SPECIAL FEATURE

**TREATMENT OF FROSTBITE TODAY**

One of the surprising characteristics of frostbite is the large amount of tissue that can be saved—even when the hand or foot seems hopelessly frozen. The best chance of preventing crippling tissue loss comes when you are lucky enough to see the patient while the tissue is still frozen and can be rewarmed rapidly. Unfortunately, he is more apt to be seen first by someone not familiar with rapid re-warming, since it only recently became the accepted treatment for frostbite.

Much of what we now know about frostbite was learned during wartime. During military campaigns, frostbite and the clinically similar immersion foot (which results when the foot is immersed in water for long periods) have reached epidemic proportions. Although immersion foot is uncommon among civilians, frostbite is still a problem in all but the southernmost parts of the United States. In five years, 54 frostbite victims have been hospitalized at Colorado General Hospital and Denver V.A. Hospital.

**The Likely Victim**

Neurasthenic, poorly adjusted, excessively sweating, or self-destructive individuals have been known since antiquity to have an increased susceptibility to frostbite. Many alcoholics are victims, having fallen asleep in a snowbank, a car, or an abandoned garage. Also, there have been patients who suffered repeated episodes of frostbite that seemed to be suicidal gestures.

Other likely victims include winter sports enthusiasts and accident survivors trapped in their wrecked automobiles. Several studies suggest that


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Negroes may have a decreased adaptability to cold.

The severity of tissue damage depends upon the degree and the duration of cold. Thus, wind, wetness, contact with metal, or anything that speeds heat loss increases the danger to exposed parts of the body.

**What Happens in Frostbite?**

In studies in rabbits, frostbite occurred when thermocouples deep in the tissue showed temperatures below 22°F., an observation generally confirmed in clinical studies during the Korean War. The nature of frostbite has been highly controversial. Until recently, most authorities believed that it was a direct thermal injury to the tissue. Others believed the indirect injury resulted from changes in the microcirculation. Now it appears that both mechanisms are important. Cold can destroy tissue directly by forming intracellular ice crystals or more commonly by interstitial freezing with consequent extraction of water from the cells. But the blood supply to the frozen area also undergoes profound changes. Microangiographic studies in animals have revealed that frostbite causes tortuous dilatation of the small vessels, as well as sharply delineated occlusions and intravascular tubular filling defects. Vascular stasis follows. If the injured area does not undergo necrosis, it is left with a distorted vascular system reduced in cross section.

The frostbitten limb undergoes acute changes within hours. First the distal areas become pale, then cyanotic. Just proximal may be a zone of intense hyperemia. In time, the most severely affected areas blister and turn black. The depth of the gangrene is difficult to assess until weeks later.

Even without tissue loss, frostbite may cause lasting symptoms: hypersensitivity to cold, causalgia, hyperhidrosis, coldness, and stiff joints. The explanation may be found in studies showing that blood vessels in frozen tissues often lose their ability to constrict or dilate normally. In children, epiphyseal damage may cause abnormal development of joints or bones.

**The New Way to Treat Frostbite**

In most cases, the hands or feet will already have been thawed when you first see them. This is unfortunate because the supreme opportunity to favorably influence the outcome of frostbite is when the extremity is still frozen.

Until a few years ago it was thought that the frozen part should be rewarmed slowly by allowing it to warm at room temperature or even by rubbing it with snow, beating it, or immersing it in cold water. Such recommendations were passed from generation to generation and often taught by physicians to first-aid classes. Then in the mid-1950’s the brilliant studies of R. B. Lewis conclusively demonstrated the fallacy of this approach. He recommended rapid rewarming instead, and clinical studies have amply confirmed his finding that much less tissue is destroyed by rapid rewarming than by slow.

The ideal warming device is a deep water bath with automatic temperature control. Because this is almost never available, use a makeshift tub and control the temperature by adding appropriate quantities of warm water as the water cools. Keep the temperature between 103° and 107.5° F. (40° to 42° C.); higher temperatures risk further injury to the tissue, lower
temperatures will not produce maximum benefit. During warming, the line between cyanotic, partially devitalized tissue and the proximal healthy tissue will move distally—first quite rapidly, then more slowly. Sedation may be cautiously prescribed if required during painful rewarming. If the patient is also suffering from exposure, as in the following case, he may require rewarming of the whole body.

A 25-year-old mountain climber was lost in a snowstorm without food for 40 hours. When rescued, he was conscious, but incoherent. His rectal temperature was 25°C; respiration, 18; heart rate, 50; and blood pressure and peripheral pulse were unobtainable. At the hospital, the BUN was measured at 56 mg%; the arterial pH, at 7.1.

When rewarming began (Figure 1), the patient’s ankles were a mottled purple; his feet were dead white with no evidence of capillary filling. In water at 37° to 42°C, the color returned gradually to his feet, moving toward the toes rapidly at first, then more and more slowly. Simultaneously, he was wrapped in blankets and treated for shock and acidosis. It was apparent when the patient’s feet were removed from the water that little if any tissue would be lost. A few hours later, oliguria set in and persisted for 72 hours, requiring dialysis. Two days later the BUN had dropped from 180 to 18 mg%.

**Healing Takes Months**

The frostbitten extremity may be dressed with fluffed gauze or left exposed. In either case, watch closely for infection. After a few days of immobilization, have the patient begin daily exercises. You can conveniently combine this with cleansing by having him flex his fingers and toes during whirlpool treatments using warm water and mild soap. In F. A. Simone’s analysis of cold injuries in wartime, exercise was one of the few measures of undoubted value.

One of the characteristics of frostbite is the large amount of tissue that can be salvaged in an apparently badly injured member. Often the black, shriveled tissue will eventually slough away like a cast, leaving far more viable tissue than had been anticipated (Figure 2). The amount of tissue loss may not be apparent for weeks or even months. That is why amputation should be delayed as long as possible—usually months. Debridement should be conservative.
The care of the patient long after frostbite is essentially supportive. The patient should avoid exposure to cold at all costs. Sympathectomy may be of value in patients with evidence of sympathetic hyperactivity.

The Controversial Role of Early Sympathectomy

For almost 25 years there have been isolated reports of the benefits of sympathectomy during the acute phase of frostbite. Such reports were difficult to evaluate; hence sympathectomy has not been applied widely. Recently Wesolowski and his associates have reported studies of patients who had symmetrical freezing of the hands or feet. Sympathectomy was performed on only one side; the other side was used as a control. On the side of the sympathectomy, the extremity had less pain, more rapid resolution of edema, less tissue loss, earlier demarcation, and faster healing.

These investigations as well as recent animal investigations suggest that the best time for sympathectomy is between 24 and 72 hours after thawing. This is not a recommendation for indiscriminate sympathectomy; it is not needed unless massive tissue damage is evident from the outset. Even then, the risk of the sympathectomy must be carefully weighed against the possible benefit. Less controversial is the role of sympathectomy for palliation of the symptoms that persist for years after cold injury—symptoms such as hyperhidrosis, coldness and pain.

Experimental Drugs

Intra-arterial vasodilators such as Priscoline, anticoagulants, fibrinolysins, and low molecular weight plasma expanders may have value, but it has not yet been established. Recent studies with low molecular weight dextran, however, suggest that it may prove valuable.