often increase with the onset of colder weather. An exaggerated vasomotor response to cold may increase the chances of another cold injury.

Skin color changes, scarring, telangiectasias or nail-bed changes may also develop after frostbite.[73] The color changes and telangiectasias are thought to be a consequence of the release of vasoactive substances during frostbite. Subsequent vessel wall weakness and endothelial damage cause the persistent vasodilation.

Cold induced neuropathy may cause paresthesias, hypesthesia, numbness, and hypersensitivity to cold. This neuropathy results in measurable difference in nerve conduction velocity.[74]

Bony Injuries

If the injury involves the blood supply to the bones, distal bone death and subsequent amputation may ensue. Once an extremity has been amputated, phantom pain may be experienced. This is the sensation of pain or discomfort in the missing body part.

If the bone is frozen, joint space and growth plate changes may occur. About 50% of the patients with frostbite of the distal extremities will develop osteoarthritis months to years after the injury.[75] The radiographic findings show erosive osteoarthritic changes. The diagnosis is made by a history of frostbite and the asymmetric distribution of physical findings.

Frostbite in children may lead to epiphyseal growth abnormalities resulting in length discrepancies and deformity.[76,77] In children, epiphyseal changes due to frostbite are not immediately visible and do not seem to be widespread.[78] The exact mechanism for the epiphyseal destruction is, of course, unknown but presumed to be due to a combination of the direct freezing and the prolonged ischemia.[79]

Ear Damage

Frostbite to the ears can lead to a rare condition known as a "petrified auricle." The pinna becomes rockhard and develops ectopic sites of ossification. Surfers who have had long-term exposure to very cold (16°C or less) water for extended periods of time can also suffer from petrified auricle.[80]

Prevention

The prevention of frostbite requires good physical conditioning, good clothing, adequate rest, and the prevention of illness and trauma. Above all, it requires vigilance on the part of the potential victim and his or her companions. In times of severe cold weather, activities should be planned carefully to minimize exposure times.

Outdoor Sports. Close attention should be paid to weather and route when planning any outdoor sports. Although feeling "encumbered," the cross-country skier, hiker, or snowmobiler should pack survival equipment suitable for the worst weather encountered in the area and know how to use it. The mountain climber or hiker must also be prepared for the vagaries of the mountains and carry appropriate equipment necessary for spending a night on the mountain in inclement, cold weather. Never be too stubborn to turn back, postpone, or even cancel an outing if the weather becomes nasty. Friends, neighbors, or family should know the route taken, access points to the route, and likely shelters along the route. Once the route is decided, outdoor enthusiasts should not deviate from it without clear markings of the changes.

Needless to say, winter outback sports are not appropriate for solo ventures. Even downhill skiers should use a buddy system.

Many cross-country skiers compete with exposed face, skintight clothing, and inadequate gloves and boots. Because of their grip on the poles, skiers may neglect their hands until frostbitten. Wetness from falling or perspiration should be attended to promptly by donning extra clothes or changing into dry ones. Thin skier's racing uniforms and jogger's clothing inadequately protect the genitalia in males and the breasts in females. Once the workout is finished, these athletes should cover themselves more thoroughly to protect against frostbite.

A few sports organizations limit competition in the cold because of the risks of hypothermia and frostbite. The Federation Internationale de Ski, which governs cross country ski competition will cancel or postpone races when the temperature falls below -4°C. Skilled cross country skiers at -4°C can easily generate a wind-chill factor of -25°C to -50°C with marked increases in risk of frostbite. Running can also generate deep wind-chill conditions.[81] As one author puts it, "It is too cold to exercise when you cannot put on or carry enough clothing layers to stay warm should you have to abruptly discontinue the activity."[82]

Wind-Chill. The wind-chill factor plays an important part in the development of conditions that lead to frostbite. The cooling effects of cold temperatures are markedly increased by the winds.[83] It should be noted that exposed flesh will freeze in approximately 30 seconds with a 30 mile per hour wind at -30°F. This set of conditions is frequently encountered by individuals approaching rescue helicopters in the mountains and less frequently by winter runners, skiers, and snowmobilers.

Drugs and Medications. Of course, it is much easier to discuss telling a patient to decrease consumption of alcohol or drugs than it is to have them actually do it. In one series of more than 100 cases of frostbite, alcohol was thought to be a contributing cause in more than one-third of the cases.[33] It is important that a person with diabetes or peripheral vascular disease understand that smoking causes vasoconstriction, making them more susceptible to frostbite. These are certainly important preventative points to mention if an emergency provider is counseling patients on the avoidance of frostbite.

Concomitant Illnesses. Patients with peripheral vascular disease, Raynaud's phenomenon, and other concomitant illnesses should be counseled about their propensity for more rapid development of cold injuries. Patients with a prior history of a cold injury are at greater risk for reoccurrence. These injuries will be more severe than the initial insult in similar patients.

Seizure and diabetic patients should be counseled on proper medication practices so that they are not caught unprepared by complications of their illness while in cold weather. An unconscious period spent in direct contact with the cold ground may be sufficient to cause severe frostbite. The increased caloric consumption during periods of cold weather may cause hypoglycemia in diabetics. Hypoglycemia leads rapidly to hypothermia as the metabolic engine runs out of fuel.

Summary

Frostbite is the freezing of a body part. It can be classified into superficial and deep frostbite depending on the depth of tissue destruction. The underlying mechanism of the injury is probably hypoxemia due to osmotic changes and/or microthrombus formation. Local metabolic changes due to thromboxane and prostaglandins may play a role in the extent of the injury. Certain predisposing factors are known to increase the severity of the insult.

Thawing is the most critical time in a frostbite injury. It should be postponed until there is no chance of refreezing. During thawing, the tissue must be protected from mechanical trauma. To thaw the injured part, immerse it in water, warmed to 108°F for 30-45 minutes. Rewarming will be painful, and analgesia should be offered to the patient.

After warming, the tissues must be managed very gently and care must be taken to postpone surgical intervention until demarcation of the injury has occurred. Demarcation of the nonviable areas may take as long as six months. Early debridement of the blisters is controversial but gaining approval. Daily whirlpool with a povidone-iodine solution is one method used to gently debride eschar and nonviable tissue. Reverse isolation will protect the patient from secondary infection.

Since the days of Napoleon, medicine has made impressive advances in the care of frostbite injuries. Mortality is rare, and less tissue is lost because of manipulation, infection, secondary thermal burns, and early amputation than even a few decades ago.

References

1. Bishop HM, Collin J, Wood RF, Morris PJ. Frostbite in Oxfordshire: The impact of a severe winter on an unprepared civilian population. *Injury* 1984;15:379-380.
2. Hunter J, Middleton FR. Cold injury amputees—a psychosocial problem? *Prosthet Orthot Int* 1984;8:143-146.
3. Robinson, WA. Competing with the cold; Part 1; Frostbite. *Physician and Sportsmed* 1991;19:19-21.
4. Smith M, Matheson GO, Meeuwisse WH. Injuries in cross-country skiing: A critical appraisal of the literature. *Sports Med* 1996;21:239-250.
5. Thein LA. Environmental conditions affecting the athlete. *JOSPT*

1995;21:158-171.
6. Percy EC, ed. Trauma rounds. *CMA* J 1972;106:261-263.
7. Jacob JR, Weisman MH, Rosenblatt SI, Bookstein JJ. Chronic pernio: A historical perspective of cold-induced vascular disease. *Arch Int Med* 1986;146:1589-1592.
8. Reinertsen JL. Unusual pernio-like reation to sulindac (letter). *Arch Derm* 1981;117:1215.
9. Herman EW, Kezis JS, Silvers DN. A distinctive variant of pernio: clinical and histopathologic study of 9 cases. *Arch Derm* 1981;117:26-28.
10. Kelly JW, Dowling JP. Pernio: A possible association with chronic myelomoncytic leukemia. *Arch Derm* 1985;121:1048-1052.
11. Barber FA, Cold injury in the military. *Medical Bulletin of the US Army*, Europe 1980;37:22-27.
12. Heggers JP, Phillips LG, McCauley RL, Robson MC. Frostbite: experimental and clinical evaluations of treatment. *J Wilderness Med* 1990;1:27-32.
13. Bourne MH, Piepkorn MW, Clayton F, Leonard LG. Analysis of microvascular changes in frostbite injury. *J Surg Res* 1986;40:26-35.
14. Weatherly-White RC, Sjostrom B, Paton BC. Experimental studies in cold injury. II. The pathogenesis of frostbite. *J Surg Res* 1964;4:17-22.
15. Raine TJ, London MD, Golucld L, et al. Antiprostaglandins and antithroboxames for treatment of frostbite. *Surg Forum* 1980;31:557.
16. McCauley RL, Hing DN, Robson MD, Heggers JP. Frostbite injuries: A rational approach based on the pathophysiology. *J Trauma* 1983;23:143.
17. Lazarus HM, and Hutto BS, Electrical burns and frostbite: Patterns of vascular injury. *J Trauma* 1982;22:581- 585.
18. Gadarowski JJ Jr, Esce JD. Acute systemic changes in blood cells, proteins, coagulation, fibrinolysis, and platelet aggregation after frostbite injury in the rabbit. *Cytobiology* 1984;21:359-370.
19. Waugh RJ. Penile frostbite, an unforeseen hazard of jogging [letter]. *N Engl J Med* 1977;296:178.
20. Hwang JCF, Himel HN, Edlich RF. Frostbite of the face after recreational misuse of nitrous oxide. *Burns* 1996;22:152-153.
21. Peterson LR, Peterson LC, Peterson AK. French vanilla frostbite (letter). *N Engl J Med* 1982;307;1028.
22. Gamble WB, Bonnecarre ER. Coffee, tea, or frostbite? A case report of inflight freezing hazard from dry ice. *Aviat Space Environ Med* 1996;67:880-881.
23. Hicks LM, Hunt JL, Baxter CR. Liquid propane cold injury: A clinicopathologic and experimental study. *J Trauma* 1979;19:701-703.
24. Kurbat RS, Pollack CV. Facial injury and airway threat from inhalant abuse: A case report. *J Emerg Med* 1998;16:167-169.
25. Sumner DS, Criblez TL, Doolittle WH. Host factors in human frostbite. *Milit Med* 1974;139:454-461.
26. Miller D, Bjornson DR. An investigation of cold injured soldiers in Alaska. *Milit Med* 1962;127:247-252.
27. Whayne TF. Cold injury in World War II: A study in the epidemiology of trauma. Washington, DC: US Government Printing Office, 1950.
28. Whayne TF, DeBakey ME. Cold injury, ground type. Washington, DC: US Government Printing Office, 1958.
29. Massey PMO. Finger numbness and temperature in Antarctica. *J Appl Physiol* 1959;14:616-620.
30. Savourey G, Guinet A, Besnard Y, et al. General and local cold responses in humans after 2 weeks at high altitude. *Eur J Appl Physiol* 1997;75:28-33.
31. Hashmi MA, Rashid M, Haleem A, et al. Frostbite: Epidemiology at high altitude in the Karakoram mountains. *Ann R Coll Surg Engl* 1998;80:91-95.
32. Sampson JB. Anxiety as a factor in the incidence of combat cold injury: A review. *Milit Med* 1984;149:89-91.
33. Technical Bulletin 81. Cold injury. Department of the Army, 1976;1-13.
34. Washburn B. Frostbite. *N Engl J Med* 1962;262:974-982.
35. Knize DM, Weatherley-White RC, Paton BC, Owens JC. Prognostic factors in the management of frostbite. *J Trauma* 1969;9:749-759.
36. Tishler JM. The soft tissue and bone changes in frostbite injuries. *Radiology* 1972;102:511-513.
37. Gralino BJ, Porter JM, Rosch J. Angiography in the diagnosis and therapy of frostbite. *Radiology* 1976;119:301-305.
38. Erikson V, Ponten B. The possible value of arteriography supplemented by a vasodilator agent in the early assessment of tissue viability in frostbite. *Injury* 1974;6:150-153.
39. Barker JR, Haws MJ, Brown RE, et al. Magnetic resonance imaging of severe frostbite injuries. *Ann Plast Surg* 1997;38:275-279.
40. Miller BJ, Chasmar LR. Frostbite in Saskatoon: A review of 10 winters. *Can J Surg* 1980;23:423-426.
41. Sumner DS, Boswick JA, Criblez TL, Doolittle WH. Prediction of tissue loss in human frostbite with xenon-133. *Surgery* 1971;69:899-903.
42. Lisbona R, Rosenthal L. Assessment of bone viability by scinti-scanning in frostbite injuries. *J Trauma* 1976;16:989-992.
43. Ristkari SKK, Vorne M, Mokka REM. Early assessment of amputation level in frostbite by ^{99}mTc-Pertechnetate scan. *Acta Scan Chir* 1988;154:403-405.
44. Kenney III A, Vyas P. Frostbite injury: Appearance on three-phase bone scan. *Clinical Nuclear Med* 1998;23:188.
45. Salimi Z, Vas W, Tang-Barton P, et al. Assessment of tissue viability by ^{99}mTc Pertechnetate scintigraphy. *Am J Radiol* 1984;142:415-419.
46. Kayser B, Binzoni T, Hoppeler H, et al. A case of severe frostbite on Mt. Blanc: A multi-technique approach. *J Wilderness Med* 1993;4:167-174.
47. Hamlet MP, Veghte J, Bowers WD, Boyce J. Thermographic evaluation of experimentally produced frostbite of rabbit feet. *Cryobiol* 1977;14:197-204.
48. Mills WJ, Jr. Frostbite and hypothermia—current concepts. *Alaska Med* 1973;15:26.
49. Mills WJ, Jr. Out in the cold. *Emerg Med* 1979;March:211-229.
50. Mills WJ, Jr., Whaley R. Frostbite: Experience with rapid re-warming and ultrasonic therapy. *Alaska Med* 1960;2:1.
51. Burkhalter EL, Burkhalter JA. A cold burn: A third degree thermal injury complicating frostbite. *Cutis* 1984;34:82-83.
52. Page RE, Robertson GA. Mangement of the frostbitten hand. *The Hand* 1983;15(2):185-191.
53. Mundth E, Long DM, Brown RB. Treatment of experimental frostbite with low molecular weight Dextran. *J Trauma* 1964;4:246.
54. Penn I, and Schwartz SI. Evaluation of low molecular weight Dextran in the treatment of frostbite. *J Trauma* 1964;4:784.
55. Salimi Z. Wolverson MK, Herbold DR, Vas W, Salimi A. Treatment of frostbite with IV streptokinase: An experimental study in rabbits. *AJR* 1987;149:773-776.
56. McCauley RL, Hing DN, Robson MC, et al. Frostbite injuries: A rational approach based on the pathophysiology. *J Trauma* 1983;23:143-147.
57. Heggers JP, Phillips LG, McCauley RL, Robson MC. Frostbite: Experimental and clinical evaluations of treatment. *J Wilderness Med* 1990;1:27-32.
58. Goldberg BD, Robinson WA, Watson WA. Impact of delayed presentation on the efficacy of thromboxane inhibtion in the treatment of frostbite. *J Wilderness Med* 1994:5:325-330.
59. Raine TJ, London MD, Goluch L, et al. Antiprostaglandins and antithromboxanes for treatment of frostbite. *Surg Forum* 1980;31:557-559.
60. Smith G. Therapeutic applications of oxygen at two atmospheres pressure. *Dis Chest* 1964;45:15.
61. Cooke JN. Hyperbaric oxygen treatment in the Royal Air Force [letter] *Proc Roy Soc Med* 1971;64:881-882.
62. Okuboye JA, Ferguson CC. The use of hyperbaric oxygen in the treatment of experimental frostbite. *Can J Surg* 1968;11:78-84.
63. Gage AA, Ishikawa H, Winter PM. Experimental frostbite: The effect of hyperbaric oxygenation on tissue survival *Cryobiology* 1970;7(1):1-8.
64. Golding MR, Mendoza MF, Hennigar GR, et al. On settling the controversy on the benefit of sympathectomy for frostbite. *Surgery* 1964;56:221.
65. Golding MR, Martinez A, DeJong P, et al. The role of sympathectomy in frostbite, with a review of 68 cases. *Surgery* 1965;57:774.
66. Bouwman DL, Morrison S, Lucas CE, Ledgerwood AM. Early sympathetic blockade for frostbite-is it of value? *J Trauma* 1980;20:744-749.
67. Porter JM, Wesche DH, Rosch J, Baur GM. Intra-arterial sympathetic blockade in the treatment of clinical frostbite. *Am J Surg* 1976;132:625-630.
68. Kaplan R, Thomas P, Tepper H, Strauch B. Treatment of frostbite with guanethidine [letter]. *Lancet* 1981;October:940-941.
69. Snider RL, Porter JM. Treatment of experimental frostbite with intra-arterial

sympathectic blocking drugs. *Surgery* 1975;77;557.
70. Snider RL, Merhoff GC. Intra-arterial sympathetic blockade in the treatment of frostbite. *Surg Forum* 1974;25:237.
71. Blair JR, Schatzki R, Orr KD. Sequellae to cold injury in one hundred patients, follow up study four years after occurrence of cold injury. *JAMA* 1957;163:1203-1208.
72. Kumar VN. Intractable foot pain following frostbite: case report. *Arch Phys Med Rhabil* 1982;63:284-285.
73. Huh J, Wright R, Gregory N. Localized facial telangectasias following frostbite injury. *Cutis*. 1996;57:97-98.
74. Arvesen A, Wilson J, Rosen L. Nerve conduction velocity in human limbs with late sequelae after local cold injuyr. *Eur J Clinical Invest* 1996;26:443-450.
75. Carrera GF, Kozin F, McCarty DJ. Arthritis after frostbite injury in children. *Arthritis Rheum* 1979;22:1082-1087.
76. McKendry RJR. Frostbite arthritis. *CMA J* 1981;25:1128-1130.
77. Brown FE, Spiegel PK, Boyle WE. Digital deformity: An effect of frostbite in children. *Pediatrics* 1983;71(6):955-959.
78. Murray JJ. Pediatric aspects of nordic skiing. *Ped Clin N Am* 1982;29(6):1423-1429.
79. Kemp SS, Dalinka MK, Schumacher HR. Acro-osteolysis: etiologic and radiologic considerations. *JAMA* 1986;255:2058-2061.
80. DiBartolomeo JR. The petrified auricle: comments on ossification, calcification, and exostoces of the external ear. *Laryngoscope* 1985;95:566-576.
81. American College of Sports Medicine Position Stand: Heat and cold illnesses during distance running. *Med and Science in Sports & Exercise* 1996;28:i-x.
82. Roberts WO. When is it too cold to exercise? *Your Patient and Fitness in Internal Medicine* 1991;5:15-19.
83. Danielsson U. Windchill and the risk of tissue freezing. *J Appl Physiol* 1996;81:2666-2673.

Physician CME Questions

1. Chilblain is a neurocirculatory skin disturbance that is primarily seen on the:
   a. face.
   b. feet.
   c. fingers.
   d. ears.
   e. All of the above

2. Chilblain occurs with repeated exposure of bare skin to cold, wet, and windy weather ranging from:
   a. -10°C to -1°C
   b. 0°C to 15.5°C
   c. 16°C to 20°C
   d. 21°C to 25°C

3. At what temperature does tissue begin to freeze?
   a. 5°C
   b. 0°C
   c. -3°C to -4°C
   d. -7°C

4. Military studies have demonstrated that blacks are from 2.8 to 6 times more susceptible to a cold injury, while caucasian southerners are more than:
   a. 20 times more susceptible than those from the northern-most states.
   b. 5.6 times more susceptible than those from the northern-most states.
   c. 2 times more susceptible than those from the northern-most states.
   d. 3.7 times more susceptible than those from the northern-most states.

5. Under no circumstances should the extremity be allowed to refreeze.
   a. True
   b. False

6. Other conditions that should be considered in children with frostbite include:
   a. dehydration or hypovolemia.
   b. anoxia.
   c. hypoglycemia.
   d. carbon monoxide poisoning.
   e. All of the above.

7. The current preferred treatment for deep frostbite is rapid rewarming in a water bath at temperatures of:
   a. 20-25°C.
   b. 26-31°C.
   c. 32-44°C.
   d. 45-50°C.

8. The prevention of frostbite requires:
   a. good physical conditioning.
   b. good clothing.
   c. adequate rest.
   d. prevention of illness and trauma.
   e. All of the above.

In Future Issues: Muscle Weakness