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COLD INJURY

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1. Purpose. This bulletin provides information regarding cold injury and is a guide to its prevention and management.

2. Introduction. *a.* Cold injury is defined as tissue trauma produced by exposure to cold. The type of injury produced depends upon the degree of cold to which the body (or its parts) is exposed, the duration of exposure, and certain concurrent environmental factors. For practical purposes, cold injuries may be divided simply into "freezing" and "non-freezing" types. The former is the well-known frostbite (superficial or deep). The non-freezing types are chilblains, trench foot, and immersion foot. There exists no real justification for distinguishing between trench foot and immersion foot with respect either to pathology or management, nor even to the environmental conditions which were causative. Both result from prolonged exposure of the feet to wet cold: trench foot to cold wet socks and boots, immersion foot to cold water—with or without socks and boots. For present purposes the term "trench foot" will be employed to cover both "immersion foot" and the more cumbersome "non-freezing cold injury." Chilblains is the only cold injury which is not of significant military importance.

b. Non-freezing cold injuries may be defined as follows:

(1) Chilblains result from intermittent exposure to temperatures above freezing accompanied by high humidity.

(2) Trench foot (and immersion foot) result from prolonged exposure to wet cold foot gear or outright immersion of the feet at temperatures usually below 50°F. At the upper range of temperatures, exposure of 12 hours or more will cause injury. Shorter durations at or near 32°F will cause the same injury. It is usually associated with immobilization and dependency of the feet.

c. Frostbite results from crystallization of tissue water in the skin and adjacent tissues and is produced by exposure to temperatures below the freezing point. The depth and severity of the injury is a function of the temperature and the duration of exposure—the lower the temperature, the shorter the time required to produce injury. At low temperatures, in the presence of wind, freezing of exposed skin can occur within a few seconds.

d. Cold injury occurs sporadically among the civilian population, but its primary concern is to the military forces. Cold injury has been recorded as a

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problem of military importance since the days of Xenophon, and Alexander of Macedonia. Larrey classically described the role which cold injury played in the defeat of Napoleon's Army in Poland in 1812. The United States had considerable experience with cold injury during World War II. In the US Army, there was a total incidence of 90,535 time-lost injuries which included trench foot and frostbite in ground troops and high altitude frostbite in air crews. More recently, in Korea, United States troops experienced more than 9,000 cases of cold injury, chiefly frostbite, in ground troops. Over 8,000 of these cases occurred in the winter of 1950—1951. This represents 10 % of the wounded casualties of these two conflicts.

3. Epidemiology. Cold injury, as it involves a military population, behaves in general according to accepted epidemiologic principles. A specific agent is present and a variety of environmental and host factors influence the incidence, prevalence, type and severity of the injury. These factors combine in the total causation of cold injury, and the influence of each may vary in every situation. Careful evaluation of these factors and their relative effects serves to guide preventive and control activities and dictates specific measures which must be employed according to conditions in the unit concerned.

a. Agent Factors. Cold is the specific agent in cold injury and is the immediate cause of tissue damage without respect to modifying factors. If, however, the effect of cold is considered to be loss of body heat, an agent relationship with modes of heat conduction and heat production is apparent, and the ways in which various host and environmental factors modify cold injury become clear. Therefore the effect of cold cannot be evaluated by ambient temperature alone.

b. Environmental Factors.

(1) *Weather.* Weather is a predominant influence in causing cold injury. Temperature, humidity, precipitation, and wind modify the rate of body heat loss. Low temperatures and low relative humidity favor frostbite, whereas higher temperatures together with moisture are usually associated with trench foot. Wind velocity accelerates body heat loss under both wet and cold conditions. This wind-chill effect is shown in table 1, and this effect must be thoroughly understood by all personnel in areas where cold injury is possible. It must be recognized also that the equivalent temperature depicted on the wind chill chart relates to the cooling effect on exposed skin. The specific implications of the wind-chill phenomenon then, relate to a freezing hazard of unprotected body surfaces—face or ears. It is important also when considering host factors in general that increasing wind should be recognized as a significant factor in total body heat loss.

(2) *Type of combat action.* The incidence of cold injury varies greatly according to type of combat action. Units in reserve or in rest areas have few cases; on holding missions or on static defense, exposure is greater and a moderate increase in incidence is expected; on active defense or offense, marked increases usually occur. Immobility under fire, prolonged exposure, lack of opportunity to rewarm and change clothing or carry out personal hygienic measures, fatigue, and state of nutrition may all be involved.

(3) *Clothing.* In warfare, where exposure to cold may be prolonged, adequate clothing properly worn is essential to welfare and survival. Clothing for cold weather combat has been designed to be worn as an assembly for protection of the head, torso, and extremities. Failure to wear the total assembly, and inadequate supplies of proper sizes of clothing are important factors in cold injury. The assembly depends upon the layering principle to conserve body heat. Loose layers of clothing with air space between them, under an outer wind- and water-resistant garment provide maximum protection. It is flexible in that inner layers may be removed for comfort and efficiency to permit escape of perspiration in higher ambient temperatures or during strenuous physical exertion. Clothing wet by perspiration loses much of its insulating value, and care must be taken to prevent perspiration from accumulating in the clothing. In all forms of cold injury, prevention of loss of body heat is important. All articles of clothing must be loose, to avoid constriction and tightness. Clothing must be kept free of grease and dirt.

c. Host Factors.

(1) *Age.* Within the usual age range of combat personnel, age is not significant; nor is there substantial evidence for greater susceptibility outside of the age range of combat personnel.

(2) *Rank.* Trench foot and frostbite have a high selectivity for the front line riflemen, and predominantly those of the lower ranks. The decreased incidence of cold injury among higher ranks is the reflection of a combination of factors such as possible lesser degree of exposure, experience, and receptivity to training.

(3) *Previous cold injury.* A previous episode of cold injury (frostbite or trenchfoot) definitely increases the individual's risk of subsequent cold injury, not necessarily involving the part previously injured. Minor degrees of superficial cold injury, when completely healed, probably do not predispose to subsequent injury sufficiently to require profiling or other restriction or assignments.

(4) *Fatigue.* Fatigue is a factor contributory to cold injury. Mental weariness may cause apathy leading to neglect of acts vital to survival. This occurs more frequently in personnel who have been in combat for 30 days or more without rest. Frequent rotation of troops from the front lines for even short periods lessens the influence of the fatigue factor.

(5) *Concomitant injury.* Experience has shown that injuries resulting in significant blood loss or shock reduce effective blood flow to extremities and predispose to cold injury.

(6) *Discipline, training, and experience.* THIS IS A PREVENTABLE INJURY. Proper use of simple preventive measures which are *inspected* and enforced by officers and noncommissioned officers will markedly reduce the incidence of cold injury. Individual and unit discipline, training, and experience are closely related in their influence upon the incidence of cold injury. Well trained and disciplined men suffer less than others from the cold, as they are better able to care for themselves through personal hygiene, care of feet, change of clothing, exercise of the extremities in pinned-down positions, and similar effective measures.

(7) *Psychosocial factors.* Cold injury tends to occur in passive, negativistic individuals, who tend to display little muscular activity and are prone to pay less attention to carrying extra footwear and changing of socks when needed.

(8) *Race.* In terms of numbers at risk, and independent of geographic origin, the Negro appears to be considerably more vulnerable to frostbite than the Caucasian. This has been a consistent observation dating from World War II through Korea and into the present in Alaska. It suggests that the Negro must be particularly vigilant respecting hand and foot care during cold exposure. By training and proper use of protective clothing, the Negro serviceman can overcome his physiological handicap and serve in cold climates without excessive risk of cold injury.

(9) *Geographic origin.* Caucasian personnel from warmer climates in the United States (where the mean minimum January temperature is above 20°F.) *appear* to be predisposed to cold injury. The basic factor involved, however, may well be psychosocial and educational, rather than geographic.

(10) *Nutrition.* Starvation or semistarvation predisposes to cold injury. Adequately clothed and protected personnel in cold climates do not require more than the normally provided military ration of 3600 to 4600 calories. Evidence of a need for specific nutrients in the diet for cold injury prevention or treatment is lacking. However, the increased exercise requirements imposed by heavy clothing and equipment and the increased difficulty of movement in snow covered terrain can increase caloric requirements up to 7000 calories per day. For example, cross-country skiing can require the expenditure of between 600 to 1200 calories per hour.

(11) *Activity.* Too great or too little activity may contribute to cold injury. Overactivity can cause the loss of large amounts of body heat by perspiration, which become trapped in excess clothing, markedly reducing the insulating quality of the clothing. Conversely, immobility causes decreased heat production with the danger of resultant cooling, especially of extremities and pressure areas of the body.

(12) *Drugs and medication.* Physicians should advise patients of any adverse effects on peripheral circulation or sweating when prescribing drugs and medications in cold climates. Morphine is an important example of a drug with a significant effect on cutaneous circulation and therefore on body heat balance. Chlorpromazine, barbiturates, clopenthixol are other drugs which can predispose to cold injury.

(13) *Alcohol.* Because of its questionable vascular effects, coupled with its influence on judgement, alcohol should be avoided under conditions of extreme cold. The dangers of hypothermia and frostbite are increased greatly under its influence.

4. Duration of Exposure. *a.* The duration of exposure resulting in immersion foot varies according to the ambient air temperature and the temperature of the water, and therefore, with season and latitude. Immersion of the torso and extremities in water below 50°F., as in northern latitudes, may result in death in less than one hour due to excessive lowering of core temperature (hypothermia).

b. The average duration of exposure resulting in trench foot is three days in a temperature range of 32° to 50°F. with a time range of from a few hours to 14 days.

c. The average duration of exposure resulting in frostbite is 10 hours, with a usual range of 1 to 20 hours, but this varies for different types of activity. For patrols and other offensive maneuvers, the period of greatest susceptibility usually begins at that time when walking ceases either because of arrival at an ambush or because of being pinned down by the enemy. In guard situations, the same can be said for the time of posting of the guard; in vehicular movements, for the time of mounting; and in defensive maneuvers (fox holes), for the time immobilization by enemy fire begins. A decrease in physical activity reduces the exposure time necessary to produce cold injury.

5. Prevention. *a. Cold Injuries are Preventable.* Successful prevention requires vigorous command leadership and proper use of preventive measures which are inspected and enforced. Prior planning, cold weather training, and the provision of proper clothing and equipment are paramount. Specific preventive measures are directed toward conservation of total body heat, avoiding unnecessary prolonged exposure of personnel to cold, moisture, and activities favoring cold injury.

b. Meterological Data. All commanders should be familiar with the utilization of simple meterological data such as humidity, temperature, wind, and ground surface conditions which influence the risk of cold injury. Some weather conditions will require shortening of the exposure time of individuals engaged in patrols, guard, or motor movements in unheated vehicles despite the adequacy of their clothing and equipment. These can frequently be anticipated by the utilization of meterological data and existing weather conditions to predict the hazard for the ensuing 12-hour period. Thus, clothing may be provided for anticipated weather conditions and the periods of exposure shortened, if indicated.

c. Cold Injury Control Officer. Each platoon and squad or comparable-sized unit should have a Cold Injury Control Officer or Noncommissioned Officer, who should be carefully selected on the basis of leadership, interest, and ability to supervise others in simple but constant preventive activities. Frequent observation of his men for early signs and symptoms of cold injury is of the utmost importance. He should check his men daily for good personal hygiene, especially of the feet where a change of socks at appropriate intervals, along with a reasonable effort to keep them clean and dry, is essential. He should likewise encourage efforts to take appropriate exercises, to avoid constriction of the extremities by clothing and footgear, and to avoid undesirable dependency of parts during rest periods or periods of unavoidable immobility.

d. The Buddy System. Members of squads and patrols should be taught to observe their companions for evidence of cold injury. If sudden blanching of the skin is noted promptly, immediate care will usually prevent the development of a more serious cold injury. Holding (not rubbing) a warm hand on the blanched area of an ear, nose, or cheek until a normal color has been restored will be adequate rewarming. The part must then be protected against further serious exposure to cold. Fingers can be warmed against the skin of the abdomen or the armpit. Toes can be rewarmed by holding them against a companion's chest or abdomen under his outer clothing. A fairly reliable symptom of incipient frostbite of fingers and toes is the sudden and complete cessation of the sensation of cold or discomfort in the part. This is often followed by a pleasant feeling of warmth. If these danger signals are instantly heeded, cold injury can be prevented.

e. Clothing.

(1) A standard number of layers of clothing cannot be prescribed for universal wear throughout winter months. Flexibility must be provided for local conditions. Certain basic principles are important, including the ventilation of the body during physical activity, the cleanliness and repair of clothing to prevent loss of insulation, and the avoidance of constriction produced by snug fitting socks, boots, underwear, sweaters, jackets and trousers.

(2) Ground forces personnel in cold areas *must* be equipped with the insulated rubber combat boot. Frequent change of socks is important with this boot because of increased sweating, retention of sweat, and a lowered resistance to epidermophytosis. Although sweating in this boot does not contribute to the loss of insulation, it nevertheless leads to the softening of the soles of the feet by the retained sweat. Trauma to macerated tissues, produced by walking, results in a denudation or loss of skin from the soles of the feet which may necessitate hospitalization. *Cold injuries to the foot have been reported when wearing this insulated boot.* These usually result from inactivity and dependency of the foot, as occurs with prolonged sitting or standing without frequent foot or leg movement. Skin cooling rates in feet protected by the insulated boot in sedentary individuals are predicted within a reasonable range of variation (e.g., 0.3°—0.5°F. drop per minute at −25°F.). If attempts at warming with increased physical activity are not begun until the sensation of cold has been stimulated (skin temperature 50° to 55°F.) only vigorous large muscle activity for a protracted period will create sufficient heat to change the foot temperature. Even more important is the fact that in some individuals, once cold, skin temperatures do not rise with exercise carried to limits of reasonable endurance. Therefore, when the tactical situation permits, it is important for unit commanders to protect against this irretrievable situation by instituting periodic vigorous activity when ambient temperatures reach −20°F. or below at hourly intervals in sedentary troops. This type of prophylaxis should not be carried to the point of perspiration.

(3) In all types of footgear, feet perspire more and are generally less well ventilated than other parts of the body, so that moisture accumulates in socks, decreasing their insulating quality. Because of this and

the fact that the feet are susceptible to cold injury and less frequently observed than the remainder of the body, special foot and sock care is essential. Extra socks should be carried by all ground force personnel. Socks damp from perspiration will dry if carried unfolded inside the shirt and should be changed daily and washed whenever the opportunity permits. Socks and other clothing charged with dirt, grease, or mineral salts from perspiration will conduct heat more rapidly, thus affording less protection against the cold.

f. Directives in Use of Clothing.

(1) When working, remove excess layers of clothing before perspiration starts so that clothing does not become wet. Avoid wetting clothing or footgear, since moisture causes loss of insulating quality.

(2) Wear clothing and footgear loose enough to permit layers of air to provide good insulation and to permit good circulation of blood to all parts of the body. Avoid tight-fitting uniforms. They are dangerous in cold climates.

(3) Keep hands well protected; mittens are more protective than gloves. Avoid lengthy exposure of bare hands and wrists that will cause stiffening and reduce circulation, since it takes a long time to recondition the hands to normal use. DO NOT TOUCH METAL, SNOW, OR OTHER COLD OBJECTS WITH BARE HANDS. DO NOT SPILL GASOLINE ON SKIN OR CLOTHING.

(4) Activity of large muscle groups of the shoulders, trunk and legs are required in order to generate and maintain body heat. When the situation prohibits such gross activities, frequent changes of positions, moving toes, feet, legs, fingers, arms, and hands, and to a lesser extent, isometric contractions are less satisfactory alternatives. In such situations, some delay in heat loss can be accomplished by sitting or standing on insulating material such as wood, cardboard, or other poor conductors rather than on cold or wet ground or snow.

(5) Remove excess clothing when in a warm enclosure or in front of a fire to avoid sweating and undue dilation of skin blood vessels.

g. Unusually Susceptible Groups. Certain groups of individuals require greater protection and supervision of preventive measures in order to prevent cold injury. These include: The fatigue group, the racial group, the geographic origin group, the previous cold-injured group, and the negativistic group (para 3c)

h. Summary. Effective prevention and control of cold injury must include the following:

(1) Thorough appreciation and understanding of the potential losses from cold injury in winter training or combat by command staff, technical personnel, and all combat components alike.

(2) Full command support by echelon of a comprehensive and practical cold injury prevention and control program.

(3) Training of all personnel in the early recognition of cold injury.

(4) Indoctrination of all personnel in individual and unit cold injury prevention and control.

(5) An effective clothing supply program that provides for the daily needs of the farthest forward combat personnel.

(6) The establishment of facilities to insure appropriate sizing and fitting of clothing and footgear and the correct utilization of clothing assemblies by all combat personnel to prevent undue exposure.

(7) The establishment and maintenance of a high level of foot and clothing discipline for individual and unit alike.

(8) A theater rotation policy under which all combat units must rotate individuals and units according to the degree of exposure and within the limits of the cold injury incubation period. Only the most critical combat situations should be permitted to interfere with the practice of rotation.

(9) The establishment of effective provisions in forward areas for evacuation and/or treatment of patients suffering from cold trauma.

6. Pathogenesis. *a.* The pathogenesis of tissue injury resulting from cold exposure is not clearly understood. Pathogenic mechanisms thought to contribute to tissue loss from cold injury are—

(1) Direct metabolic impairment to the cold.

(2) Intracellular molecular changes due to hyperosmolarity subsequent to crystallization of extracellular water.

(3) Cellular and structural damage due to the mechanical effect of freezing.

(4) Vascular damage with decreased tissue perfusion and tissue hypoxia.

b. Experimental evidence relative to pathogenic contribution of direct metabolic impairment of tissue exposed to cold is largely inferential. Acute necrosis of muscle fibers with nuclear, mitochondrial, and plasma membrane changes, with intact basement membrane have been seen following "momentary" *in situ* application of extreme cold. This has been interpreted as an implication of direct thermal injury. However, similar muscle fiber changes have been described following tourniquet ischemia maintained for as little as 30 minutes and clearly related to tissue hypoxia. There is little question, however, that cold by itself has been

shown to differentially affect certain cellular enzymes systems which could have an adverse effect on cellular function. Prolonged exposure to non-freezing cold has been shown to produce elevations in serum enzymes derived from muscle, suggesting alteration of cell plasma membrane integrity and, indeed, histologic changes have been observed. Cold temperature in the above freezing range will also denature certain lipoproteins and cause precipitation of the denatured complex. Freezing accelerates this reaction. The possible correlation of cold induced lipoprotein denaturation and the observed preferential nerve tissue damage as seen in cold injury is apparent.

c. Intracellular hyperosmolarity consequent to extracellular tissue water crystallization and plasmolysis occurs with freezing and results in cellular dehydration. That this can cause protein denaturation has been shown experimentally. Supporting this mechanism as a source of cellular damage with freezing is the fact that low molecular weight substances which have the capacity to diffuse into intracellular fluid space have been shown to act as osmotic buffers and to prevent the severe cellular damage occurring with freezing.

d. In extremely rapid induction of freezing, intracellular as well as extracellular ice crystal formation can occur with probable mechanical disruption of the cell membranes. There is little evidence that such a mechanism operates in the usual clinical freezing injury inasmuch as there is much more gradual heat loss and the formation of ice is almost totally extracellular. Even with rapid freezing, successful preservation and subsequent viability of spermatozoa, aortic grafts, whole blood, marrow, ovarian tissue, and renal cell suspension indicate that individual cellular damage from mechanical distortion is negligible.

e. There is persuasive evidence that a major contributing factor to tissue loss subsequent to freezing injury is hypoxia resulting from microcirculatory failure. By means of microangiographic techniques and special histologic methods, corpuscular clumping, capillary stasis, and irreversible occlusion in small vessels by aggregates of cells has been demonstrated in rewarmed tissue following freezing. Intravital microscopic observation, after thaw of frozen tissue has demonstrated progressive obstruction of small vessels by aggregations of what appear to be platelets, white cells, and red cells, and eventual thrombus formation. In addition, it has been shown that skin, subjected to a freezing injury severe enough to cause necrosis and total slough, will survive if autotransplanted to a normal graft bed where vascularization can occur. Normal skin, autotransplanted to the frozen graft bed, however, fails to vascularize and sloughs due to vascular damage in the cold injured tissue. Perhaps the most compelling evidence substantiating the role of microcirculatory failure in the pathogenesis of freezing injury comes from studies in experimental animals in which the blood flow has been serially measured by venous occlusion plethysmography, or radioactive substances such as Xe^{133} or I^{131} RISA, following thaw of a previously frozen extremity. Such studies have shown diminution in blood flow minutes following thaw in those extremities suffering severe injury with tissue loss with progressive fall off in blood flow as gangrene develops. Conversely, less serious injuries showed no significant change in capillary blood flow. These differences were apparent sufficiently early in experimental animals that one could reliably predict the outcome of a particular injury. In addition as a result of tissue hypoxia and possibly the direct effect of cold, there is increase in capillary permeability with loss of plasma into extravascular space and further hemoconcentration with increased blood viscosity and stasis.

f. The above techniques have not been applied as yet to clinical injury, but some clinical evidence for the role of microvascular integrity in the pathogenesis of freezing injury comes from studies which have demonstrated a beneficial effect on tissue survival by measures such as rapid rewarming, sympathectomy, anticoagulations, vasodilators, and administration of intravascular cellular aggregation inhibitors which improve local tissue perfusion. Because of the difficulty in predicting the outcome in clinical cold injury, there is conflicting opinion as to the efficacy of some of these procedures.

g. In summary, vascular damage with decreased tissue perfusion and resulting tissue hypoxia leads to tissue damage in both non-freezing and freezing cold injury. With freezing, there is additional contribution of cellular dessication. Although cold may alter normal cellular metabolic pathways by means of differential suppression of enzyme systems, the significance of this mechanism related to cold injury remains unclear.

7. Clinical Manifestations. *a. Symptoms and Signs During Exposure.* The lack of warning symptoms emphasizes the insidious nature of cold injury, which, unfortunately, is casually overlooked by many troops and commanders temporarily subjected to cold climatic conditions. The only warning symptoms may be tingling, stinging, or at most a dull aching sensation of the exposed part followed by numbness. The skin briefly may appear red and then becomes pale or waxy white. At this stage the affected part may feel "like a block of wood." If freezing has occurred, the tissue

appears "dead white", and is hard or even brittle, with complete lack of sensation and movement. Medical personnel rarely see these stages of cold injury, as thawing or rewarming with the development of the manifestation of the injury has usually occurred. Data from previous wars indicate that a medical officer saw less than 2 percent of cold injury casualties before rewarming had occurred.

b. Differentiation. The differentiation of types of cold injury, (i.e., freezing vs. non-freezing), may be diagnostically difficult early after rewarming has occurred. The classification into trench foot, immersion foot, and frostbite is of use chiefly as a description of the mode of injury. Tissue injury is largely the result of vascular damage and hence will be similar in all forms of cold injury, the major variable being the severity. The type of cold injury usually encountered is a graded injury, which may involve superficial and deep frostbite, and conceivably adjacent non-freezing injury in less exposed parts.

c. Degree of Cold Injury. Early evaluation of the severity of injury is extremely difficult even to the most experienced medical officer. Definitive classification of severity into first, second, third, and fourth degree is possible only in retrospect, after the case is completed. However, since at the two extremes there can be some clinical distinction, this classification has some value.

(1) *First degree—hyperemia and edema.* After rewarming, the skin becomes mottled, cyanotic, red, hot, and dry. The apparent hyperemia blanches poorly on pressure and capillary filling is sluggish or absent. There frequently is intense itching or burning, and a later deep seated ache. The swelling begins within three hours and may persist for ten or more days if the individual remains on duty. Edema usually disappears in less than five days if the patient is kept at bed rest. Desquamation of the superficial layers of skin may begin within five to ten days after injury and may continue as long as a month. In more severe cases, deep aching pain, paresthesia, cyanosis, hyperhidrosis and coldness of the injured part may appear two or three weeks after injury and persist for many months. In milder cases, symptoms may persist for several hours, causing intense discomfort but gradually disappear without serious sequelae.

(2) *Second degree—hyperemia and vesicle formation.* Hyperemia, edema, and burning pain are early manifestations after rewarming. The skin becomes deep red, with mottled cyanosis and feels hot and dry to touch. Swelling begins within two or three hours. There may be a sensation of tingling and burning of gradually increasing intensity, but light touch and position sense are frequently absent. In more severe cases, blisters and even hugh blebs may appear within 6 to 12 hours. The large clear appearance of these blebs appeared early and extending nearly to the tips of involved digits is generally felt to be a valuable sign identifying the injury as second degree. Pain, usually a deep aching sensation in association with intense burning, increases and may require medication. Edema is usually not marked and disappears within 3 to 5 days after rewarming if the patient is not ambulatory. The vesicles are superficial to the germinative layer and frequently occur on the great toe and heel or the dorsum of one or more fingers. They dry, forming black eschars within 10 to 24 days after rewarming. There may be slight limitation of motion. Throbbing or aching pain is usually noted 10 to 20 days after injury. Hyperhidrosis frequently occurs between the second and third week. The eschar gradually separates, revealing intact skin which is thin, soft, poorly keratinized, and easily traumatized.

(3) *Third degree—necrosis of skin and cutaneous tissue.* This injury involves whole skin thickness and extends into subcutaneous tissue leading to ulceration. Vesicles may be present but are more commonly violaceous or hemorrhagic, smaller, and do not extend to the tip of the involved digits. Edema of the entire foot or hand is usually present and appears in an average of 6 days. Most patients have burning, aching, throbbing, or shooting pains beginning on the 5th to 17th day following an early period of anesthesia. The skin overlaying the area of third degree frostbite may form a black, hard, dry eschar. This finally separates, exposing underlying granulation tissue. Healing occurs in an average of 68 days. Hyperhidrosis and cyanosis may appear between the 4th to 10th week after the injury and persist for months, resulting in a prolonged, uncomfortable convalescence. Trauma and infection due to injury other than cold may complicate this degree of injury and may result in extensive tissue loss, systemic manifestations of infection, and even wet gangrene requiring emergency amputation.

(4) *Fourth degree—complete necrosis and loss of tissue.* There is destruction of the entire thickness of the part including bone resulting in loss of the injured part. Upon rewarming, the skin may turn deep red, purple, or appear mottled and cyanotic. The area involved is usually anesthetic and although there may be rapidly developing edema proximal to the area of fourth degree injury, reaching a maximum within 6 to 12 hours, the area itself may show no significant increase in volume, but rapidly progress to dry gangrene and mummification. In other fourth degree injuries, however, tissue injury slowly progresses and

edema may be more prominent, and eschar formation and gangrene not evident until 2 or 3 weeks after injury. There may be severe paresthesias appearing 3 to 13 days after rewarming. The line of demarcation becomes apparent in an average of 36 days and extends down to the bone between 60 to 80 days after injury.

d. Early Diagnosis and Reliable Prognostic Signs. As previously pointed out, classification of cold injury as to degree is a retrospective diagnosis. Even to the experienced, early diagnosis of severity of the cold injury is extremely difficult. In the early stages (first 48 to 72 hours) one can hope to differentiate accurately only between the term *superficial* (loss of skin or less) or *deep* (loss of skin and tissue) injuries. Such an estimation should be made since it has both clinical as well as epidemiologic importance.

(1) *Good prognostic indicators.* Indicative of superficial injury.

(*a*) Large, clear blebs developing early and extending to the tips of the digits.

(*b*) Rapid return of sensation.

(*c*) Return of normal (warm) temperature in injured area.

(*d*) Rapid capillary filling time after pressure blanching.

(*e*) Pink or midly erythematous skin color which blanches.

(2) *Poor prognostic indicators.* More indicative of deep injury.

(*a*) Hard, white, cold and insensitive.

(*b*) Cold and cyanotic without blebs or blisters.

(*c*) Complete absence of edema.

(*d*) Dark hemorrhagic blebs.

(*e*) Early mummification.

(*f*) Constitutional signs of tissue necrosis, i.e., fever, tachycardia, prostration.

(*g*) Superimposed trauma.

(*h*) Cyanotic or dark red skin color which does not blanch on pressure.

e. Other Prognostic Clues. Four additional pieces of information are of valuable assistance in the initial estimate of severity of injury.

(1) History of freeze—thaw—re-freeze injury, as not infrequently encountered among mountain troops or long range patrols, should serve to alert the clinician to the likelihood of severe freezing injury.

(2) Inquiry into duration of exposure may give valuable insight into the degree of injury. One clinical series showed that all patients whose injured extremity was exposed for longer than one hour at ambient temperatures below 20°F. lost tissue.

(3) Similarly, knowledge of ambient temperature at which exposure took place will improve accuracy of judgement as to degree of injury.

(4) Contact of unprotected skin with metal with freezing ambient temperature increases the rate of heat loss and consequently the potential for serious injury.

8. Treatment. Treatment of cold injury depends upon the time elapsed after the injury, the severity of the injury, the presence of complications, and the area affected. In military operations, treatment will be influenced by the tactical situation, as well as the facilities available for the evacuation of casualties. Most cold injuries appear en masse, during periods of entense combat, and at the time that large numbers of casualties occur. *The examination and treatment of life-endangering wounds must take precedence over cold injuries.* Highly individualized treatment is difficult during military operations because of the large numbers of patients who require treatment almost simultaneously.

a. The treatment of cold injury is divided into buddy system or first aid, initial or emergency medical treatment in forward areas, and definitive treatment after the patient has reached a hospital.

(1) *First aid.*

(*a*) If cold injury occurs, the patient should be restricted from his usual duties or activities until the severity of injury can be evaluated. A doctor should see the injury as soon as possible.

(*b*) All constricting items of clothing such as boots, socks or gloves should be removed from the site of injury. The injured area must then be protected from further cold injury by blankets or any available clothing which is not constricting.

(*c*) Smoking, drinking of alcohol and the application of medications, salves or ointments are prohibited. Blisters must not be opened. Drinking hot liquids is encouraged, if available.

(*d*) If the lower extremity is involved, treat as a litter patient with the part level or slightly elevated. In unusual circumstances where travel on foot is the *only* means of evacuation for frostbite of the feet, thawing of the injured area is not indicated until the patient reaches an aid station and medical help.

(2) *Initial or emergency treatment.*

(*a*) In many ways cold injury is not unlike a burn. It should never be treated lightly as the tissue loss and nerve damage associated with cold injury frequently results in permanent disability. The two most important tenets in the treatment of cold injuries are conservative surgical management and prevention of infection. If a diagnosis of cold injury is made, the patient should be restricted from his usual activities or

duties until the severity of the injury can be determined.

(*b*) Systemic hypothermia if present should be treated as described in paragraph 9*c*.

(*c*) All constricting items of clothing, such as boots, gloves, and socks should be carefully removed from the site of injury.

(*d*) If freezing has occurred and the affected tissue is still frozen, it should be rapidly thawed in a water bath *carefully controlled at 40°C.* (104°F.), not to exceed 42°C. (109°F.). Thawing is determined by return of sensation (usually pain), return of color (frequently deep red or even purple) and the observation that the tissue is soft and no longer brittle or hard. If warm water and a thermometer are not available, the part may be rewarmed with body heat from another portion of the body (e.g. axilla) or another person. Under no circumstances should snow, ice water, grease, massage, walking, or dry heat be used. There is great danger of furthering injury from over-zealous application of heat, particularly when the frozen tissue is anesthetic. The only safe and effective method is the use of a water bath with thermometer control. Rapid warming should not be continued beyond the time when thawing is complete and should not be instituted if thawing has already occurred. Warming above 37°C. (98°F.) is not recommended for nonfreezing cold injuries. Intense pain may ensue from rapid thawing and pain medication such as morphine may be necessary. Cold injury is no contraindication for narcotics or other pain medications, but accompanying injuries may govern the choice of medication.

(*e*) Smoking is prohibited in all types of cold injury. Nicotine causes vasoconstriction and may further decrease the blood supply to the injured tissue. Mild stimulants such as tea or coffee are useful, especially if the patient is generally cold. Alcohol is not recommended because of its variable effect on peripheral blood flow.

(*f*) All patients with cold injuries of the lower extremities should be treated as litter patients. The affected part should be level or slightly elevated. Extreme care must be taken to prevent further trauma and infection.

(*g*) Blisters and blebs should not be ruptured. Salves, ointments, and greases are contraindicated. If transport is necessary, the affected part should be loosely wrapped with fluff bandages of sterile gauze, care being taken to prevent chafing blebs.

(*h*) Tentanus toxoid (0.5 cc I.M.) booster should be administered.

(*i*) Prophylactic antibiotics for the cold injury are not advised at this time. If infection is apparent, suitable antibiotics should be started, preferably after a culture is taken.

(*j*) All victims of cold injuries should be evaluated by a medical officer at the earliest possible time.

(*k*) Very mild cold injuries such as chilblains require little specific treatment. However, because the manifestations of tissue damage are slow to appear, care must be taken not to underestimate any cold injury, and the patient should be seen by the medical officer. If hives (urticaria) are a prominent feature, antihistamines such as diphenhydramine hydrochloride (50 mg. orally three times a day) may give benefit.

(3) *Definitive treatment in the hospital.*

(*a*) Absolute bed rest is mandatory for those patients with cold injury of the feet. Bed rest should be maintained in first and second degree cold injuries until subsidence of edema and/or complete drying of vesicles or bullae have occurred. Patients with third degree injuries should remain at bed rest until the area of ucleration is epithelialized or definitive surgery has been completed.

(*b*) All cold injuries are treated "open" in a comfortably warm (72° to 78°F. room). Sterile precautions including sterile drapes under the injured part, clean gowns and masks for attendants, and sterile gloves for handling the injured parts or linen changing is recommended for all cold injuries of second degree or worse. A cradle supporting sterile drapes over the injured part is recommended. As in burns, extreme care must be taken to prevent further trauma and infection.

(*c*) Twice daily 20-minute whirlpool treatment in 37°C. (98.6°F.) water to which hexachlorophene has been added is recommended, starting immediately. The gentle debridement and cleansing action of the whirlpool are extremely effective in minimizing maceration and infection. If whirlpool is not available the affected part should be gently agitated back-and-forth in a basin containing water and hexachlorophene at 37°C. (98.6°F.). Following this, the extremity should be gently dried using sterile towels and sterile pledgets of cotton placed between digits of the feet. Only under circumstances of mass casualties where this treatment is impossible should the injury be washed with any solid material to secure superficial debridement.

(*d*) Periodic wound cultures for bacterial growth and antibiotic sensitivity are recommended. If infection develops, prompt systemic administration of the appropriate antibiotic is indicated. Routine administration of antibiotics for cold injuries is not advised.

(*e*) Active physiotherapy should be instituted at the bedside as soon as possible. Attention is directed toward positioning of the feet and movement of fin-

gers, toes and other major joints in order to prevent ankylosis of the joints. The importance of active physiotherapy by the patient during the first six weeks cannot be overemphasized. Very effective active physiotherapy can be accomplished during the whirlpool treatments.

(*f*) Smoking should not be permitted.

(*g*) *Of all the factors which may influence the results of treatment, premature debridement of moist semi-necrotic tissue or premature surgical intervention of any kind are the most deleterious to a good result.*

Unless life-endangering systemic reactions demand an open amputation, the physician should allow physiologic amputation to occur. This may require many weeks or months. Surgical debridement other than whirlpool therapy is almost never required until a stabilized healthy granulation bed is formed at the demarcation line. Definitive amputations or plastic reconstructive procedures should be delayed until a healthy granulation tissue bed is evident following removal of mummified separate tissue and necrotic bone.

(*h*) Any constricting circumferential hard eschar which may jeopardize distal blood flow must be bivalved.

(*i*) The use of surgical sympathectomy following frostbite is of some possible use. There is no evidence of any greater saving of tissue after lumbar or cervical sympathectomy. On the other hand, sympathectomy does cause, due to increased blood flow, a rapid reduction in infection, as well as in edema, pain and hyperhidrosis. Sensation returns more rapidly after sympathectomy. About 6 months to a year after sympatechtomy many patients complain of the extremities being "too dry." The possible use of ganglionic blocking agents to produce a "chemical sympathectomy" should be considered.

(*j*) Patience, understanding, and constant encouragement by the attending physician are essential to good results.

b. The treatment of cold injury is as controversial as the theories regarding its pathogenesis. Many different therapeutic approaches have been suggested, including the use of plaster casts and pressure dressings to control edema and prevent tissue necrosis; rutin to reduce capillary permeability; antihistamines to combat theoretical histamine-like substances liberated at the time of thawing; corticotropin and cortisone; rapid rewarming; anticoagulants, sympathectomy, vasodilators and intravascular cellular aggregation inhibitors to improve blood flow.

(1) The possible beneficial effects of edema control, rutin, antihistamines, and steroids in the treatment of cold injury have not been experimentally nor clinically substantiated.

(2) There is sufficient experimental evidence that rapid thawing at above body temperature improves tissue survival following freezing injury. Clinical experiences has supported this. The mechanism by which tissue injury is benefited is not clearly understood, but probably is related to an improvement in blood flow to the injured tissue. In practical terms, however, the number of frostbite patients who are received prior to thawing are few. There is, therefore, a continuing need for the development of better methods of treatment of all types of cold injury.

(3) Vasodilators have generally not proved effective in the treatment of cold injury either experimentally or clinically. One reason for their failure is the systemic hypotensive effect negating the possible increase in blood flow due to vasodilation proximal to the cold injury.

(4) Anticoagulants should be of value in the treatment of cold injury considering the probable role of vascular damage and microthrombosis in the pathogenesis of tissue injury. However, these same studies have shown that intravascular cellular aggregation, stasis and thrombosis occur rapidly after freezing injury and, to be effective, anticoagulation must be achieved within an hour after thawing. Experimental studies where adequate heparinization had been achieved early after freezing injury and then maintained resulted in improved tissue survival. Most of the clinical experience in Korea was with the delayed use of heparian and was ineffective. There did appear to be a beneficial effect if heparinization was early, within 36 hours after injury. Maintaining adequate coagulation without complications and early anticoagulation of a cold-injured individual who may have other serious injuries are very real problems which would tend to limit its use.

(5) Recent experimental studies have investigated the potential use of the plasma expander, low-molecular-weight dextran, in the treatment of cold injury. This drug, which is administered intravenously, has the unique properties of inhibiting intravascular cellular aggregation, decreasing blood viscosity, and improving blood flow in injured tissue. Beneficial results gained by its use in experimental frostbite have been encouraging. As with anticoagulants, the maximal effect is gained by early administration after injury. There are fewer contraindications for this drug than for anticoagulants. Clinical experience in evaluating the drug for treatment of cold injury, at present, is limited.

9. General Hypothermia. *a. Definition and Classification.* Hypothermia can be defined as a state of internal or core temperature which is below normal in a homothermic organism. This term, however, has come to have a variety of connotations so that is essential in referring to total body hypothermia, to understand that the term may have different meanings according to the degree of hypothermia under discussion, Despite individual differences, age, debility, anesthesia, duration of hypothermia and underlying disease states, sufficient uniformity of clinical and experimental experiences assures a logical basis for categorization. A classification is proposed as follows: Mild hypothermia, 35-32°C; moderate hypothermia, 31-28°C; deep hypothermia, 27-25°C; profound hypothermia, below 25°C.

(1) *Induced hypothermia.* The controlled, elective reduction in core temperature has become increasingly popular and important in a variety of surgical procedures. In this situation, the careful and continuous assisted ventilation and short duration of exposure clearly modify the therapeutic management and the resuscitative result.

(2) *Accidental hypothermia.* Uncontrolled hypothermia has become a well recognized clinical condition with an extremely high mortality rate. The fact that such conditions as drug or alcohol intoxication, diffuse dermatologic disorders, hypothalamic lesions, paraplegia, extremes of age, head trauma, and various endocrinopathies may increase the susceptibility to accidental hypothermia, complicates the formulation of any standard approach to therapy. The military significance evolves from those cases of individuals inadequately protected or otherwise injured and exposed to cold for prolonged periods and following immersion in cold water as in ship, submarine or aircraft accidents. The changing military tactics in an arctic or subarctic environment which involve sustained operations of small units with increasing frequency, the use of snowmobiles with long range capability in military operations as well as helicopter and fixed wing flights for support and rescue missions increase the likelihood of accidental hypothermia. Although it is generally accepted that the incidence of hypothermia and low ambient temperature are directly related, this condition is not confined to any particular geographical location. In cases of accidental hypothermia, the duration of low body temperature is usually unknown with the degree being lower than that permitted during induced hypothermia. Because of the usual severity and the unreliability of the usual clinical signs of "death" under such circumstances, it is imperative that resuscitative measures be undertaken at once. The ever-present threat of fatal cardiac arrhythmias makes accidental hypothermia a medical emergency demanding immediate concern when the diagnosis is made.

b. Clinical Manifestations. Accidental hypothermia may frequently be confused with cerebrovascular accidents, cardiac and respiratory problems and apparent clinical death. In profound cases, the patient is cold, pale, comatose and shows no vital signs. Ventilation is markedly reduced in frequency and may be sufficiently shallow that casual observation will fail to identify any respiratory movement. Pulse may be very faint or unpalpable at the periphery and precordial pulsations may be indefinite or, because of rigidity of skin, undiscernible. The blood pressure may be unobtainable. The patient is unresponsive to painful stimuli with the pupullary reflexes and corneal reflex sluggish or lost below 28°C. Tissue feels semi-rigid and passive movements may be difficult. These physical findings make the diagnosis of accidental hypothermia very difficult particularly in view of the fact that rectal temperatures are most often below that which can be measured on the standard clinical thermometer. A high index of suspicion is necessary to suspect accidental hypothermia with the scant history frequently available. Physical examination, including measurement of a core temperature below 35°C (95°F) with a low reading clinical thermometer or appropriate thermister or thermocouple, and identifying electrical activity of the heart by electrocardiogram, confirm the diagnosis. The fact that drug or alcohol intoxication, pneumonia, pancreatitis, myxedema, renal failure, fluid and electrolyte imbalance, trauma and frostbite are frequently associated with clinical hypothermia create additional problems in resuscitative management. Since no reliable prognostic indicators are available in accidental hypothermia and death may occur abruptly even after apparently successful initial resuscitation and restoration of body temperature toward normal, each case requires constant surveillance.

c. Treatment

(1) The primary intent in the initial treatment is to restore the core temperature to normal levels. The means by which rewarming may be accomplished are generally divided into two broad categories: External or surface methods and internal or core methods. Surface rewarming results in the body shell being warmed in advance of the core, and may be accomplished actively (immersion in 40°C water, heating blankets, hot water bottles, heat cradles) or passively warm room (with or without blanket cover for patient, no active heat applied). External rewarming by active or passive means has been the most widely advocated. Scattered clinical reports and some animal experimentation pre-

viously supported the view that active rewarming may be especially efficacious in cases where exposure (cold water immersion accidents) had been of brief duration. The usual inability to estimate the duration of exposure and the serious hazard of "rewarming shock" and an "after-fall" in core temperature which may result from active rewarming has urged the more conservative course of passive rewarming. However, the mortality rate in clinical identified cases of accidental hypothermia in which resuscitation was attempted using either active or passive means remains extremely high.

(2) In contrast to external rewarming, internal rewarming refers to warming the core in advance of the shell. Although only a few reports have appeared in the clinical literature using internal techniques such as extracorporeal circulation, peritoneal dialysis, and gastric lavage, all cases resulted in successful resuscitation. Furthermore, the advantages of internal rewarming as compare to external methods have been elaborated in recent animal reports. These include the rapidity with which normothermia can be achieved, the more rapid return of cardiac output and the electrocardiogram toward normal and the avoidance of "rewarming shock" or further drop in core temperature during early rewarming. Internal rewarming using extracorporeal circulation has been criticized on the basis of the limited availability of the necessary equipment as well as the delay attendant with assembling the medical-surgical teams necessary to operate complex and expensive equipment. In recent animal experimentation, peritoneal dialysis using 40°C dialysis solutions and hematogenous rewarming using an arteriovenous shunt with disposable interposed heat exchanger have been found to be effective means of core rewarming. Peritoneal dialysis represents a practical, inexpensive therapeutic modality that is widely available, requires minimal specialized training, and can be utilized in almost any medical facility. In addition, peritoneal dialysis has the potential for managing renal insufficiency as well as certain drug intoxications which present all too frequently in association with accidental hypothermia. Although the use of an arteriovenous shunt requires the availability of a surgeon familiar with vascular surgical techniques, elaborate or expensive equipment is not necessary. Either method of internal rewarming can be used rapidly with no more previous preparation and planning than is required for other medical emergencies of similar severity. Review of the available clinical experience combined with experimental evidence strongly recommends that more consideration be given to the use of internal methods of resuscitative rewarming.

(3) The successful management of accidental hypothermia cannot be simplified to relate only to the choice of the rewarming technique used. Particular attention should be directed toward ventilatory support, monitoring of core temperature, vital signs and electrocardiogram, monitoring of arterial pH and blood gases as well as close attention to urine output and blood chemistries. Immediate institution of assisted or mechanical ventilation is indicated with O_2 and with protective measures against aspiration. In the absence of a specific indication, large volume transfusions of IV fluids are contraindicated due to the danger of serious fluid overload on an already compromised cardiovascular system. Stimulant drugs and oral administration of warm liquids are also contraindicated. Monitoring blood gases and pH is essential to intelligent management and forms the only reliable basis for fluid administration. However, pO_2, pCO_2 and pH measured at 37°C must be corrected to the patient's core temperature. In addition, important information regarding the clinical course can be obtained from measuring arterial pressure, central venous pressure and urinary output by indwelling catheter.

(4) Vital signs must be closely monitored and continuous display of the electrocardiogram is essential since fatal arrhythmias may develop without warning. The characteristic electrocardiogram of hypothermia consists of: bradycardia and a concomitant prolongation of the P-R, QRS and Q-T intervals; S-T deviations both upwards and downwards with the T-wave becoming flattened or inverted; development of a "J-wave," or a positive deflection at the junction between the QRS and S-T segment. Many experimental reports have dealt with the use of various pharmacologic agents in controlling the severe ventricular arrhythmias which may occur during hypothermia and/or rewarming. Anti-arrhythmic drugs appear to be of little avail in cases of ventricular fibrillation occurring at core temperature below 28°C. Indeed, at such temperatures most attempts, by whatever means, usually fail and external compression must be employed until the core temperature rises above 28°C. Other cardiac arrhythmias such as ventricular tachycardia and premature ventricular beats have been treated with some success at temperatures below 28°C with quinidine, or lidocaine. The latter appears to be the drug of choice for most of these arrhythmias when given in bolus form.

(5) Hypokalemia is commonly seen in hypothermia despite the usually associated acidosis, which is probably secondary to the intracellular migration of potassium and should not be interpreted as a compelling requirement for vigorous replacement.

(6) As body temperature rises, the usual close at-

tention to tracheobronchial toilet and early identification of developing pneumonia or identification of other intercurrent illness or injury becomes increasingly important. Close observation even after initial successful resuscitation should be maintained for several days inasmuch as catastrophic arrhythmias have occasionally developed when it appeared that acid base balance and cardiovascular performance had returned to normal. The most frequently seen late complications as a result of accidental hypothermia are pneumonia, renal failure, pancreatitis and peripheral cold injury.

10. Sensitization. Frequently in all degrees of cold injury the affected individual becomes sensitized to further exposure to cold. With milder injuries, sensitization to cold may persist only days or weeks, but in more severe injuries, cold sensitivity of the injured part may be permanent. Consequently, additional precautions must be taken by person once injured by cold to prevent further injuries. In certain cases, true "cold sensitivity" or "allergy" may exist. Such persons may demonstrate urticaria or "hives" with intense burning, itching and swelling on exposure to cold. This is generally seen in persons who have had previous cold injury but rarely may occur as a familial trait, or in an individual who has plasma cryoglobulin or cold agglutinins. In such individuals, cold exposure may cause a systemic reaction of generalized urticaria, asthma, and even shock.

11. The Wind Chill Chart (Table 1). *a.* The human body is continually producing and losing heat. Wind increases the loss of heat by reducing the thin layer of warm air next to the skin. This loss increases as the wind speed increases. When the temperature of the air is below freezing and the wind is such that it removes the heat faster than the body can replace it, frostbite may occur. Thus, decreasing the ambient (air) temperature or increasing the wind speed act to increase the danger of frostbite to exposed skin. The combined effect of wind and temperature is expressed in the Wind Chill Chart as an equivalent temperature. This expresses the effective temperature acting upon exposed flesh.

b. Any movement of air past the body has the same cooling effect as wind. This may be produced by walking, running, skiing, or riding in open vehicles. The speed of movement must be considered, in addition to natural wind, when using the Wind Chill Chart.

c. It is emphasized that this chart is of value in predicting frostbite only to exposed flesh. Any clothing or material which stops or reduces the wind will give a degree of protection to the covered area. No attempt should be made to estimate this protection in the use of the Wind Chill Chart. Wet clothing or boots have a much reduced insulating value and will result in heat loss nearly equal to that of exposed flesh.

d. To use the chart, find the estimated or actual wind speed in the lefthand column and the actual temperature in degrees F. in the top row. The equivalent temperature is found where these two intersect. The description below the columns indicates the comparative danger of frostbite to exposed flesh under these conditions. For figures intermediate to those shown, make an interpolation as indicated. For example, at a temperature of $-10°$F., under calm conditions, rate of heat loss from exposed flesh is that of the ambient temperature, that is, ten degrees below zero, Fahrenheit and the danger of frostbite to a properly clothed person is slight. However, if the wind increases to only 10 miles per hour, or if the individual is riding in an open vehicle which is being driven only 10 miles per hour, the rate of heat loss of exposed flesh increases to that which would be experienced with no wind at ambient temperature of $-33°$F. This lies within the zone of increasing danger of frostbite, indicating that protective measures must be taken. *Trench foot and immersion foot may occur at any point on this chart.*

e. The Wind Chill Chart shows only the cooling power of wind on exposed flesh, giving the equivalent *rate* of cooling as compared to what would be experienced under calm conditions at the lower temperature. No matter how great the wind velocity, exposed flesh will not freeze so long as the temperature of the wind remains above freezing. This statement applies only to dry skin, since moisture introduces the added factor of cooling by evaporation.

12. References: *a.* Frostbite and Hypothermia—Current Concepts—A Symposium. Alaska Medicine, Volume 15, Number 2, March 1973.

b. Soldier's Handbook for Individual Operations and Survival in Cold Weather Areas.

c. TM 10-275, Cold Weather Clothing and Sleeping Equipment.

d. FM 31-70, Basic Cold Weather Manual.

e. FM 31-71, Northern Operations.

Table 1. Cooling Power of Wind on Exposed Flesh Expressed as an Equivalent Temperature (under calm conditions)

Estimated wind speed (in mph)	Actual Thermometer Reading (° F.)											
	50	40	30	20	10	0	—10	—20	—30	—40	—50	—60
	EQUIVALENT 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	4	—9	—24	—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	16	0	—15	—29	—44	—59	—74	—88	—104	—118	—133
30	28	13	—2	—18	—33	—48	—63	—79	—94	—109	—125	—140
35	27	11	—4	—21	—35	—51	—67	—82	—98	—113	—129	—145
40	26	10	—6	—21	—37	—53	—69	—85	—100	—116	—132	—148
(wind speeds greater than 40 mph have little additional effect.)	LITTLE DANGER (for properly clothed person) Maximum danger of false sense of security.				INCREASING DANGER Danger from freezing of exposed flesh			GREAT DANGER				
	Trenchfoot and immersion foot may occur at any point on this chart.											

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