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TREATMENT OF FROSTBITE(U) ARMY RESEARCH INST OF
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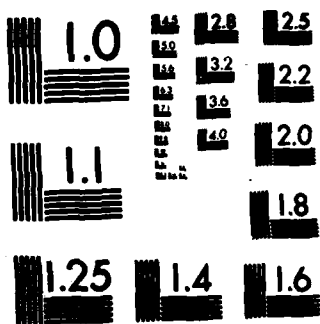
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TREATMENT OF FROSTBITE

by

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1982

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With an increasing interest in outdoor recreation, especially in the winter time, hypothermia and frostbite victims will increase as presentations at our hospitals in northern tier states indicate. The military has historically had an interest in cold injury because of the significant number produced in combat settings in all parts of the world. Almost every Army that has been exposed to cold has had serious cold injuries. Ten percent of our wounded casualties in both World War II (90,000) and Korea (9,000) were cold injuries. p. 4

Pathophysiology

Cold injury usually occurs during sudden changes of weather with a dramatic drop in temperature below freezing. If the sudden change in temperature is accompanied by wind and/or rain or snow, then the risk increases further (8).

Frostbite is the actual freezing of tissue and should be differentiated from abrasion trauma which is common in loose fitting boots and may occur in a cold setting. A similar injury, chilblains, requires having cold, damp extremities for an extended period of time (1). Trenchfoot or immersion foot (3) is a more severe form of a cold/wet injury. This occurs from having wet dependent extremities for an extended (12 hour) period of time. If the temperatures are warm (about 10°C), then immersion foot results; if cold, near freezing, then trenchfoot occurs. These are time/temperature injuries. Longer times at milder temperatures are required to produce lesions similar to mild wetness with severe cold. These are neurovascular injuries in which sympathetic nerves and small blood vessels have been damaged which compromises blood flow. Late complications of cold/wet injuries include ulceration and chronic infections. Although rare in peacetime, this devastating injury occurs in large numbers in combat. Recent experience of the British marines in the Falkland Islands remind

us that even a modern army can be severely compromised by trenchfoot. Frostbite, the actual freezing of the tissue, generally starts distal and works its way proximal, starts superficial and works its way deep. The freeze line is a bullet shaped freeze line through the ends of the fingers. The damage of freezing is a combination of the actual physical destruction of cells and membranes by ice crystal formation and the cellular dehydration associated with this ice crystal formation (14). Water is drawn out of the cells and delicate intracellular structures are damaged by rising ionic concentrations. Add to this the explosive growth of ice crystals around the cells, and one can understand the severe destruction associated with this injury. There is a significant direct metabolic impediment of the cold on different cell types. Vascular damage which accounts for the demarcation generally occurs first to capillaries and endothelial cells. This loss of tubular integrity of the vascular system compromises nutritive tissue flow and a combination of these factors; metabolic impairment, hyperosmolarity, ice crystal formation, and vascular destruction leads to the loss of tissue.

This injury is still a wait-and-see disease. Modern therapeutic inroads to improve tissue salvage have proven to be minimal at best. The body does a very effective job of defining tissue which will survive from that tissue which will not. There is a fine 1 or 2 millimeter line of liquefaction and demarcation which occurs between these areas. Early surgical intervention is to be avoided as viable tissue is often removed and retraction problems are significant.

Clinical Presentations

Patients arriving with cold injury often have a history of concomitant injury, alcohol or drug abuse, or working in the cold for long periods of time (5). Medical personnel will seldom see individuals still frozen and some degree of thaw has usually occurred prior to presentation. Patients will describe a tingling pain or numbness with waxy, yellow skin or more commonly describe an

anesthetic injury. They often are unaware of the progress of the freezing until some rewarming has occurred. Although described as a painless injury on freezing, it can be extremely painful during rewarming usually starting as a tingling or burning pain followed by throbbing, swelling, and increased redness throughout the area. Distal portions of the fingers may be blanched white and stay blanched throughout the first two or three days of treatment indicating poor capillary filling time or nonexistent blood flow. As thawing continues there will be pulsatile pain with a change in color to pale blue or burgandy.

Grading of Cold Injury

→ The literature contains a description of the degrees of frostbite ranging from 1st degree through 4th degree. This is a retrospectively grading system and generally of little significance, especially to the treatment procedure. Even highly experienced physicians find it difficult to grade frostbite in the first 24/48 hour time period. It is much more important to differentiate superficial from the deep injury; however for reference, the differentiation of the degrees will be listed here.

First degree cold injury (14) - usually defined as hyperemia and edema after rewarming. The tissue is cyanotic, red, hot and dry. There is very poor capillary filling time in the nail beds and distal portions of the fingers. Swelling begins early in the thaw process and may become quite severe over the first twelve hours. This swelling may persist for ten days to two weeks and may result in paresthesia, hyperhidrosis and coldness of the portion exposed to cold for months or years after the injury. Most mild first degree injuries resolve with minimal subsequent clinical manifestations.

Second degree cold injury - hyperemia and edema with rather severe burning pain occurs during the early rewarming phase. Swelling begins early followed by blister formation. These blisters grow rapidly, are filled with a clear

serous fluid, and patients may experience severe pain requiring analgesia. Vessicles are superficial to the germinative layer and generally dry forming eschars within 10 to 24 days. There is often a throbbing pulsatile pain or ache late in the treatment. Hyperhidrosis and cold sensitivity are a common sequelae after this degree of injury.

Third degree cold injury - involves necrosis of skin and cutaneous tissue. This is a full skin thickness injury leading to ulceration of the extremities. Blisters that form generally do not extend to the tips of the digits and severe shooting pains are often associated with this injury between the fifth and tenth day. Black eschars formed as blisters dry. Blisters are hemorrhagic rather than serous. If moist gangrene develops, this injury often requires early surgical intervention.

Fourth degree cold injury - complete necrosis and loss of tissue, muscle and bone. Upon rewarming, the tissue is deep red, purple, and cyanotic. The area is usually anesthetic, but tissue proximal to the fourth degree area will have pulsatile pain, burning and blister formation. Fourth degree injury rapidly mummifies with little edema formation. Paresthesia are common after three days and the line of demarcation between viable and mummified tissue occurs between 30 and 36 days. This demarcation line is evident through bone at 60 to 80 days.

Cold injury is a continuum of events and the severity is difficult to differentiate early. It should also be noted that an individual with fourth degree injury will have first, second, and third degree proximal to that injury, but much more superficial. It is more important to be able to define good and poor prognostic signs early in the treatment process than to attempt to state a specific degree. Good indicators include large, clear blisters extending to the tips of the digits or the base of the nails, a rapid return of sensation to the

extremities, return of blood flow to the tips of the digits with warmth, good capillary filling time and pinkness to the tips of the digits (6). Poor prognostic indicators include hard, white, cold and insensitive extremities that are cyanotic without blisters. There is often hemorrhagic blisters, complete absence of edema, and early mummification indicating severe injury. If trauma is superimposed on cold injury with compromised blood flow, than the likelihood of severe injury is high. A freeze/thaw refreezed injury has a very poor prognosis with early demarcation and rapid mummification.

Contact cold injury such as touching cold metal is usually a superficial injury in which the tissue freezes to the surface producing severe pain and rapid removal of the fingers stripping superficial epithelium. This injury is generally not serious on the hand but may be a problem on the lips.

Treatment of frostbite involves rapid rewarming in water with temperatures between 100 and 108°F (7). Higher or lower temperatures result in more tissue loss. Whirlpooling is the best method of thaw, but any pan or pail of water will suffice. Betadine or hexachlorophene should be added to the thaw water and subsequent whirlpool water to control superficial infection. The patient will experience rather severe pain during the rewarming process and aspirin or Demerol analgesia may be necessary. The temperature of the water should be monitored and warm water stirred in to maintain the temperature during the thaw process. Usually a half hour thaw time is all that is necessary. A rapid return of color often deep red or purple, and the softening of the tissue is good indication that thawing is complete. Rapid thaw produces a rapid return of sensation. While the tissue is frozen it must not be rubbed or massaged and the use of any other material, such as snow, cold water, grease, butter, ointments, etc. is contraindicated. Smoking should be prohibited in these individuals because of the vasoconstrictive action of nicotine. The blisters

should not be ruptured, although the patient will ask this to be done. Blisters will resolve in seven to ten days; however, if they are ruptured early, infections are difficult to control. Salves and ointments are contraindicated. Tetanus boosters should be administered if the patient's vaccination record indicates its need. Prophylactic antibiotics should not be used unless. If obvious deep infection occurs they should be given after culture and sensitivities. Twice daily whirlpooling for 20 minutes with betadine is recommended followed by a gentle pat dry. Complete sterile handling with mask, gown, and gloves is required early. Sterile cotton pledgets should be placed between the fingers to prevent abrasion of the blisters. Tents or cradles should be used in the bed to protect the extremities and they should be left open to the air. Alternately, sterile towels can be wrapped loosely around the hand and pinned to the gown. Full range of motion exercise should be encouraged during each whirlpooling session to prevent flexion contractures (5,8). Early surgical intervention is to be avoided as superficial infections, osteomyelites and retraction problems lead to increased tissue loss. More proximal amputations and prolonged hospitalization times result. Amputations when done 3-8 weeks after clear demarcation of soft tissue with mummification require little effort to close. If demarcated at a joint, spontaneous amputation with no closure is common. Unless life threatening systemic infections develop, physicians should allow for physiologic amputation which may take weeks or months. Circumferential eschars that constrict blood flow to viable distal tissue or over joints should, however, be bivalved. This bivalving should not extend into viable tissue. Surgical sympathectomies, although recommended in the past, are contraindicated and interarterial injections of reserpine (0.5 mg/injection) (5,9) seem to have the same early benefits without the late sequelae. Ganglionic blockade to produce a chemical sympathectomy can be considered also. Other therapeutic modalities have had

mixed results or minimal beneficial effects. Use of antihistamines or corticosteroids have been universally unproductive on experimental models. The use of vasodilators to improve blood flow have shown some improved blood flow and tissue salvage. Anticoagulants, if given during the thaw, may help; however, late utilization of heparin, for example, has not been beneficial. Plasma expanders, such as low molecular weight dextran, have shown slight improvement in tissue salvage.

The use of excessive heat during rewarming can cause severe damage. Because the limb is anesthetic, patients will often dip their hands in hot, occasionally boiling water, put them in front of a heater, over a stove, or in an oven. This produces a burn on top of a cold injury which is a devastating injury. In special cases, i.e., patients demonstrating compartmental swelling, fasciotomy, along with vasodilators, such as dibenzaline, have shown improved circulation. This procedure has been used most commonly in feet rather than hands. The use of diathermy for rewarming has not been beneficial and the use of hyperbaric oxygen for treatment has also not shown any improvement in tissue salvage.

Patients with amputation type injuries may, after some weeks in the hospital, be sent home and encouraged to use a whirlpool or sink bath with betadine to manage the injury until demarcation is complete.

Skin grafting over frostbitten extremities should be done later after revascularization has occurred and a good bed for grafting has developed.

A silver nitrate treatment has shown no improvement over other cleansing agents. Bone lesions especially in young individuals appear as punched out lytic lesions in articular and periarticular surfaces some months after the injury. Young patients should be followed radiographically for a year (sequentially) to note the progress or development of these lesions. Digital curvatures and carpal lysis have been reported in frostbite of the hands of young patients.

Even mild degrees of cold injury produce lasting problems of paresthia, cold sensitivity, hyperhidrosis, causalsia and Raynaud's symptoms. More severe injuries, especially in young patients, produce lytic bone lesions, early ephysial closure with curvature of digits and loss of subcutaneous tissue. Treatment of the sequellae is generally supportive and symptomatic but good results with interarterial reserpine have been reported. Technique of reconditioning peripheral constriction associated with cold exposure may be useful in prior cold injury patients. The technique is simple and involves immersing the hands in hot water, walking the patient into a cold room (32°F) for ten minutes, bringing the patient out of the cold room; with the hands immersed in hot water at all times. This procedure is done three times a day, 3 days a week and over a five week period and results in a significant relief of Raynaud's symptomatology (10).

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