MANAGEMENT OF FROSTBITE INJURIES

Gustavo A. Espinosa, MD
Chicago, Illinois

The role of interventional radiology in the management and diagnosis of frostbite injury in 18 patients at the West Side VA Medical Center is presented. Conservative treatment and the use of intra-arterial long acting vasodilators, such as reserpine, are emphasized, even in patients referred 48 to 72 hours after the initial injury. This modality of treatment should be attempted before surgical decision or amputation is necessary. Satisfactory responses were obtained in most of the treated subjects and in some cases, results were dramatic with significant recovery and a minimum of sequelae. Generally clinical principles and physiopathology in frostbite injuries are reviewed.

Several forms of cold injury have been described, most of which are uncommon in civilian practice. Frostbite continues to claim victims in areas with colder climates, due to the severity of recent winters and increased popularity of winter sports. A clinical case usually is a result of slow freeze-thaw injury in an individual subjected to prolonged accidental exposure while comatose or intoxicated. Vascular damage is the prominent result of such an injury. Angiography has a unique role both in diagnosis (Figure 1) and, recently, in treatment of this injury. Results are sometimes dramatic.

Four catagories of cold injury have been described: A Grade I injury comprises edema and redness without tissue necrosis in the affected area. Blister formation represents a Grade II injury (Figure 2). In the Grade III injury, tissue necrosis is present (Figures 3 and 4). Development of gangrene, requiring amputation, constitutes a Grade IV injury (Figure 5).

From the West Side VA Medical Center and the University of Illinois, Chicago, Illinois. Requests for reprints should be addressed to Dr. Gustavo A. Espinosa, Department of Radiology (114), West Side Hospital, PO Box 8195, Chicago, IL 60680.

MATERIALS AND METHODS

Eighteen patients with different degrees of frostbite were treated in the winters of 1977, 1978, and 1979, at the West Side VA Medical Center. Of these, eight were seen in the first four days after the initial injury and the remainder approximately ten days following exposure (Tables 1 and 2).

Degree of injury varied and response was favorable in most patients (14). The most frequently employed treatment consisted of intra-arterial injection of a long acting vasodilator (reserpine, 0.5 mg) introduced via a polyethelene catheter in the ipsilateral axillary artery (French sizes 5 and 6). Severity of frostbite was graded from I to IV. The best response was obtained with injuries Grades I, II, and III (Figures 1-3). All injuries of Grade IV severity required amputation (four patients). The primary purpose of this report is to emphasize the use of long acting vasodilators and the conservative management before amputation is necessary.

GENERAL PRINCIPLES

It has been shown experimentally that freezing of mammalian tissues begins at approximately 10 C.[1] The extent of injury is determined by duration of exposure, humidity, and wind speed. The speed with which freezing occurs has particular importance. High-altitude "burns" are those produced experimentally by *rapid* freezing with deposition of ice crystals in tissue.[1] Little ultrastructural tissue damage occurs and full return of function often is seen in this unusual type of injury.[2,3] *Slow* freezing is likely to induce severe cellular damage. The physiology of a slow-freeze injury has been simulated by numerous clinical studies on animals and volunteers.[4] The initial response is constriction in the exposed part.

With continued exposure, the intense vasoconstriction causes decreased blood flow resulting in stasis, clumping of red cells, and ultimately

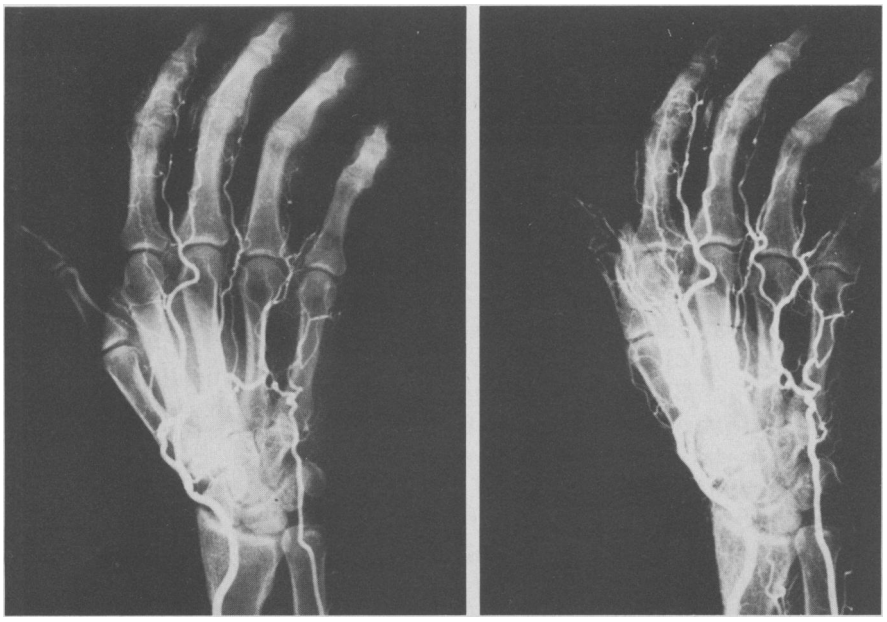

Figure 1. Grade I Injury. The arteriogram reveals the severe vasospasm and lack of distal perfusion. Injection of a rapid acting vasodilator (tolazoline, 50 mg) did not prove helpful. Dilatation, however, was obtained in the normal vessels (right side)

widespread vascular occlusion.[4] Upon rewarming, there is an intense reddening, increase in temperature, and edema of the area accompanied by severe pain. Blisters develop rapidly and contain a sterile fluid nearly identical to plasma (Figure 2). Edema becomes maximal at 24-48 hours when gangrenous areas may begin to demarcate. The extent of gangrene is often difficult to estimate and may require observation for up to 30 days. In many cases, the skin becomes gangrenous while the underlying tissues remain salvageable (Figure 4).

PHYSIOPATHOLOGY

Despite many studies, the fundamental injury remains uncertain. Vascular occlusion is an outstanding feature of slow freeze injuries[4] but recent ultrastructural studies[2] indicate that the disruption of mitochrondrial and endothelial cell membranes may be the initial injury. A swelling of endothelial cells with partial occlusion of the vascular lumen also is a prominent finding.[2,3] Subsequent increased capillary permeability allows plasma transudation, edema formation, and stasis, culminating in vascular occlusion. Histologic examination of damaged tissues shows fibrin deposition and thrombosis in capillaries and lymphatics. In addition, edema and inflammatory infiltrates are seen in areas with focal necrosis.

THERAPY

Treatment of frostbite begins with rapid rewarming of injured tissues.[5] Frozen tissues should be immersed in a water bath or whirlpool at 40-44 C. Higher temperatures are detrimental. Surgical soap may be added at one ounce per ten gallons of water to aid in debridement. When rewarming is completed (after approximately 20 minutes), the limb should be protected by sterile dressings and elevated to minimize edema. Blisters should be preserved intact to prevent infection. Prophylactic antibiotics are indicated.[6] While vasodilatation and edema occur in the most severely affected areas, vasoconstriction persists in the proximal, less injured portion of the limb or limbs.[7,8] Several systemic modes of pharmacotherapy have been advocated to alleviate these imbalances. These include intravenous procaine, tolazoline, nicotinic acid, and sympathetic blockers.[9] Many feel that regional sympathectomy[10] is effective in minimizing tissue loss by decreasing vasospasm. It has been reported also to decrease the sequelae of cold injury, ie, paresthesias and hyperhidrosis. Although of theoretical value in combating sludging

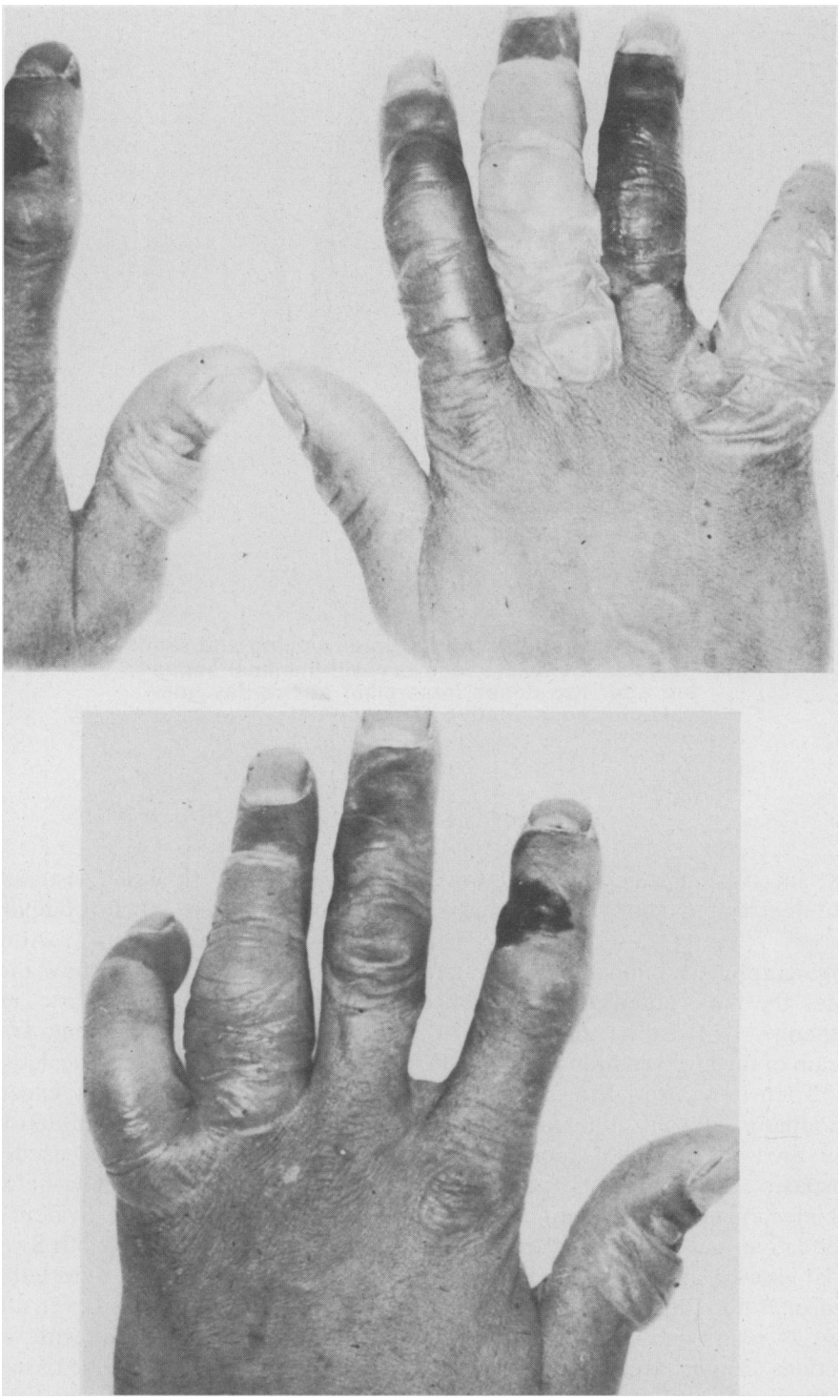

Figure 2. Grade II Injury. Blister formation occurred after the initial whirlpool bath. The patient recovered completely after conservative management and reserpine (0.5 mg) intra-arterially in three weekly doses

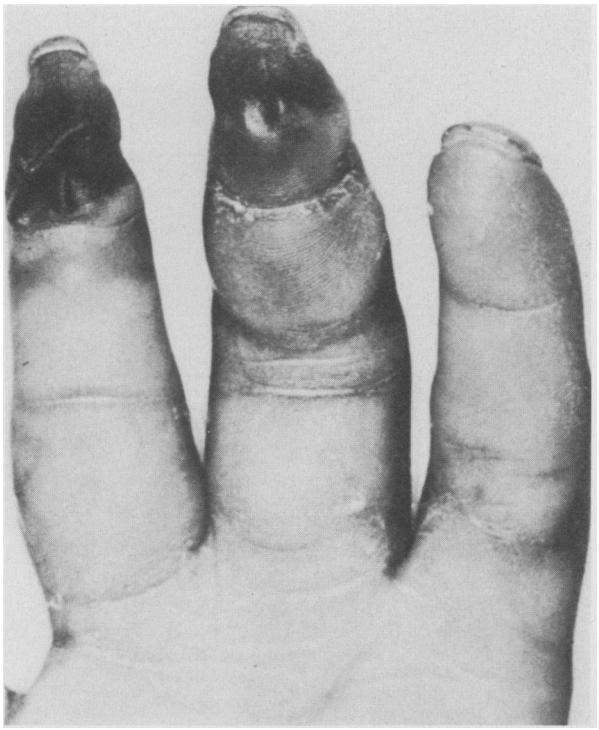

Figure 3. Grade III Injury. Note swelling and skin necrosis. Complete recovery with minimal sequelae after two doses (reserpine) at five-day intervals plus conservative management

and thrombosis, intravascular heparin and low molecular weight dextran have not yielded consistent results.[11]

The use of angiography in conjunction with vasodilators provides the most potent tool for both diagnosis and therapy of frostbite injuries.[9,12] A baseline angiogram of the affected limb is obtained first (Figure 1). Then intra-arterial injection of a rapid acting vasodilator (ie, tolazoline) is given to differentiate the area of organic stenosis from functional vasospasm. For therapeutic purposes, the intra-arterial injection of 0.5 mg reserpine to the limb provides effective and long lasting relief of functional arterial vasospasm.[13] Reserpine acts by depletion of neuronal norepinephrine, with onset of action in 3 to 24 hours and duration of 14-21 days per injection. Limbs treated using this protocol become red and warm. Continued blanching and coolness in frostbite injuries is a well-known indicator of impending gangrene. Repeat angiograms 48 hours after injection of reserpine show increased flow and visualization of smaller terminal arteries when compared with initial studies of untreated controls. Animal experiments and human case studies show that a definite and sometimes dramatic decrease in tissue loss is achieved with this treatment. Gralino et al[14] reported that their subjects healed without residual hyperesthesias or disability excepting the loss of function resulting from amputation in one patient. A frequent cause of increased morbidity from frostbite is variable delay before treatment is sought. Gangrene may be evident when the patient first presents. However, both Swedish and American investigators have reported that vasodilator therapy may be beneficial even after 72 hours post injury.[9,13-15] Angiography with vasodilators may limit or dramatically reverse tissue loss by increasing blood flow to viable tissue beneath gangrenous skin (Figure 4). If tissue loss (gangrene) cannot be prevented, up to a month may be required to demarcate its extent. The response of effectiveness

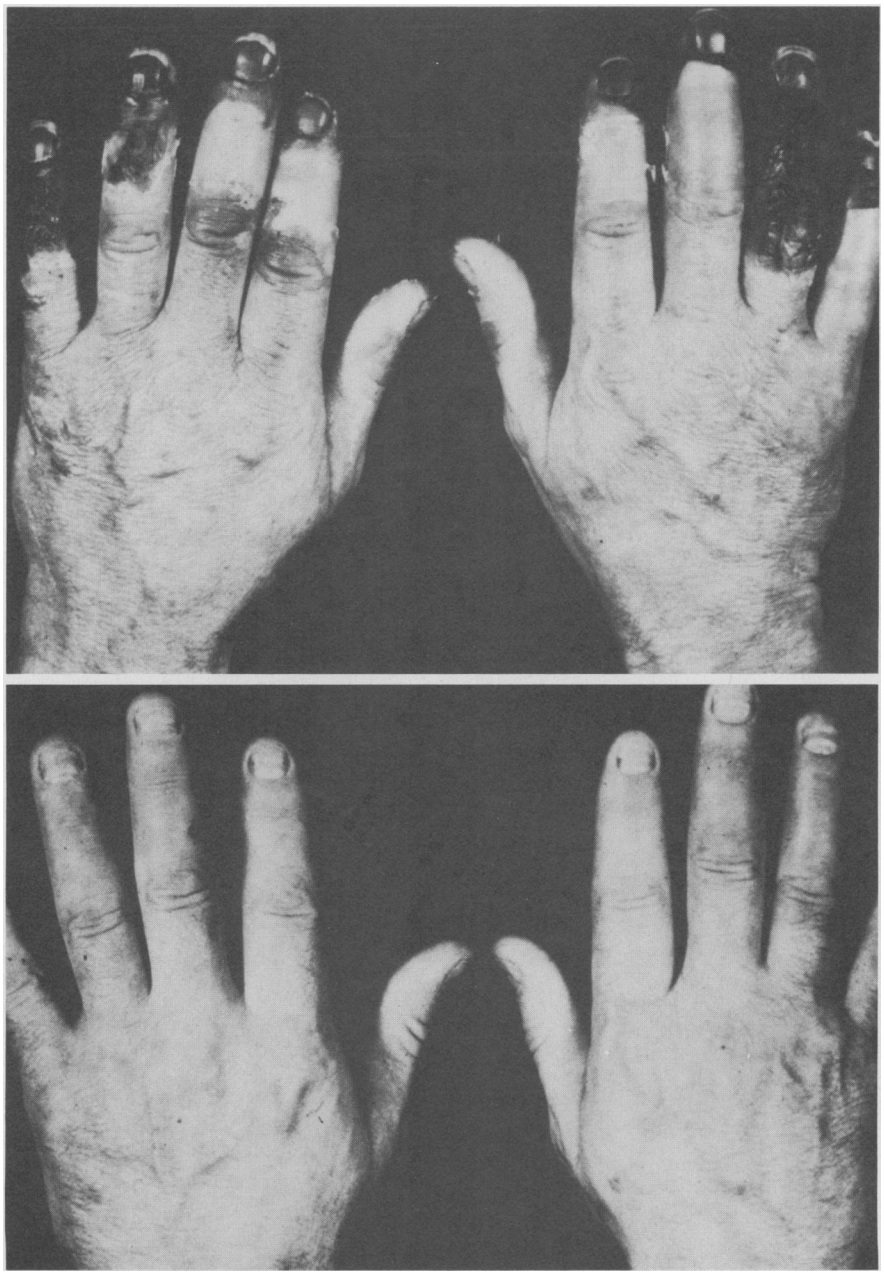

Figure 4. Grade III-IV Injury. Soft tissue edema, skin necrosis, and "black" nails. Amputation was considered, but significant recovery took place after five weeks with good restoration of function (bottom). Three doses of intra-arterial reserpine were required at weekly intervals

to vasodilator injection appears to provide an early and accurate prediction of the limit of viable tissue. Vessels that remain unresponsive to vasodilators after two injections three days apart are likely to cause gangrenous changes. Thus, the level of amputation may be determined more precisely without excessive tissue removal and delay prior to surgery may be somewhat shortened.

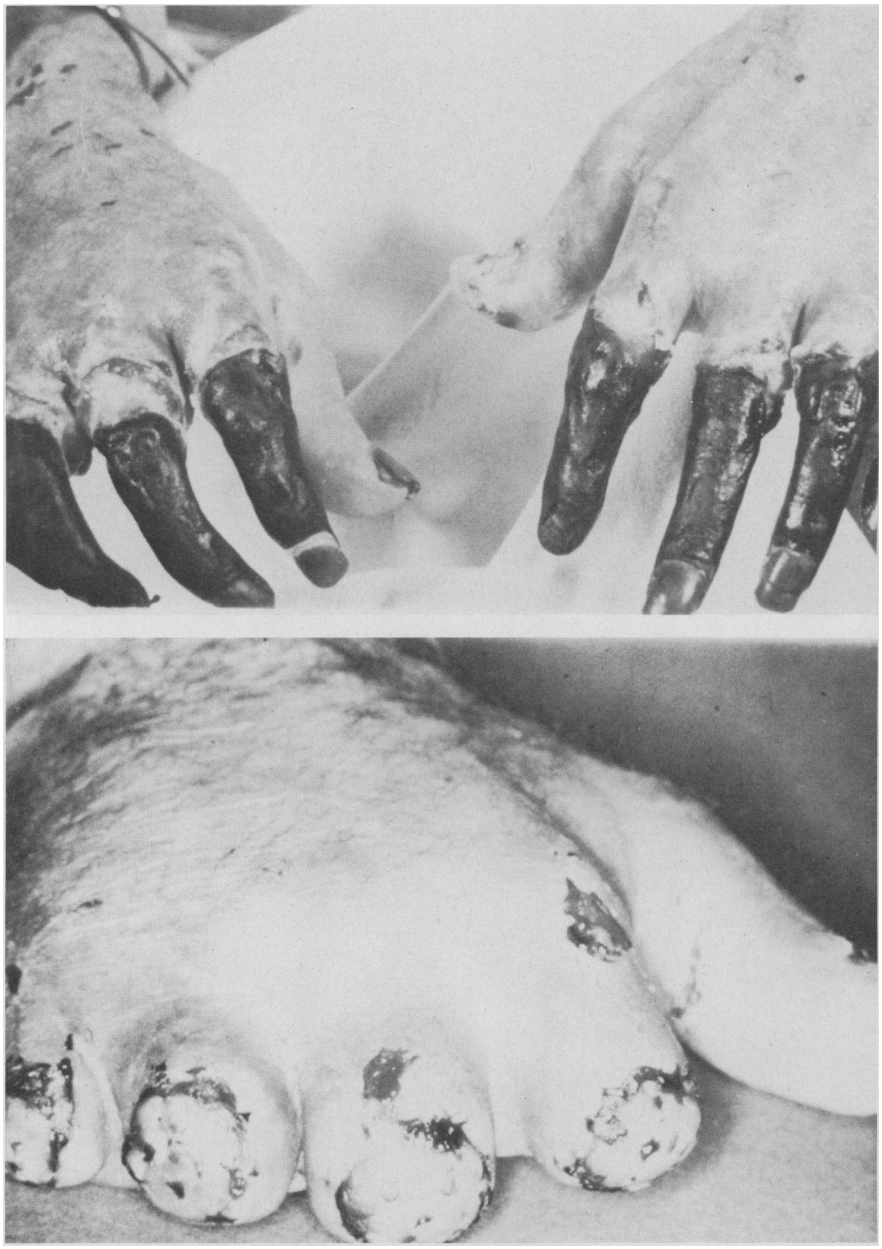

Figure 5. Grade IV Injury. Patient admitted with severe skin necrosis, as shown (top). No response was obtained after the usual management. Patient required amputation (bottom), five weeks later

CONCLUSION

Angiography has entered a unique triple role in the management of slow freezing injuries. It is the method of finding the extent of injury. Combined with intra-arterial vasodilator injection, it apparently minimizes both loss of tissue and sequelae of frostbite injury. In those injuries where surgical amputation becomes necessary, angiography allows faster and more precise demarcation of viable from non-viable tissue, thus aiding the surgeon in his pre-operative assessment.

TABLE 1. RESPONSE TO INTRA-ARTERIAL RESERPINE (N=18)

Injury Grade	Number of Patients
I	5*
II	5*
III	4
IV	4

*No response after initial treatment with tolazoline, 50 mg (intra-arterial) as seen angiographically

TABLE 2. TIME INTERVAL BETWEEN INJURY AND INITIATION OF TREATMENT

Time Period	Number of Patients	Injury Grade
1st 24 hours	6	I
2-4 days	2	II
Over 10 days	10	III, IV

Literature Cited

1. Asahina E, Shimada K, Hisada YA: A stable state of frozen protoplasm with invisible intracellular ice crystals obtained by rapid cooling. Exp Cell Res 59:349-358, 1970

2. Bowers W Jr, Hubbard R, Daum R, et al: Ultrastructural studies of muscle cells and vascular endothelium immediately after freeze-thaw injury. Cryobiology 10:9-21, 1973

3. Price H, Howes E, Blumberg J: Ultrastructural alteration in skeletal muscle fibers injured by cold: The acute degenerative changes. Lab Invest 13:1364-1277, 1964

4. Bellman S, Adams RJ: Vascular reactions after experimental cold injury: A microangiographic study on rabbit ears. Angiology 7:339-367, 1956

5. Arledge R: Treatment of patients with frostbite. Phys Ther 53:267-268, 1973

6. Fontaine R, Klein M, Bollack C, et al: Clinical and experimental contribution to the study of frostbite. J Cardiovasc Surg 2:449-455, 1961

7. Erikson U, Ponten B: The possible value of arteriography supplemented by a vasodilating agent in the early assessment of tissue viability in frostbite. Injury 6:150-153, 1974

8. Erikson U: Circulation in traumatic amputation stumps. Acta Radiol [Suppl] (Stockh) 238, 1965

9. Snider R, Porter J: Treatment of experimental frostbite with intra-arterial sympathetic blocking drugs. Surgery 77:557-561, 1975

10. Golding MR, Martinez A: The role of sympathectomy in frostbite with new review of 68 cases. Surgery 57:744-749, 1965

11. Mindith E, Long D, Brown RB: Treatment of frostbite with low modular weight dextran. J Trauma 4:246, 1964

12. Smith S, Walker W: Arteriography in cold injury. Br J Radiology 37:471-474, 1964

13. Porter J, Lindell T, Leung B, et al: Effect of intra-arterial injection of reserpine on vascular wall catecholamine contact. Surg Forum 23:183-185, 1972

14. Gralino B, Porter J, Rosch J: Angiography in the diagnosis and therapy of frostbite. Radiology 119:301-305, 1976

15. Ervasti E: Frostbite of the extremities and their sequelae: A clinical study. Acta Chir Scand [Suppl] 299, 1962