Chapter 2

COLD-INDUCED INJURY

DAVE CORBETT, D.O.* AND PAUL BENSON, M.D.†

*Captain, Medical Corps, U.S. Navy; Dermatology Branch, National Naval Medical Center, Bethesda, Maryland 20814
†Lieutenant Colonel, Medical Corps, U.S. Army; Dermatology Service, Walter Reed Army Medical Center, Washington, D.C. 20307-5001

INTRODUCTION

Cold injuries have played an important role in the loss of combat effectiveness for armies since early recorded history. References to frostbite have been found in the writings of Hippocrates, Aristotle, and Galen. Other historical entries have documented significant problems with cold injuries among the Greek armies of the late fourth and early third centuries BC.[1]

During the American Revolution, James Thatcher described serious losses from cold injury in 1777; an army of 10,000 men lost 2,900 to combat operations. Even in 1777, there was some understanding of the pathogenesis of cold injury. A physician general to the military hospitals, Benjamin Rush, wrote in a small pamphlet with directions for preserving the health of soldiers:

> The commanding officer should take the utmost care never to suffer a soldier to sleep, or even to sit down in his tent with wet clothes, nor to lie down in a wet blanket or upon damp straw. The utmost vigilance will be necessary to guard against this fruitful source of diseases among soldiers.[1(pp29–30)]

Baron Larrey, surgeon to the French armies in the Napoleonic Wars, described frostbite and "congelation" as important reasons for the defeat of the army in Poland in 1812. He also noted that "general remedies should always precede surgery"[1] and described the detrimental effects of sudden rapid warming of frozen body parts by the camp fire.

The Crimean War (1854–1856) revealed the impact of proper training and equipment on the number of casualties caused by cold. During the first winter (1854–1855), British troops fought trench warfare, with static defensive positions. Soldiers were inexperienced and unfamiliar with the potential hazards of cold weather. They were also hampered by a lack of adequate food and clothing, and the debilitating effects of diarrhea and dysentery. In a force of slightly less than 50,000 soldiers, 1,924 cases of cold injury were reported, with 457 fatalities—23.8% of the total cold injuries. During the winter of 1855 to 1856, there were only 474 cases of cold injury and 6 fatalities (1.3%). Weather conditions and precipitation were essentially the same in both years. But during the second year the troops had far better living conditions along with improved winter clothing and ample food. The average soldier was hardened and knew better how to care for himself and prevent cold injury.[1]

In the Franco-Prussian War of 1870, 1,450 cases of severe frostbite occurred among 92,067 Prussian troops. During the Russo-Turkish War (1877–1878), 4,500 frostbite casualties were reported, representing 1.5% of the 300,000-man force in Bulgaria and 5.1% of the 87,989 casualties evacuated.[1]

British medical observers with the Japanese Army made detailed reports from 25–29 January 1905 during the Russo-Japanese War. Of 7,742 total casualties, 505 soldiers were hospitalized for frostbite, approximately a 1:15 ratio of frostbite to battle wounds. The toes were affected in 67% of the cold injury cases and the fingers in 28%. Injuries were mild and amputation was seldom required. In a subsequent engagement, the British observers noted that the Japanese troops were given extra socks and rations. Halts were called during marches to remove boots and replace them with Chinese felt or straw shoes. The number of frostbite casualties dropped dramatically to only 70 soldiers hospitalized for cold injury.[1]

The most detailed history of cold injuries among combat soldiers can be gleaned from World War I. The British experience is the most extensive, and U.S. casualties were lower as a result of lessons learned before our involvement. Additionally, most of the trench warfare for U.S. forces occurred during times of the year when exposure to cold and wet was not great. The British, however, did not seem to profit from their experience in the Crimean War and, as a result, their expeditionary force suffered a high rate of cold injury during the first winter of World War I.[1] Injuries were reported as frostbite, water bite, footbite, cold bite, puttee bite, trench bite, chilled feet, or only as "feet cases."[1] Not until after the first winter did the term "trench foot" come into general usage. Medical and nonmedical officers promptly realized the significance of this affliction, which is caused by prolonged exposure to cold and dampness at temperatures above freezing. Forces suffered not only loss of manpower but the additional financial consideration of casualties that were entitled to disability pensions.[1]

Cold injury began insidiously among British troops with one case of frostbite in August 1914, one in September, 11 in October, 1,555 in November, and 4,823 in December, when the term "trench foot" first appeared in medical records. The highest incidence of cold injury was in the infantry, with officers affected slightly less severely than enlisted

soldiers. The total number of cold injuries in all theaters of operations and bases including the United Kingdom was 115,361 cases. During 1915, the cases of frostbite and trench foot were separated (they were not thereafter), with 30,691 admissions for frostbite and 29,172 for trench foot. Total cold injuries for 1914 to 1915 numbered 97,414 cases. Only 443 cases were noted in the hospital records of 1916 to 1918.[1]

The American records for World War I reveal a total of 2,061 admissions for trench foot, which included 27 officers. The total number of man-days lost was 92,249, an average of 45 days per case. Preventive measures included regular inspections of the feet by officers, as well as changing socks once daily, foot exercises, dry clothing, and a nutritious diet.[1]

The lessons from World War I were forgotten and the United States sustained many casualties in World War II before the problem was again taken seriously. Cold injuries ranged from high-altitude frostbite injuries to trench foot and immersion foot injuries suffered by ground troops from the Mediterranean and European theaters to the Aleutian Islands. Lessons learned in one theater or service were not used to full advantage in other theaters.[1]

The Eighth Air Force could attribute varying proportions of the total number of casualties in airborne personnel to high altitude frostbite from the beginning of their operations in 1942 to the end of conflict in May 1945. During fiscal year 1943 to 1944, more crew members sustained wounds from cold than from enemy fire. These losses were even more significant in that one third required hospitalization, with an average loss of 4 to 14 days of duty even for mild injuries. Of these, many required months of recuperation and many were unable ever to return to duty.[1] For one 14-month period ending in December 1943, 1,634 men were removed from flying status as a result of cold injuries. During this same period, 1,207 men were removed from flying status because of injuries sustained from enemy action. In 1944 the number of casualties increased because of increased combat operations, but the percentage of losses from cold injuries decreased presumably because of lessons learned as well as improved equipment and training.[1]

In the Mediterranean theater, during the winter of 1943 to 1944, combat ground losses from cold injuries, mainly trench foot, were significant. For the 6-month period ending 30 April 1944, there were 5,700 cold casualties of 27,602 wounded in action, or an approximate 1:5 ratio of cold-to-combat loss, for the Fifth U.S. Army alone.[1] These losses become even more significant when we realize that almost all these injuries occurred among combat troops, the most difficult to replace. Many of these troops were never able to return to full duty. Of those who did return to duty, about 60% were casualties the following winter, as recurrent trench foot was a significant problem. Improved clothing and education and changes in the daily routine (such as changing socks at least daily and providing laundry and exchange of dirty socks for clean socks) were instrumental in reducing these numbers the following winter. The British had much lower cold casualty rates yet they fought in the same weather conditions and intensity of combat as the American forces. The British had troop rotation, much stricter enforcement of foot care, and better clothing than the Americans. Their cold-to-combat loss ratio was 1:45, compared to a 1:4 ratio for the Americans during the same period.[1]

The bitter lessons learned by the Fifth U.S. Army in Italy during 1943 to 1944 unfortunately did not reduce cold injuries in the European theater. Records show 46,000 cold injuries in the European theater from autumn 1944 to spring 1945, or about 5% of all hospital admissions for medical treatment. In the Mediterranean theater, the rate had dropped to 1.3% of admissions from 4% the previous winter. Cases peaked the week ending 17 November 1944 with 5,386 cases, with another smaller peak of 3,213 cases appearing during the week ending 29 December 1944, the Battle of the Bulge. The incidence of frostbite was much higher during December, with more cases of trench foot in November. After the spring thaw began 1 February 1945, the character of the epidemic returned to increasing numbers of trench foot cases. During November and December 1944, there were an astonishing 23,000 cases of cold injury on the Western Front. In terms of combat riflemen (4,000 to a division), the loss amounted to about 5.5 *divisions*.[1]

Changes in clothing types and design had occurred because of U.S. experiences in Italy during the winter of 1943 to 1944, but requisitions were made too late and clothing did not arrive in theater until after the worst of winter was over. Poorly fitting and poorly insulated shoes, a lack of adequate socks, and poorly fitting outer garments all played a role in producing the large number of cold casualties sustained in the European theater. The mistaken belief that the war would end before winter arrived in 1944 was a factor in not requisitioning the necessary cold weather clothing.[1]

During the Korean conflict, experiences of previous wars were once again forgotten, and 9,000 cold

injuries were reported, mostly frostbite.[2] Of these injuries, 8,000 occurred in the winter of 1950 to 1951.[2] Inactivity such as often occurs in heavy combat seemed to be a prime factor in developing cold injuries. In one study of over 1,000 cases, 67% of cold-injured individuals had been pinned down by enemy fire, sleeping in a foxhole, or riding in a truck.[3] Comparing injuries from the winters of 1950 to 1951 and 1951 to 1952 shows a decrease not only in numbers, but in severity as well. Nearly 50% of frostbite injuries were third and fourth degree during 1950 to 1951 compared to only 25% during 1951 to 1952.[3] This lessened severity seems best related to the change in U.S. military position during the second winter from more active combat to a more static defensive position.[3] One third of the cold injuries during the winter of 1951 to 1952 occurred during the period 22 to 26 November during an enemy attack. American troops were changing positions previously held by Republic of Korea personnel and were pinned down.[3] To show the importance of combat tempo on the cold casualty rate, Orr[4] noted the following statistics from the winter of 1950 to 1951, based on 320 soldiers from one unit admitted to Osaka Army Hospital over a 90-day period: days with no enemy contact had an average of 0.7 admissions per day; light contact, 1.2 admissions per day; moderate contact, 6.4 admissions per day; and heavy action, 9.3 admissions per day.

History has repeatedly demonstrated the devastating effects of underestimating the impact of cold weather and of failing to plan for cold weather injuries. The British may have had fewer casualties because of the way they approached the prevention of cold injuries. Their view was that cold injuries were a result of poor leadership rather than a need for better medical care. Cold injuries, like heat injuries, are avoidable in all but the most intense combat situations. Close supervision, constant education, and adequate equipment are essential to avoiding cold injuries (Exhibit 2-1). As medical professionals, our duties and obligations are to provide the best educational support to the line unit commanders as well as to provide the best treatment for injuries.

EXHIBIT 2-1

PREVENTION OF COLD INJURIES

Leadership
Buddy system
Instruction and training
Appropriate clothing
Protection of skin from contact with metals or liquids
Advance meteorologic data
Frequent troop rotation
Adequate diet and hydration
Adequate rest

Data source: Corbett D. Cold injuries. *Journal of the Association of Military Dermatologists*. Fall 1982:8(2):34–40.

MECHANISMS OF HEAT LOSS

Cold injury is the damage to tissue produced by heat loss, resulting from exposure to cold. This loss can occur by several mechanisms: conduction, convection, radiation, evaporation, and respiration.

Conduction

Conduction loss is heat loss by direct contact with a cold object. This loss is infrequent but can be a severe source of injury. Contact with cold metal is a common injury. Liquids such as gasoline and other solvents cause rapid evaporative cooling because of their low freezing points, which can result in instant frostbite on contact in subfreezing temperatures. Heat loss by conduction may occur up to 32-fold faster in water than in air.[5] The source of the moisture can be perspiration, water, or other liquids.

Convection

Convection loss occurs when air currents dissipate the thin warm air layer that surrounds us. Body hair and clothing help to maintain this warm air layer. Wind chill is important in that the faster the wind blows, to a maximum of 40 mph, the faster the protective layer of warm air is removed (Figure 2-1).

Radiation

Radiation of heat occurs from exposed body surfaces. The hands, face, head, and neck are the most commonly exposed sites. At 4°C approximately 50% of body heat can be lost from an uncovered head and at –15°C this loss may increase to 75%.[5] Proper headgear can help elevate body tempera-

Wind Speed (mph)	Actual Temperature (°F)											
	50	40	30	20	10	0	–10	–20	–30	–40	–50	–60
	Equivalent Chill Temperature (°F)											
Calm	50	40	30	20	10	0	–10	–20	–30	–40	–50	–60
5	48	37	27	16	6	–5	–15	–26	–36	–47	–57	–68
10	40	28	16	3	–9	–21	–33	–46	–58	–70	–83	–95
15	36	22	9	–5	–18	–32	–45	–58	–72	–85	–99	–112
20	32	18	4	–10	–25	–39	–53	–67	–82	–96	–110	–124
25	30	15	0	–15	–29	–44	–59	–74	–89	–104	–118	–133
30	28	13	–2	–18	–33	–48	–63	–79	–94	–109	–125	–140
35	27	11	–4	–20	–35	–51	–67	–82	–98	–113	–129	–145
40	26	10	–6	–22	–37	–53	–69	–85	–101	–117	–132	–148
Wind speeds > 40 mph have little additional effect	**Little Danger** (in < 5 h with dry skin; greatest hazard is from false sense of security)				**Increasing Danger** (exposed flesh may freeze within 1 min)				**Great Danger** (exposed flesh may freeze within 30 sec)			

Fig. 2-1. Potential heat loss, skin cooling, and lower internal temperature can be increased by air movement. The wind-chill index integrates windspeed and air temperature to estimate associated risk of cold injury. The wind-chill temperature index is the equivalent still-air (no wind) temperature that would produce the same heat loss on bare skin. A full description of the medical aspects of military operations in the cold is the subject of US Army Research Institute of Environmental Medicine (USARIEM) Technical Note 92-2, *Sustaining Health and Performance in the Cold.* Source of chart: US Army Research Institute of Environmental Medicine Technical Note 92-2. *Sustaining Health and Performance in the Cold: Environmental Medicine Guidance for Cold-Weather Operations.* Natick, Mass: USARIEM; July 1992: 37.

ture by reducing heat loss; by removing headgear during heavy exercise, soldiers can increase heat loss.

Evaporation

Evaporation that occurs when water vapor is released from the skin is an important form of heat loss. Clothing that allows water vapor to escape helps to conserve heat because wet skin requires increased heat loss to dry the skin. This loss of heat keeps us alive in hot climates, but is detrimental in a cold environment.

Respiration

Respiration is an additional source of heat loss and results from breathing out humidified, warm air. Heavy exercise results in rapid breathing and significant loss of heat and water. Covering the mouth with a mask (eg, wool) can retain some of the exhaled heat and prewarm the incoming air.[3]

FACTORS INFLUENCING HEAT LOSS

Multiple factors can contribute to heat loss and the risk of cold injury. These factors include the degree of cold encountered, intensity of combat, protective clothing available, and others. Combat conditions often involve several of these factors and leave military and involved civilians at high risk for cold injuries.

Weather and Duration of Exposure

Short exposure (ie, only a few minutes) to intense cold, especially with high winds, moisture, or skin contact with metal or volatile solvents, can lead to frostbite, whereas prolonged exposure to higher temperatures contributes to trench foot, immersion foot, and pernio. The average duration of exposure resulting in frostbite is 10 hours. During the Korean conflict, 80% of the cases in 1950 to 1951 reported a duration of exposure of 12 hours or less, with a range of 2 to 72 hours.[4] Trench foot and immersion foot occurred in as little as a few hours or as long as 14 days, with an average of 3 days.[2]

Type of Combat Action

Units on active defense, under attack, or on the attack are at the greatest risk of cold injury. Static situations do not allow for movement and lead to prolonged exposure. Active combat and defense increase fatigue, and often do not allow for rewarming, clothing changes, or proper nutrition.

Clothing

Modern cold weather clothing is based on the "layering" principle and employs a wind- and water-resistant outer layer. Multiple layers of loosely fitting clothing make use of the insulating properties of dead air spaces. This looseness extends to the boots as well. Footgear must be loose enough to allow for thick socks and not constrict the circulation and yet must be water-resistant. As exercise increases, clothing layers must be removable to allow for heat loss equal to the increased output. Wearing or not wearing a hat, as previously noted, can make a significant difference. Newer water-vapor–permeable outer garments permit the escape of moisture, which helps preserve the garment's insulating capability. Finally, mittens are more protective than gloves; the individual finger slots in gloves increase surface-area heat loss.

Other Factors

Several other factors can significantly increase the risk of suffering cold injuries:

- Young children and the elderly have greater susceptibility to cold injuries.
- Junior enlisted ranks are more susceptible to cold injury than senior enlisted ranks and officers because of their inexperience and increased exposure to cold. They may also be less receptive to preventive training.
- Previous cold injury predisposes to reinjury.
- Fatigue leads to apathy, improper wearing of clothing, and neglect of hygiene (eg, keeping feet dry).
- Lack of adequate troop rotation can increase the risk of cold injury.
- Combat injuries can be complicated by shock and decreased blood flow, further increasing the risk of cold injury.
- Race is an important risk factor, with blacks being 2- to 6-fold more vulnerable to frostbite than whites.[6,7]
- Overactivity leads to excessive perspiration and wetting of clothing, resulting in its loss of insulating capacity. However, underactivity is equally detrimental, causing stasis and lack of heat generation (Exhibit 2-2).

EXHIBIT 2-2

COLD INJURY RISK FACTORS

Inadequate clothing
Harsh weather (low temperatures, high winds, moisture)
Active combat or defense
Lack of troop rotation
Decreased blood flow from combat injuries
Age (children and elderly)
Low military rank
Previous cold injury
Fatigue
Discipline and training
Psychosocial factors (eg, homeless or mentally ill out in the cold)
Race
Geographic origin
Poor nutrition
Erratic physical activity
Vasoconstricting medications
Use of alcohol

Data source: Corbett D. Cold injuries. *Journal of the Association of Military Dermatologists*. Fall 1982;8:34–40.

PATHOGENESIS OF COLD INJURY

Several mechanisms are responsible for cold injury with its subsequent tissue damage and loss. Cellular damage can occur from intracellular ice crystal formation as tissue freezes. Other mechanisms of injury are believed to be secondary to vascular damage with resulting microcirculatory failure and tissue hypoxia. This leads to clumping of erythrocytes and capillary stasis. Irreversible occlusion of small vessels by aggregates of cells with thrombus formation has been demonstrated in rewarmed tissue after freezing. In addition, as a result of tissue hypoxia and possibly the direct effect of cold, there is an increase in capillary permeability with loss of plasma into the extravascular space. These events lead to further hemoconcentration, increased viscosity, and stasis.[8]

Cold causes direct metabolic impairment, affecting sensitive cellular enzyme systems adversely and impairing cellular function. Injuries resulting from cold exposure can be classified as direct and indirect.

DIRECT COLD INJURY

Direct cold injuries are caused by exposure to low temperatures and are not associated with exacerbation of an underlying disease. Examples of direct cold injury include asteatotic eczema and frostbite.

Asteatotic Eczema

Asteatotic eczema is a pattern of skin that is dry, scaly, rough, and less flexible than normal, often with a cracked appearance. The dermatosis is more frequently seen in the elderly, though quite common in young adults, and is aggravated by cold. Dry pruritic skin leads to scratching, excoriations, and often secondary infection. The most common site is the anterior lower legs (Figure 2-2).

Etiology

The dry skin of asteatotic eczema is due to decreased hydration of the stratum corneum. Decreasing sebaceous secretion, which occurs with aging, results in insufficient lipid to maintain water within the stratum corneum. Atmospheric conditions, cold, and low humidity play definite roles in developing dry skin. Poor nutrition is also a factor.[9]

Clinical Manifestations

Scaling and pruritus is prominent in asteatotic eczema, particularly on the lower legs, but any body area can be involved. With increasing severity, fissuring in a lacework pattern and follicular hyperkeratosis eventually occur. Rubbing and scratching can lead to lichenification of affected areas.

Treatment

Lubrication is the mainstay of therapy for the dry skin associated with asteatotic eczema. Any moisturizer is acceptable, and common petrolatum is excellent and usually readily available. Applying after a shower will help seal water in the skin.

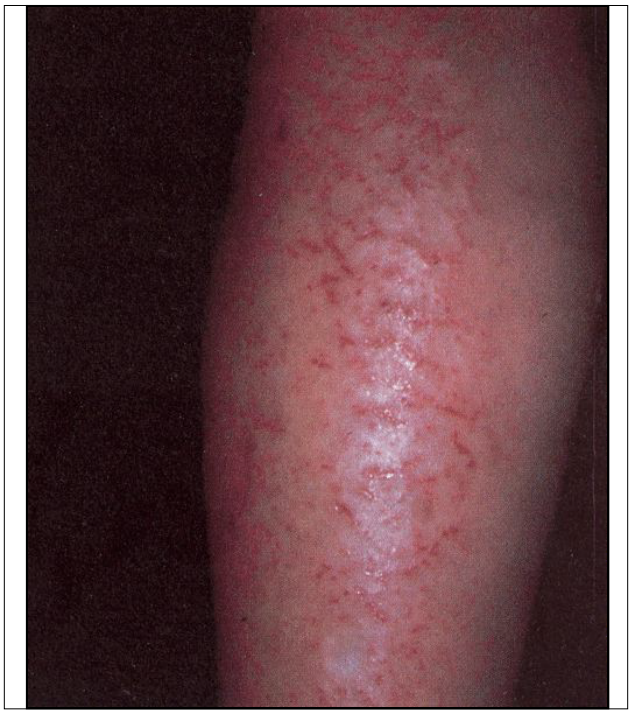

Fig. 2-2. An example of typical asteatotic eczema on a lower extremity. Note the almost fish-scale appearance and mild lichenification secondary to scratching.

Frostbite

Frostbite is destruction of local tissue induced by temperatures below freezing. All the predisposing factors mentioned in Exhibit 2-2 may interact and lead to injury.

Etiology

Frostbite is caused by both the actual freezing of tissue and the subsequent vascular changes that occur. The human body's initial response to cold exposure is vasoconstriction of skin vessels to reduce heat loss and conserve core temperature. The vasoconstriction persists with continued cold exposure. Blood vessel walls and endothelial cells[10] are altered with increased permeability and sludging of blood. Arteriovenous shunting eventually occurs and areas of tissue are bypassed and devitalized. Rapid freezing leads to the formation of intracellular ice crystals and consequent cell damage. Proteins are denatured and enzyme systems impaired. Cells dehydrate when they are damaged by cold, and water moves into the extracellular spaces.[8]

Clinical Manifestations

Frostbite is separated into four categories, which will be discussed in increasing order of severity: first-degree frostbite, or frostnip; second-degree or superficial frostbite; and third- and fourth-degree or deep frostbite.

First-Degree Frostbite (Frostnip). Frostnip is the superficial freezing of skin, often facial skin or fingertips, which becomes blanched and numb. If no further cold exposure occurs and the area is rewarmed, no permanent damage or tissue changes ensue. There can be redness, itching, and mild edema beginning within 3 hours after thawing and lasting up to 10 days.[2,8,11]

Second-Degree Frostbite (Superficial Frostbite). Superficial frostbite freezes the skin and subcutaneous tissues but spares the deeper structures, which are still soft to deep palpation. Blanching and numbness are present. As the tissue thaws, the patient develops pain, erythema, and swelling of the affected sites. The skin can take on a mottled cyanotic appearance (Figure 2-3). Blisters may form (Figure 2-4), usually within 24 to 48 hours but sometimes as soon as 6 hours after thawing. These blisters are clear and often extend to the tips of digits, which is considered a good prognostic sign (Exhibit 2-3). Only ruptured blisters should be debrided. Over the next several weeks, the tissue may mummify, turn black, and slough, revealing red, atrophic new skin (Figures 2-5 and 2-6). Hyperhidrosis often occurs by the second to third week, and long-term cold sensitivity can also arise.

Third- and Fourth-Degree Frostbite (Deep Frostbite). Both third- and fourth-degree frostbite involve loss of deep tissue. Third degree frostbite involves freezing of skin and subcutaneous tissue with tissue loss and ulceration. With thawing, vesicles may form, but they are often smaller and hemorrhagic and do not extend to the tips of digits. After early anesthesia, severe pain can begin within a few days. Hard black eschars form and separate over several weeks, leaving a granulation base (Figure 2-7). Healing time averages 68 days, and

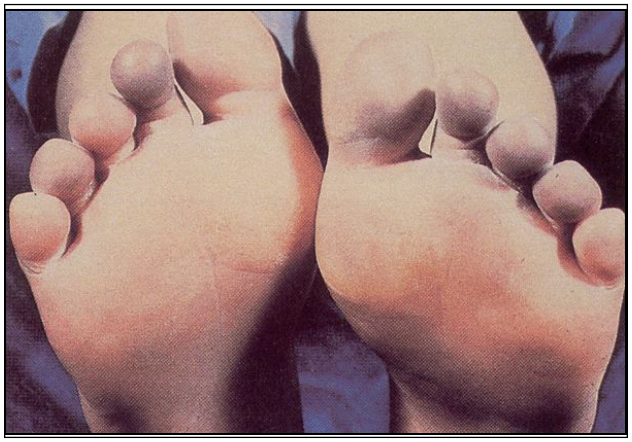

Fig. 2-3. Early appearance of either superficial or deep frostbite. Note the mild edema and cyanotic appearance of the toes.

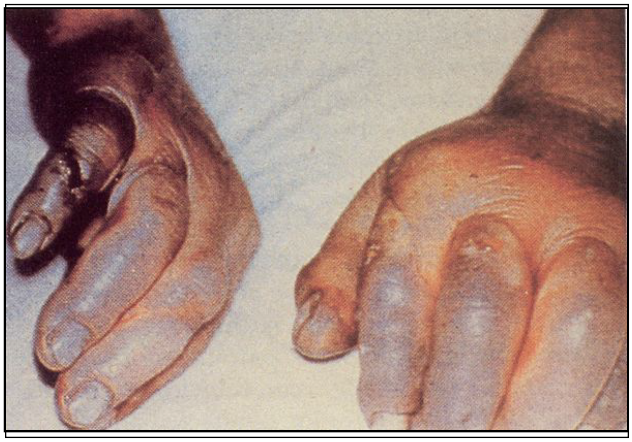

Fig. 2-4. Superficial frostbite with large blebs extending to the fingertips.

EXHIBIT 2-3

PROGNOSTIC SIGNS OF FROSTBITE

Favorable Indicators

- Large, clear blebs extending to the tips of the digits
- Rapid return of sensation
- Rapid return of normal (warm) temperature to the injured area
- Rapid capillary filling time after pressure blanching
- Pink skin after rewarming

Unfavorable Indicators

- Hard, white, cold, insensitive skin
- Cold and cyanotic skin without blebs after rewarming
- Dark, hemorrhagic blebs
- Early evidence of mummification
- Constitutional signs of tissue necrosis, such as fever and tachycardia
- Cyanotic or dark red skin persisting after pressure
- Freeze-thaw-refreeze injury

Data source: US Department of the Navy. *Cold Injury*. Washington, DC: DOD; NAVMED P-5052-29. March 1970: 1–14.

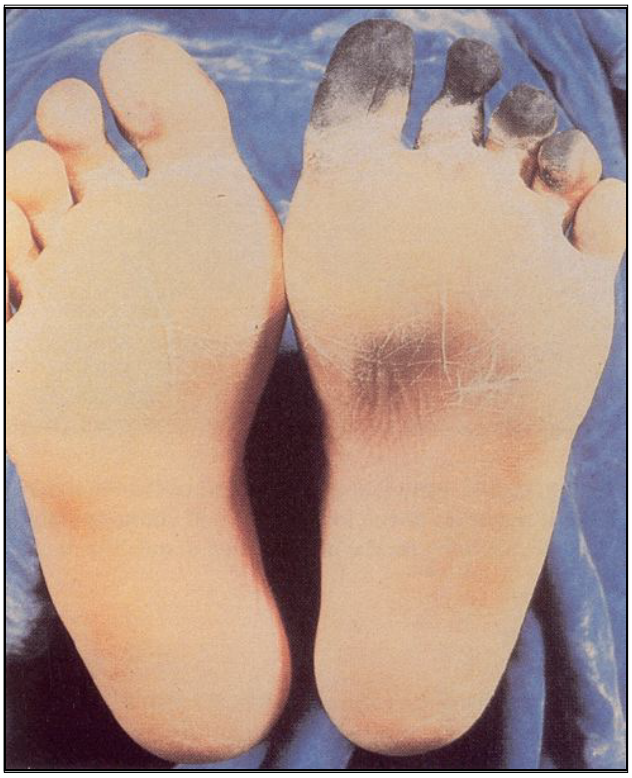

Fig. 2-5. Same patient seen in Figure 2-3, approximately 6 weeks later, showing demarcation.

hyperhidrosis, which can appear at 4 to 10 weeks, may persist for months.[2]

Fourth-degree frostbite involves complete necrosis and loss of deep tissue including bone (Figure 2-8). The deeper tissues are solid to deep palpation. With rewarming, the skin can become purple to red and anesthetic. Edema is usually found proximal to the area of fourth-degree injury, reaching a peak by 6 to12 hours.[2] The area may then rapidly progress to a dry type of gangrene with lines of demarcation noted as early as 72 hours post-thaw.[11] A slower progression also can occur, with eschar and gangrene formation not evident until 2 to 3 weeks after thawing. Severe pain and intense burning will occur with thawing, and large amounts of analgesics may be needed.[9] Paresthesias appear 3 to 13 days after rewarming, lasting for at least 6 months in more than 50% of frostbite victims.[12] The line of demarcation becomes apparent in an average of 36 days and extends to bone 60 to 80 days after injury.[2] Long-term frostbite sequelae consist of cold sensitivity, paresthesia, numbness, pain, and hyperesthesia.[12] Other problems include frostbite arthritis, which occurs weeks to years later and resembles osteoarthritis,[13] and hyperhidrosis.[9]

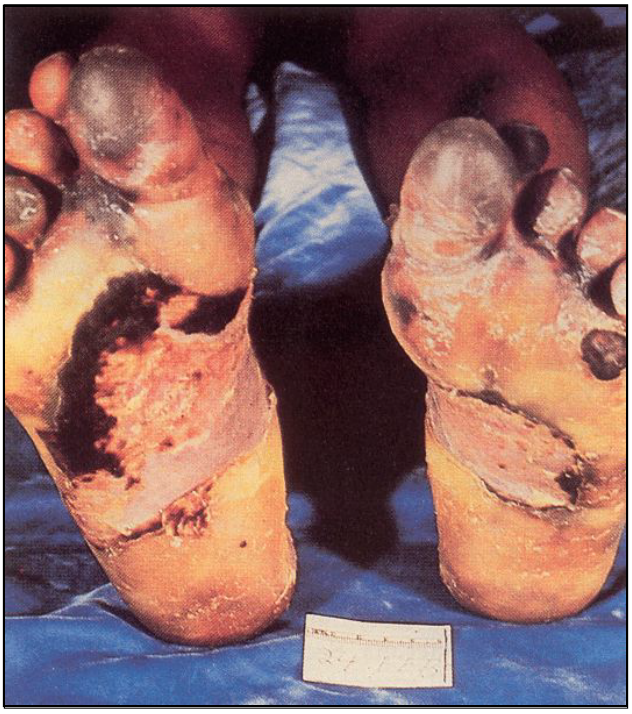

Fig. 2-6. Early presentation of superficial frostbite. No loss of digits took place. Note shallow ulcerations on plantar aspect of feet.

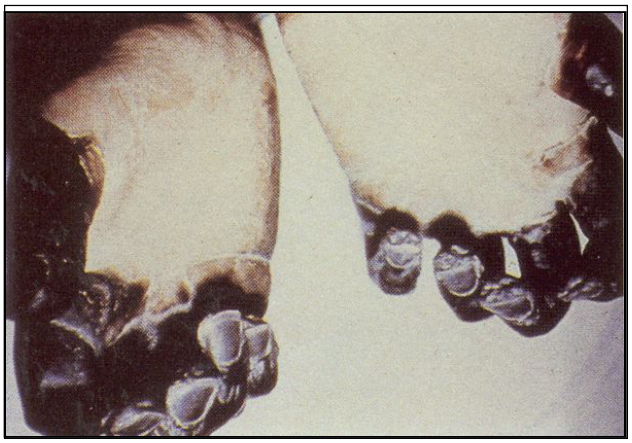

Fig. 2-7. Hands with deep frostbite and well-demarcated areas of eventual tissue loss. These will autoamputate if left alone, allowing for the preservation of maximal tissue.

Treatment

Prevention of frostbite is the best form of therapy. Frostnip can be treated immediately in the field. Rewarming can be accomplished by placing fingers in the axilla, blowing warm air over the frozen surfaces, or placing a warm hand on the area. Recovery is usually prompt and no long-term therapy is necessary.

Frozen parts should not be thawed until definitive care is available (ie, no possibility of refreezing). Once thawed, the part is painful, and if the feet are involved, the individual will be unable to walk. Freeze-thaw-refreeze cycles are also very damaging and must be avoided. When treating military personnel, one must consider the availability of definitive care. If transportation is not available, the soldier with frostbite may have to continue to fight or walk out of the combat area. Neither action would be possible if frostbitten feet were permitted to thaw.

The more serious types of frostbite are treated by rapid rewarming of the affected areas in a warm-water bath at 40°C to 42°C until the most distal part is flushed.[4] Rewarming is painful and analgesia is required. Bed rest, elevation of the affected extremity, and protection of the injured area with twice daily antiseptic whirlpool baths for gentle debridement are essential. Tetanus toxoid should be given to all victims. Secondary infection is common, but not all sources recommend prophylactic antibiotics. Smoking is not permitted. Early

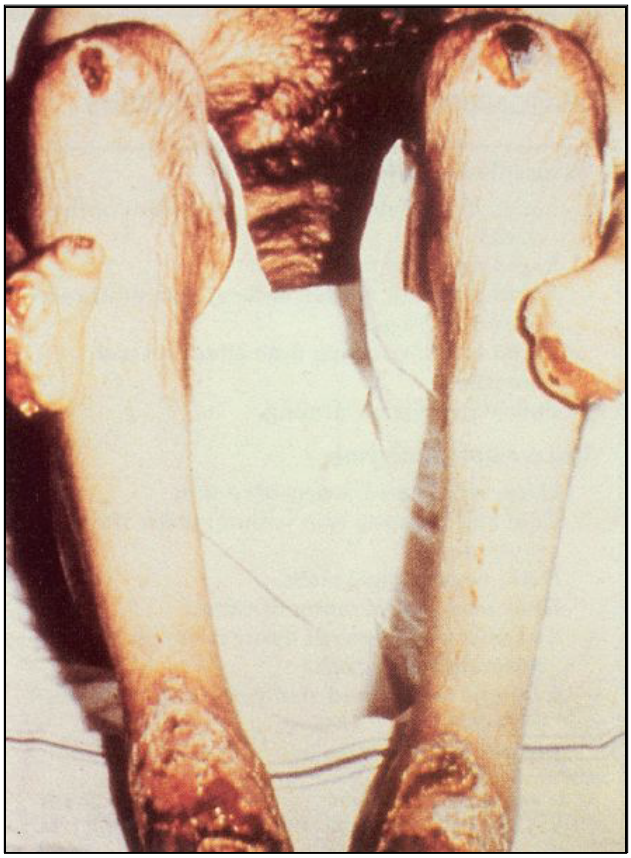

Fig. 2-8. This hobo suffers from deep frostbite. He was locked in a freezer car for approximately 18–24 hours, with these unfortunate results.

heparinization, begun within 36 hours[2] and stopped in 2 to 3 days,[11] has been shown to be of help, but anticoagulation of individuals in a field setting may be impossible and there may be other trauma to consider.[2] Low molecular weight dextran, 1.5 gm/kg intravenously on the first day, then 0.75 mg/kg intravenously daily for 5 days, has been shown to decrease blood viscosity and increase tissue perfusion in limited trials.[14] Intraarterial reserpine, 0.25 to 0.5 mg, can be used if vascular spasm is noted on angiogram at about the 10-day point, with repeat angiogram in 2 days.[11] Surgical sympathectomy has been used late in the healing process for severe cases[8] but its use is not universally recommended.[6] Both extensive surgical debridement and amputation are to be avoided until devitalized tissue is clearly demarcated and spontaneous sloughing has occurred, which may take months.[9] Moist gangrene with infection is, of course, an exception to this rule.

INDIRECT COLD INJURY

Indirect cold injury is associated with exposure to low temperatures as well as other factors, and is often linked to disease processes that are exacerbated by cold. Finding skin lesions after cold exposure can lead to the discovery of underlying disease.

Pernio

Pernio, or chilblain, refers to the development of bluish red patches on arms and legs that are chronically exposed to cold, damp climates. A mild form of cold injury, pernio was described as early as 1680.[8] This condition is prevalent in England and is less commonly seen in very cold climates where heating is usually more adequate and protective clothing is worn on a regular basis.[9]

Etiology

Vasospasm is the primary mechanism causing pernio in predisposed individuals who appear to have an inherently high peripheral vascular tone. The nonfreezing cold is amplified by high humidity. The vasospasm produces local tissue hypoxia, which leads to the initial cyanotic and erythematous skin lesions.[8]

Histological evaluation of pernio reveals edema of the papillary dermis and a superficial or both superficial and deep perivascular lymphocytic infiltrate. Necrotic keratinocytes and lymphocytic vasculitis have also been reported.[9]

Clinical Manifestations

Pernio is divided into *acute* and *chronic* disease. The acute form is seen primarily in children and young adults. The chronic form is seen most commonly in adults. Acute pernio presents with bluish red, slightly edematous patches, most commonly on the lower extremities. Prolonged exposure to damp cold with inadequate protection precedes the development of lesions. The skin findings persist for 7 to 10 days or less, and resolve spontaneously with a residual brownish red hyperpigmentation in many cases.[8]

Chronic pernio—edematous, cyanotic lesions with secondary ulceration and hemorrhage—is seen after repeated episodes of acute pernio. Patients can present with subcutaneous nodules accompanied by a burning sensation, pruritus, or both. Acute pernio is more common in winter, but with repeated episodes lesions may become persistent, with the atrophy and ulcerations lasting throughout the year. The differential diagnosis includes nodular vasculitis, erythema induratum, erythema nodosum, and livedo vasculitis with ulcerations.[8]

Treatment

Protective clothing and warmth are the mainstay of therapy for pernio. Emollient creams can be of benefit. Antibiotics and topical care may be needed if ulcerations or secondary infection is present. Vasodilating drugs can be useful in some patients. Nifedipine has been effective at a dose of 20 mg three times daily.[9] Sympathectomy has been utilized in extreme cases only, but does not prevent recurrences.[8]

Livedo Reticularis

Livedo reticularis is a persistent reticulated pattern of red to bluish coloration of the skin (Figure 2-9). The skin mottling can be transient as seen with infants exposed to cold and is known as *cutis marmorata*. If the pattern persists after rewarming then the term *livedo reticularis* is used. Congenital livedo reticularis (cutis marmorata telangiectatica congenita)[15] results from several inherited syndromes, such as Down's syndrome, Cornelia de Lange syndrome, homocystinuria, and neonatal

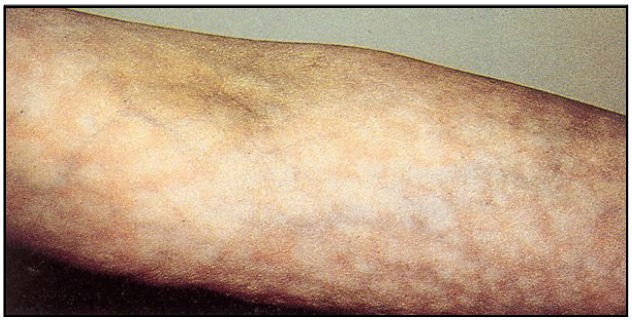

Fig. 2-9. Livedo reticularis. The netlike vascular pattern is typical of the disease.

lupus erythematosus. These disorders display persistent forms of livedo that are not related to cold exposure. The causes of acquired livedo reticularis are numerous (Exhibit 2-4). It can be benign, as with cold, or may be a sign of serious underlying disease such as vasculitis, connective tissue disease, or malignancy. Livedo derives from the Latin *liveo*, meaning "blue, black, or livid." The term was first used by Hebra in 1868.[8]

Etiology

Similar physiological explanations for the livedo pattern were given by Renault (1883), Unna (1896), and Spalteholz (1927).[16] It was postulated by all three authors that the cutaneous circulation is arranged in "cones," 1 to 3 cm in diameter, with the apex deep in the dermis and a central ascending arteriole. They proposed that the density of the

EXHIBIT 2-4

CAUSES OF ACQUIRED LIVEDO RETICULARIS

Ex 2-4 is not shown because the copyright permission granted to the Borden Institute, TMM, does not allow the Borden Institute to grant permission to other users and/or does not include usage in electronic media. The current user must apply to the publisher named in the figure legend for permission to use this illustration in any type of publication media.

Adapted with permission from Fleischer A, Resnick S. Livedo reticularis. *Dermatol Clin*. 1990;8(2):347–354.

arterial bed is decreased at the margins, but that the superficial venous plexus is more prominent at the periphery.[16] If this theory were valid, any process that impedes blood flow would result in a larger proportion of deoxygenated hemoglobin. There would then be a more livid color at the margins of the cones, and the reticulated pattern would be prominent.[8] This hypothesis does help to explain the clinical findings. However, anatomical studies using serial thick sections have failed to demonstrate the proposed pattern.[16]

Clinical Manifestations

The congenital form of livedo reticularis can be regional or widespread, and is associated with superficial ulcerations. Other anomalies are found in up to 50% of these cases.[16] The acquired form can be seen in many conditions (see Exhibit 2-4). These conditions are exacerbated by exposure to cold, but rewarming does not cause resolution of the livedo pattern. The red-blue net pattern is usually asymptomatic unless ulceration has occurred, and is seen most commonly on the lower legs. The pattern can be seen on other body areas as well, including upper legs, buttocks, lower trunk, and arms.[8] Finding the underlying cause for livedo is important, and cold exposure may make the disease more evident and lead to a definitive diagnosis and treatment.

Treatment

No specific therapy for livedo reticularis exists. Underlying treatable causes must be addressed.

Acrocyanosis

Acrocyanosis is characterized by symmetrical bluish discoloration and coolness of the extremities and is often associated with hyperhidrosis of the palms and soles. The disorder is usually seen in colder climates.

Etiology

The exact cause of acrocyanosis is unknown but there are several theories. Nailfold biomicroscopy reveals decreased blood velocity and dilated capillaries and venules in patients with acrocyanosis. Various investigators have localized initiating events to arterioles, capillaries, and the venous system. It has also been proposed that the initial change is an alteration in blood viscosity that is enhanced by cooling and leads to damage of the capillaries in the papillary dermis.[8]

There have been various reports of acrocyanosis associated with conditions such as mental deficiency,[17] but these have not been independently confirmed.[8] A familial predisposition has been noted.[17]

Clinical Manifestations

Persistent symmetrical cyanosis and coolness of the hands and feet are the most common findings of acrocyanosis, with occasional involvement of the chin, lips, and nose. Hyperhidrosis and edema of the palms and soles are also commonly noted. Cyanosis increases as the temperature decreases and changes to erythema with elevation of the dependent part. Peripheral pulses are usually intact, and the symptoms may persist for years after typically beginning in the second or third decade. Vascular disease and ulceration are absent, which distinguishes acrocyanosis from other diseases such as Raynaud's (Figure 2-10).[8]

Treatment

The mainstay of therapy for acrocyanosis is protection from the cold. Other forms of treatment have included α-adrenergic blocking agents, which may provide temporary relief.[8]

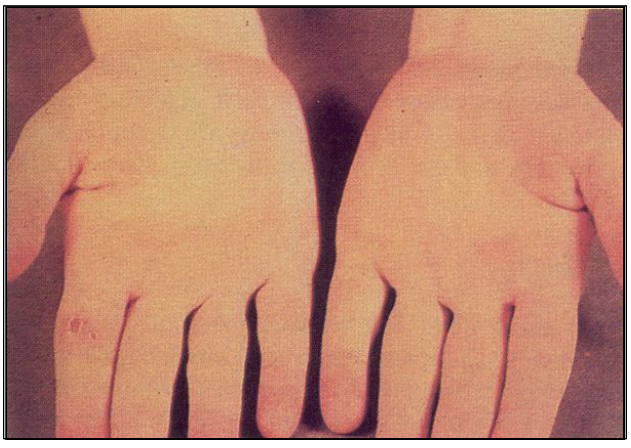

Fig. 2-10. Acrocyanosis. Note that the cyanotic appearance stops at the palmar crease, which is common in this disorder.

Erythrocyanosis

Erythrocyanosis consists of cyanotic discoloration occurring over areas of thick, subcutaneous fat such as the thighs, and is seen most commonly in winter. The lesions may be nodular and similar to pernio. Discussions of this disorder are usually found in older British literature. Erythrocyanosis is rarely encountered now.[9,17]

Etiology

Erythrocyanosis may be a form of cold panniculitis; however, the exact etiology is unknown.[9,17]

Clinical Manifestations

Erythrocyanosis is usually seen in women on the legs and thighs and is often found in horseback riders. The skin is at times tender.

Treatment

Warm clothing and reduction of the insulating fat layer are the only effective treatments for erythrocyanosis.

Trench Foot

Trench foot, or immersion foot, is seen after prolonged exposure to cold and dampness at temperatures above freezing (0°C–10°C).[8] This subject is discussed in detail in Chapter 4, Immersion Foot Syndromes.

Cold Panniculitis

Cold panniculitis consists of tender subcutaneous nodules occurring with cold exposure on uncovered areas. Cheeks and legs are the most common sites, and children are more often affected than adults.

Etiology

Because the nodules of cold panniculitis are usually seen in children, it has been postulated that the more highly saturated fat seen in newborns solidifies at a higher temperature than the adult's less saturated fat.[17] Excessive fibrinolytic activity and cryofibrinogens have been detected in the serum of affected individuals.[8] It has also been thought to be a hypersensitivity reaction.[8,17]

Clinical Manifestations

Tender nodules develop several hours after cold exposure of unprotected areas. The legs and cheeks are common sites, as previously mentioned. The nodules may enlarge and become plaques, which then spontaneously resolve over 2 to 3 weeks.

Treatment

No specific therapy exists for cold panniculitis. Proper clothing and avoidance of cold will prevent most cases.

Raynaud's Disease and Phenomenon

Raynaud's disease is a paroxysmal constriction of small arteries and arterioles of the extremities, usually precipitated by cold. However, it can be induced by other stimuli including emotions. The etiology of Raynaud's disease is unknown. By definition, Raynaud's phenomenon (syndrome) occurs secondary to an underlying systemic disease or occupational trauma (Exhibit 2-5).[8,17,18]

Raynaud's disease was first described by Raynaud in 1862. Hutchinson noted in 1901 that the clinical manifestations of Raynaud could be seen in association with several different conditions. He

EXHIBIT 2-5

CAUSES OF RAYNAUD'S PHENOMENON

- Cold injury sequelae (frostbite and trench foot)
- Arterial occlusive disease (arteriosclerosis)
- Collagen vascular diseases (systemic lupus, dermatomyositis)
- Occupational trauma (jackhammer, typist, mechanic, etc.)
- Cold agglutinins (atypical pneumonia, lymphoma, etc.)
- Cryoglobulins (neoplastic, collagen vascular, chronic disease)
- Neurologic disorders (central and peripheral)

suggested that when an underlying disease process could be identified, the condition should be called Raynaud's phenomenon. When the condition is idiopathic, it is referred to as Raynaud's disease.[8]

Raynaud's is seen in females 5-fold more often than in males, with an average age of onset of 31 years and a range of 4 to 68 years of age. In males, 73% of patients were less than 40 years of age. After long-term follow-up in two studies, 60% to 70% of the patients with Raynaud's could be described as having primary Raynaud's disease.[8]

Etiology

The pathophysiology of the vasospastic attacks in primary Raynaud's disease is unknown. In secondary Raynaud's phenomenon, the vasospasm can be caused by sympathetic stimuli, external physical pressure (seen in farmers, typists, pianists, and individuals who use vibrating tools, eg, hard-rock miners), and decreased blood flow secondary to increased blood viscosity or arterial disease. A study on Raynaud's disease and phenomenon showed that vasospasm could be induced in fingers even with a lidocaine digital nerve block in place, arguing against the widely believed etiologic role of sympathetic hyperactivity.[19]

Histological evaluation of vessels in Raynaud's patients has revealed intimal thickening, thrombus formation, and recanalization in the digital arteries of patients with severe disease, especially those with secondary disease.

Clinical Manifestations

With Raynaud's disease and phenomenon, the skin classically undergoes a triple response with initial blanching or pallor proceeding to cyanosis indicative of vasospasm. The third (recovery) stage is erythema, probably secondary to an end-stage hyperemia.[8] Typically the problem is noted distally in a bilateral digital distribution, and eventually the entire digit becomes involved. Involvement of the hands, feet, and thumbs is less common. Unilateral involvement has been reported. The lips, tip of the nose, ears, and, less commonly, even the tongue can be involved.[8,17] Numbness is common, and pain may be mild or absent; paresthesia can occur during the recovery phase. Nails may show longitudinal ridging, increased brittleness, onycholysis, koilonychia, and thinning of the plate.[8,17] Ulcerations and stellate scars occur on the tips of fingers and toes. In severe cases gangrene develops, and amputation of the gangrenous tips of digits has been necessary on rare occasions (0.4% of cases).[8]

Treatment

The goal of treatment for Raynaud's is vasodilation of the involved vessels with minimal side effects. Prevention of episodes is of primary importance. Protective warm clothing, especially for the hands and feet, is important to avoid localized and generalized cooling. Sudden cooling of the skin, such as occurs when reaching into refrigerators and freezers, entering air-conditioned buildings, and handling cold objects, is to be avoided. Patients with underlying disease that causes the vessels to have limited capacity to dilate are the most difficult to manage.

Vasodilating agents typically have too many side effects to be of extensive use in treating Raynaud's. These sympatholytic agents, which include methyldopa, phenoxybenzamine, tolazoline, guanethidine, and oral or intraarterial reserpine, have generally been abandoned. However, prazosin hydrochloride has been shown to be effective and has acceptable side-effect levels. In one 8-week study,[20] the drug was effective when given in doses of 1 to 2 mg orally three times daily. Doses up to a maximum of 20 mg per day are used to treat hypertension.[20] Postural hypotension can occur suddenly, and the initial doses should be given cautiously and while the soldier is supine.[8]

Biofeedback has been shown to increase digital blood flow but time and expense can be prohibitive. Surgical sympathectomy, once popular, has been shown to be less useful, and now conservative therapy is usually advocated. Sympathetic stellate ganglion block can be useful in acute cases of severe digital infarction and can be given daily by an experienced anesthesiologist.[8]

Direct vasodilating agents, such as the calcium channel–blocking agent nifedipine, have been useful in treating Raynaud's. In a 7-week study,[21] effective doses were begun at 10 mg three times daily and, if well tolerated, were increased to 20 mg three times daily. Again, Raynaud's patients with fixed vascular disease benefit the least.[8] Topical nitrates appear to improve digital blood flow and temperature and can be useful in conjunction with

other therapies. Two percent glyceryl trinitrate in lanolin applied for 3 minutes and allowed to remain on for 20 minutes before removal may provide temporary improvement in digital blood flow and skin temperature.[22]

Patients must *not* smoke under any circumstance.

SUMMARY

History has demonstrated that cold injuries may cause a loss of combat troops that can exceed battle casualties. Proper training of our combat forces, combined with proper supplies and planning, can minimize losses to cold injury. Good planning requires that a number of questions be posed: What weather conditions and intensity of combat will be encountered? Are sufficient supplies such as food and clothing available? Have the officers and enlisted soldiers been trained in prevention? Do commanders have sufficient troops for frequent rotations to the line?

Cold injuries most often involve front-line infantry, a commander's most valuable commodity. These injuries often require months of rehabilitation or cause permanent disability. Because large numbers of cold injuries usually occur only during times of war, exact treatment protocols have not been developed, and further research needs to be done. Early recognition of signs and symptoms and immediate institution of therapy are critical. Training of medical officers in recognition and treatment of diseases caused or exacerbated by cold must be a priority. Line officers also need our help as their medical advisors in establishing adequate preventive measures and enabling the swift return of troops to duty.

REFERENCES

1. Whayne TF, DeBakey ME. *Cold Injury, Ground Type, in World War II*. Washington, DC: Medical Department, US Army, Office of The Surgeon General; 1958.

2. US Departments of the Army, Navy, and Air Force. *Cold Injury*. Washington, DC: DOD; TB MED 81, NAVMED P-5052-29, AFP 161-11. 30 September 1976: 1–15.

3. Hanson H, Goldman R. Cold injury in man: A review of its etiology and discussion of its prediction. *Milit Med*. October 1969;134(11):1307–1316.

4. Orr K, Fainer D. Cold injuries in Korea during winter of 1950–1951. *Medicine*. 1952;39:177–220.

5. Reuler J. Hypothermia: Pathophysiology, clinical settings, and management. *Ann Intern Med*. 1978;89:519–527.

6. Vaughn B. Local cold injury—menace to military operations: A review. *Milit Med*. 1980;145(5):305–310.

7. Sumner D, Criblez T, Doolittle W. Host factors in human frostbite. *Milit Med*. 1974;139:454–461.

8. Demis DJ. *Clinical Dermatology*. 17th ed. Philadelphia: JB Lippincott, 1990.

9. Page E, Shear N. Temperature-dependent skin disorders. *J Am Acad Dermatol*. 1988;18(5):1003–1019.

10. Marzella L, Jesudass R, Manson P, Myers R, Bulkley G. Morphologic characterization of acute injury to vascular endothelium of skin after frostbite. *Plast Reconstr Surg*. 1989;83(1):67–75.

11. Leecost T. Frostbite, diagnosis and treatment. *Journal of the American Podiatry Association*. November 1981;71(11):599–603.

12. Taylor M, Kulungowski M. Frostbite injuries during winter maneuvers: A long-term disability. *Milit Med. 1989;*154(8):411–412.

13. Glick R, Parnami N. Frostbite arthritis. *J Rheumatol*. 1979;6(4):456–460.

14. Bangs C, Boswick J, Hamlet M, Sumner D, Weatherley-White RCA. Winter problems: When your patient suffers frostbite. *Patient Care*. 1 Feb 1977:132–157.

15. Cohen P. Cutis marmorata telangiectatica congenita: Clinicopathologic characteristics and differential diagnosis. *Cutis*. 1988;42:518–522.

16. Fleischer A, Resnick S. Livedo reticularis. *Dermatol Clin*. 1990;8(2):347–354.

17. Corbett D. Cold injuries. *J Assoc Military Dermatologists*. Fall 1982;8:34–40.

18. Coffman J. The enigma of primary Raynaud's disease. *Circulation.* 1989;80(4):1089–1090.

19. Freedman R, Mayes M, Sabharwal S. Induction of vasospastic attacks despite digital nerve block in Raynaud's disease and phenomenon. *Circulation.* 1989; 80(4): 859–862.

20. Surwit R, Gilgor R, Allen L, Duvic M. Double-blind study of prazosin in the treatment of Raynaud's phenomenon in scleroderma. *Arch Dermatol*. March 1984;120:329–331.

21. Rodeheffer R, Rommer J, Wigley F, Smith C. Controlled double-blind trial of nifedipine in the treatment of Raynaud's phenomenon. *N Engl J Med*. 1983;308(15):880–883.

22. Kleckner M, Allen E, Wakim K. The effect of local application of glyceryl trinitrate (nitroglycerine) on Raynaud's disease and Raynaud's phenomenon, studies on blood flow and clinical manifestations. *Circulation*. May 1951;3:681–689.