

## CHAPTER III

# Burn Injury

Extensive use of the various fuels needed to provide both ground and air mobility for the present-day armed forces increases the risk of thermal burns in military personnel. During times of conflict, the possibility of the unintended ignition of these fuels is greatly increased, as is the chance of thermal injury from antipersonnel and other weapons. The development of thermonuclear devices has created the possibility of virtually instantaneous generation of large numbers of burn patients, creating not only medical but also severe logistical problems.

Even under the best conditions, the simultaneous arrival of many extensively burned patients at any hospital disrupts the activities of the professional and paramedical staff and places heavy demands upon the logistical system of that treatment facility. Recent laboratory developments and the clinically demonstrated efficacy of topical chemotherapy have resulted in general acceptance of simplified burn treatment techniques readily adaptable to the combat surgery environment.

The first priority in the management of the burn patient is given to maintenance of the airway, control of hemorrhage, and prompt institution of resuscitative therapy. The presence of associated traumatic wounds in patients with burn injuries may complicate the management of their burns and vice versa. The essence of the successful treatment of burn patients, with or without other traumatic injuries, is effective triage, timely diagnosis, accurate assessment of surgical priority, and appropriate resuscitation.

### ETIOLOGIC AGENTS

Ignition of gasoline and other fuels accounts for the greatest number of thermal injuries. Flame or flash burns may be caused by various other agents contained in explosive devices. Casualties

with chemical burns and burns from white phosphorus require immediate wound care in contrast to those with "conventional" burns. Thermal injury created by electric current also deserves separate consideration because of special treatment requirements.

Even in the combat zone, burns resulting from carelessness outnumber those resulting from hostile action. The enforcement of safety procedures and existing regulations will reduce such occurrences. The use of gloves, goggles, protective headgear, and flame-retardant clothing by personnel at high risk will also minimize, if not prevent, thermal injury in those individuals. This equipment is particularly important to fire-rescue personnel, and the use of these items should be strictly enforced, even (within limits) at the expense of personal comfort.

### MAGNITUDE OF INJURY

The severity of thermal injury is dependent upon the depth and extent of the burn. These two factors determine not only mortality and initial treatment requirements, but also morbidity, metabolic consequences of injury, character of healing, and the ultimate functional result.

The extent of the body surface burned can be estimated by employing the "rule of nines." The distribution of surface area by anatomical part in the adult is illustrated in Figure 15, showing the percentage of total skin surface represented by each body part to be: head and neck, 9%; anterior trunk, 18%; posterior trunk, 18%, upper extremities, 9% each; lower extremities, 18% each; and genitalia and perineum, 1%.

To estimate the extent of irregularly disposed burns one can make use of the fact that one surface of the casualty's hand represents approximately 1% of his total body surface. Patients with burns of more than 15% of the body surface typically require some resuscitative treatment and, in most situations, are best cared for in the hospital. Young soldiers tolerate thermal injury best, while older casualties (above 50) and the very young have greater mortality rates for a given extent of burn. The location of the burn influences not only prognosis but also the need for hospitalization. Small burns of the face, hands, feet, or perineum may require hospitalization, even if these limited areas are the only sites of burn injury.

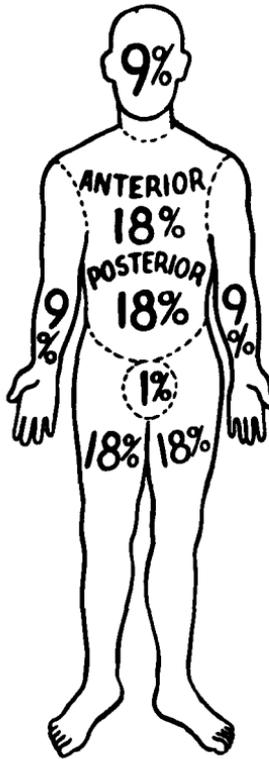


FIGURE 15.—Rule of nines, showing distribution of body surface area by anatomical part in the adult.

### DEPTH OF INJURY

The depth of thermal injury can be determined with certainty only by histologic examination. However, the clinical criteria in Table 1 will permit an initial, usually quite accurate differentiation between second- and third-degree burns. The total percentage of skin surface involved in second-degree and third-degree burns is the primary concern during resuscitation. Differentiation between second-degree and third-degree burns is more important later in the postburn course as related to the duration of hypermetabolism, the anticipated functional result, and the ultimate need for autograft closure of the burn wound.

TABLE 1.—*Diagnosis of depth of burns*

Criteria	Second-degree Burns	Third-degree Burns
Cause	Hot liquids, flashes of flame	Flame, electricity, chemicals
Color	Pink or mottled red	Dark brown or black charred, translucent with thrombosed superficial veins visible, pearly white $\pm$
Surface	Vesicles or weeping	Dry and inelastic
Pinprick	Painful	Anesthetic

Those areas of thermal injury that are waxy-white, soft and pliable, yet nonpainful formerly were regarded as full-thickness injuries, but in actuality are deep, partial-thickness burns; they frequently heal without the need for grafting if protected from invasive infection by topical chemotherapy. Charring with thermal injury of subcutaneous and deeper tissues is infrequent, but may occur in the unconscious victim, in individuals trapped by burning debris or in a burning vehicle, or in individuals with high-voltage electric injury. Injuries of less than partial thickness, that is, first-degree burns (erythema of intact epidermis), are important only so far as patient comfort and vasomotor lability are concerned and are, with few exceptions, treated symptomatically without need for resuscitation.

The depth of thermal injury after a thermonuclear explosion is dependent upon the intensity and duration of the thermal pulse, but burns also may be sustained from ignition of clothing or burning debris. These burns do not differ from burns of other etiology except for the associated effects of ionizing radiation, which decrease survival for a given size burn.

## PATHOPHYSIOLOGY

Thermal injury, regardless of the etiologic agent, results in cell death by coagulation necrosis. In areas of cell death and cell

damage, capillary permeability is increased with the loss of integrity of the vascular system and the escape of the nonformed blood elements. This is manifested clinically by edema, which forms most rapidly in the immediate **postburn** period and reaches a maximum in the second **postburn** day. Thereafter, as vascular integrity is restored and fluid resorption begins, edema slowly resolves. This increase in capillary permeability results in a decrease in blood volume and an increase in blood viscosity, causing an increase in peripheral resistance and a decrease in cardiac output. Fluid resuscitation is carried out in the immediate **postburn** period to minimize these changes by maintaining blood volume at a level adequate for organ perfusion.

### FIRST AID

The first consideration is removal of the source of thermal injury from the patient. Burning clothing should be extinguished and removed, and the patient should be removed from a **burning** vehicle or building. In electric injury, the patient should be removed from the point or points of contact, with the rescuing personnel taking care to avoid contact with the power source. Chemical agents should be washed immediately from the skin **surface** by copious water **lavage**. First aid should be reduced to a minimum, and nothing must be done that could prejudice subsequent treatment. All constricting articles, such as rings, bracelets, wristwatches, belts, and boots must be removed, but the patient is not undressed unless the injury has been caused by a chemical agent, in which case all contaminated clothing must be removed. The patient should be covered with a clean sheet and a blanket, if appropriate, to maintain body temperature and prevent gross contamination during transport to a treatment facility. If available, burn dressings can be used for such initial wound coverage.

Patency of the airway should be assured, hemorrhage should be controlled, and fractures should be splinted. If at all possible an intravenous pathway should be established in an unburned area and in an upper extremity vein if there are associated abdominal wounds. Resuscitation may be safely begun with electrolyte solution alone, and should be continued before and during movement to an installation where definitive medical care is available. An intravenous cannula is preferable in all situations.

since large volumes of fluid are required for patients with extensive burns, and patient restlessness, transportation, or edema may dislodge an intravenous needle. Patients with injuries from white phosphorus should have the burns dressed with saline-soaked dressings to prevent reignition of the phosphorus by contact with the air.

Pain is seldom a major problem in patients with severe burns, but patients with extensive partial-thickness burns may have considerable discomfort, which can be relieved by appropriate doses of morphine or meperidine administered intravenously. Subcutaneous or intramuscular injections of analgesics will not be mobilized during the period of edema formation and will be ineffective in pain control. A patient who has received multiple subcutaneous or intramuscular doses of an analgesic may later mobilize them simultaneously and develop severe respiratory depression, which must be treated promptly.

On the day of injury, after hemorrhage is controlled, ventilatory stability achieved, and urinary output established, one should promptly move the extensively burned patient to a definitive treatment facility. Intravenous fluid administration should be maintained throughout transportation and, if any question exists as to adequacy of the airway, a tracheostomy should be performed or, preferably, an endotracheal tube placed and secured.

#### INITIAL TREATMENT OF EXTENSIVE BURNS

At the definitive treatment facility, control of hemorrhage and airway adequacy again must be insured. Initial consideration of the burn patient includes a complete physical examination following removal of the patient's clothing. Once a secure intravenous pathway has been established, one then must estimate the resuscitation fluids to be given to the burned patient.

TABLE 2.—*Formula for estimating fluid requirements in burn patients***First 24 hours postburn:**

Adult: 2 ml lactated Ringer's solution/kg body weight/% burn

Child: 3 ml lactated Ringer's solution/kg body weight/% burn

**Second 24 hours postburn:****Adult and child:**

Colloid: Estimated deficit and replace with a plasma equivalent, e.g., albumin diluted to physiologic concentration in normal saline or fresh frozen plasma

(a) 30-50% burn: 0.3 ml/kg body weight/% burn

(b) 50-70% burn: 0.4 ml/kg body weight/% burn

(c) &gt;70% burn: 0.5 ml/kg body weight/% burn

5% Dextrose in water: Volume necessary to maintain urinary output.

Several formulas exist for calculation of the fluid requirement of the burn patient. They are based upon body weight and extent of the burn. Clinical success has been reported for each such formula and, in a civilian setting with unlimited amounts of the full spectrum of intravenous fluids available, the attending physician's preference can certainly dictate the resuscitation regimen employed. In a combat situation, logistical considerations speak strongly for simplicity of resuscitation using readily available fluids in a volume sufficient to prevent renal or other organ failure, yet avoid later complications of fluid overload. Extensive clinical and laboratory studies have demonstrated that: (1) in the first 24 hours postburn, colloid has no specific restorative effect on cardiac output beyond that of electrolyte-containing fluids and is retained within the vascular compartment to no greater extent than an equal volume of electrolyte-containing fluid, and (2) in the second 24 hours postburn, capillary integrity is largely restored so that fluid and salt loading can be minimized by using colloid-containing fluid to correct any persistent plasma volume deficit. These studies have led to a revision of the Brooke formula, simplifying the logistics of initial resuscitation (only electrolyte-containing fluid is administered in the first 24 hours postburn) and reducing fluid and salt loading (no electrolyte-free water is administered in the first 24 hours postburn and no electrolyte-

containing fluid is administered in the second 24 hour period postburn). The formula, which is detailed in Table 2, should be modified according to the individual patient's response in terms of urinary output, vital signs, and general condition. The fact that children have a greater cutaneous surface area per unit body mass and therefore form a relatively greater amount of edema per unit body surface burn necessitates that their initial electrolyte fluid resuscitation needs be estimated on the basis of 3 ml/Kg of body weight multiplied by the percentage of body surface burned. One should plan to administer one-half of the total fluid volume estimated for the first 24 hours postburn within the first 8 hours following injury, the time of most rapid edema formation. The actual rate of administration is adjusted according to the patient's response as noted below. If the casualty is not received immediately following burn injury, the first half of the resuscitation fluid should be administered in the time remaining prior to 8 hours postburn. The remaining half of the estimated fluid should be administered, ideally at a uniform rate, in the succeeding 16 hours of the first 24 hours postburn. Patients with massive burns (greater than 70% of the body surface) and those in whom initiation of resuscitation has been delayed may require considerably more than the estimated volume of resuscitation fluid. Such patients require frequent observation and examination, and one must not hesitate to increase the volume or infusion rate of resuscitation fluids, or to otherwise alter therapy to obtain the physiologic response desired. Even in these patients, the proposed formula should be used to plan fluid therapy, keeping in mind that it is safer to add fluid as necessary than to deal with the complications of excessive fluid administration. Only in this manner can treatment be properly supervised and individualized.

The electrolyte-containing solution should be lactated Ringer's, which contains a more physiologic concentration of the chloride ion, but isotonic saline may be employed if the former is not available. Even though red blood cell destruction occurs after thermal injury, whole blood is not administered as a portion of the resuscitation fluids, since loss of the plasma, due to increased capillary permeability and intravascular retention of the red cells, would further elevate the patient's hematocrit and adversely affect the rheological properties of the blood. The colloid solution administered during the second 24 hours postburn can be fresh frozen plasma or albumin, with each 25 gram bottle of that

material diluted with normal' saline for administration as a 5% solution.

Potassium supplements are not needed and may be deleterious during the first 48 hours, since the serum potassium is commonly elevated as a result of the destruction of red blood cells and other tissue Potassium, lost from injured cells, appears in the blood at a time when renal function may be depressed. From the third postburn day onward, potassium supplements should be added to the intravenous fluids if renal function is unimpaired. Average daily potassium requirements range from 60-200 meq per day.

From the third postburn day onward, an adequately resuscitated burn patient commonly has a normal and, in some instances, a supranormal plasma volume, so that further administration of salt- or colloid-containing fluids is usually unnecessary and should be carried out with great caution. In patients treated by the exposure technique, the burn wound acts essentially as a free-water surface with considerable evaporative losses (that is, 6-8 liters per day in patients with very extensive burns), following the third postburn day and until it is healed or grafted. Evaporative water losses can be estimated according to the formula: evaporative water loss in ml/hr = (25 + percent of body surface burned) x total body surface in square meters. This formula estimates evaporative water loss at the low end of the observed range, and replacement of the evaporative water loss should be guided by assessing the adequacy of hydration, which can be determined by careful monitoring of patient weight, serum osmolality, and serum sodium concentration. In patients treated with occlusive dressings, evaporative water loss is considerably less. Following resuscitation, salt-containing fluid need be given only for the treatment of symptomatic hyponatremia. Following elimination of the resuscitation-related salt and water load, salt-containing fluid should be administered in the amount needed to maintain a "normal" serum sodium concentration. Later in the postburn course, whole blood should be administered to maintain the hematocrit between 30-35%.

## URINARY OUTPUT

The most readily available clinical guide to the adequacy of resuscitation is the hourly urinary output, which should be

maintained between 30-50 ml in patients weighing more than 30 kilograms and 1 **ml/kg/hr** in patients weighing less than 30 kilograms. In patients who require fluid resuscitation, an indwelling urethral catheter should be placed and the hourly urine output should be measured and recorded. Except possibly in patients with electric injury, oliguria in the first 48 hours **postburn** is rarely caused by acute renal failure and is treated by increasing fluid administration rather than by decreasing fluid administration or **giving** a diuretic

Three categories of patients may require an osmotic diuretic: (1) those patients with significant electric injury in whom liberated hemochromogens increase the risk of acute renal failure, (2) those patients with associated crush or other injuries with extensive tissue death and large hemochromogen loads in the urine, and (3) those patients with large burns to whom one has given considerably more than the estimated fluid requirement but in whom oliguria persists. Osmotic diuretics, such as mannitol, will insure an adequate urinary output, but one must remember that this will occur at the expense of blood volume even in hypovolemic patients. Urinary output in patients who have received a diuretic is no longer a guide to the adequacy of resuscitation. Other diuretics, such as **furosemide** and ethacrynic acid, also have been used in burn patients.

## ENDOTRACHEAL INTUBATION

The indications for endotracheal intubation are essentially those that exist in any other surgical patient: namely, acute laryngeal or upper airway edema or obstruction, inability to handle secretions, and associated chest wall injury. Severe smoke inhalation with respiratory insufficiency is another indicator for **endotracheal** intubation. The presence of inhalation injury and the adequacy of the airway should be assessed by direct examination of the oropharynx and the upper airway using a fiberoptic laryngoscope or bronchoscope.

If the burn patient is to be evacuated and the adequacy of the airway is at all questionable, the caregiver should perform **endotracheal** intubation or **tracheostomy** before movement rather than risk the possibility of acute airway obstruction in transit. Three categories of patients are most apt to require endotracheal

intubation on the basis of the indications listed: (1) patients with severe head and neck burns, (2) patients with steam burns of the face, and (3) patients burned in a closed space who have inhaled smoke or other noxious products of incomplete combustion.

The severe chemical tracheobronchitis which results from inhalation injury may cause acute respiratory insufficiency. Such patients may have marked hypoxemia persisting for several weeks. Marked bronchospasm and frequent bouts of coughing are common and the patient may raise sputum containing carbonaceous material, confirming the diagnosis of inhalation injury. Conservative therapy with administration of humidified air or oxygen and nasotracheal aspiration, as indicated, is employed initially. The ability of the patient to clear the tracheobronchial tree and the quantity of endobronchial debris will determine whether bronchoscopy is necessary and the frequency with which it should be employed. Endotracheal intubation should be performed for the indications previously noted. Tracheostomy should be carried out only if prolonged mechanical ventilation is necessary or if the endobronchial toilet cannot be adequately performed through an endotracheal tube. Daily chest roentgenograms must be obtained of all patients with significant inhalation injury, with endobronchial cultures obtained if pneumonic infiltrates appear. Antibiotic treatment is guided by the results of the microbiology reports of those cultures. Mucolytic agents and bronchodilators may also be useful. Mechanical ventilatory assistance may be necessary in the treatment of those patients who have severe bronchospasm, profound hypoxemia, or significant hypercarbia.

Steroids in large doses are employed only in those patients with unrelenting bronchospasm, and such treatment should be terminated at the earliest possible time to minimize the increased risk of infection attendant upon their use.

## **ESCHAROTOMY**

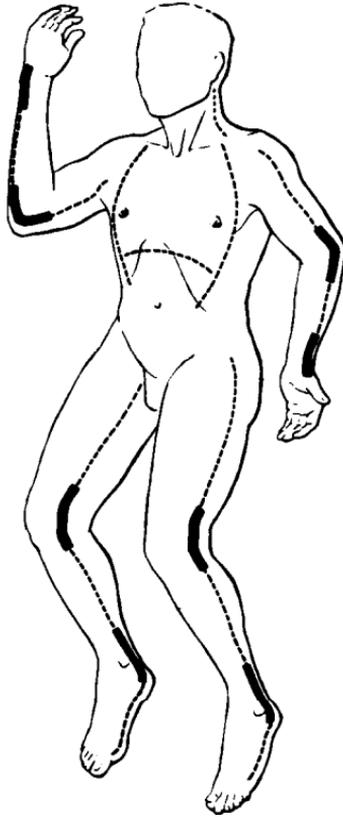
Circumferential full-thickness burns of the limbs may impair the circulation to distal and underlying unburned tissue. To prevent secondary ischemic necrosis of these tissues, an escharotomy may be necessary to relieve constriction caused by edema beneath the inelastic, unyielding eschar. The adequacy of the circulation of a burned limb must be assessed on a scheduled basis, e.g.,

hourly. The most reliable assessment of the circulation can be made by using an ultrasonic flowmeter to detect pulsatile flow in the distal palmar arch vessels in the upper limb and the pedal vessels in the lower limb. Absence of pulsatile flow or progressive diminution of flow on sequential flowmeter examinations is an indication for escharotomy. If a flowmeter is not available, the caretaker must depend upon the less reliable clinical signs of circulatory compromise. Swelling and coldness of the distal unburned parts are not indications for escharotomy, but cyanosis, impaired capillary refilling, and signs of neurologic dysfunction, such as relentless deep tissue pain and progressive paresthesia, are. Should evidence of vascular impairment be present, escharotomy should be promptly performed. The procedure can be carried out in the ward or emergency room without anesthesia, since it is performed through insensate full-thickness burn. An initial escharotomy incision is placed in the midlateral line of the involved extremity and, if this does not improve distal blood supply, a second escharotomy incision is made in the midmedial line in the longitudinal axis of the limb. The escharotomy incision should be carried throughout the entire length of full-thickness burn to ensure adequate release of vascular compression. The incision must cross involved joints, since in those areas the paucity of subcutaneous tissue permits ready compression of vessels and nerves. The escharotomy incision is carried through the eschar and the immediately subjacent thin connective tissue to permit expansion of the edematous subcutaneous tissue. When performed in this manner, blood loss from the escharotomy incision is not excessive and is readily controlled by either electrocoagulation or brief application of pressure.

Fasciotomy is rarely necessary for relief of vascular compromise in a limb with conventional thermal injury. Fasciotomy may be required in patients with high voltage electric injury, in other patients with burns involving deep tissues, or in patients with associated traumatic injuries (i.e., patients in whom edema is present below the investing fascia). Fasciotomy should be performed in the operating room using appropriate anesthesia.

Patients with circumferential truncal burns may also require escharotomies in the anterior axillary line to relieve restriction of chest wall movement by the eschar and permit a more satisfactory ventilatory exchange. This is particularly important in children with truncal burns who may be rapidly exhausted by the

increased respiratory effort required. These patients frequently will be restless, agitated, and hypoxemic before escharotomy and will show prompt clinical improvement as well as improved ventilatory exchange and blood oxygenation following escharotomy. An incision along the lower margin of the rib cage may be necessary in those patients with deep burns extending onto the upper abdominal wall (Figure 16).



**FIGURE 16.**—The dashed lines indicate the preferred sites for escharotomy incisions. The solid segments of the lines emphasize the importance of extending the incisions across involved joints.

### ADJUVANT TREATMENT

The burn patient who has been actively immunized against tetanus should be given a booster dose of tetanus toxoid. Those

patients who have not received prior active immunization should receive hyperimmune human antitetanus serum as well as an initial dose of tetanus toxoid, with active immunization continued at weekly intervals until complete.

Unless specifically contraindicated, penicillin is administered to all burn patients for the first 5 days postburn to prevent beta-hemolytic streptococcal burn wound infection. Thereafter, antibiotics are administered only on the specific indication of clinical infection supported by positive bacteriologic cultures.

As previously noted, restlessness and agitation frequently can be relieved by insuring adequate oxygenation. The need for analgesia is usually minimal except in those patients with extensive partial-thickness burns. Analgesia, when required in the first 3 days postburn, should be administered intravenously in appropriately small dosages.

Ileus is a common accompaniment of thermal injury involving 20% or more of the total body surface, and nasogastric intubation and drainage to prevent emesis and aspiration are critically important in these patients. It is also important to maintain nasogastric intubation in all patients who are to undergo air evacuation, not only in the early postburn period but also later, if evidence of gastrointestinal dysfunction exists.

## WOUND CARE

Attention is directed to the burn wound only after hemodynamic stability and the previously mentioned aspects of patient care have been accomplished. General anesthesia is not required for burn wound debridement; in fact, during this period of vascular instability and hypovolemia, it is ill-advised. Intravenous analgesia will suffice for pain control during such a procedure. The body hair is shaved from the area of thermal injury and well back from the margins. The burns are gently cleansed with a surgical soap solution, and nonviable epidermal remnants are debrided. Bullae are excised, since the proteinaceous fluid contained within them is an ideal culture medium for bacteria. After this initial debridement, the patient may be placed in bed, on surgically clean sheets. During the period of active wound exudation, placing bulky dressings beneath the burned parts to absorb the serious exudate has been found helpful. These dressings

should be changed as necessary and patients with circumferential burns should be turned on a scheduled basis to expose the burned areas on an alternating basis and to prevent maceration.

Patients with burns of the buttocks, perineum, and thigh do not require colostomy. The frequency of anal stricture is greatly increased by performance of such a procedure. Even when an abdominal operation is required to treat associated injuries, performing a colostomy is unwise solely for the treatment of buttock, perineal, or upper thigh burns. If a colostomy is indicated for other reasons, daily anal dilations are mandatory.

Fractures associated with thermal injury are best treated by skeletal traction or external fixation, to permit exposure of the burns and their treatment with topical chemotherapy. The application of a cast over an area of thermal injury promotes suppuration and enhances the possibility of the development of invasive burn wound infection. Nevertheless, plaster is acceptable over areas of burn in preparation for and during evacuation, if the cast is bivalved and removed promptly when the patient arrives at the definitive treatment installation.

## ELECTRIC INJURY

Although the pathologic change resulting from electric injury is coagulation necrosis, the extent and severity of such injury may initially be seriously underestimated. Limited areas of cutaneous necrosis may be evident at points of entry, exit, or arcing, yet be associated with extensive, subcutaneous, deep tissue involvement, leading to an inappropriate estimation of resuscitation fluid requirements. This "iceberg" effect also may necessitate the performance of fasciotomy rather than escharotomy to insure viability of distal unburned parts. The prophylactic use of an osmotic diuretic may be indicated because of extensive muscle necrosis with consequent liberation of hemochromogens. The presence of brawny, deep induration in a limb involved in electric injury, with signs of vascular impairment, indicates a need for fasciotomy. Approximately one-third of all patients with significant electric injury of the extremities will require amputation. This procedure should be delayed until resuscitation has been completed unless signs of systemic toxicity develop. Amputations in this situation as in any thermal injury should be consistent with conservative

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principles of limb salvage and should be carried out by disarticulation without opening a narrow cavity in the presence of the contaminated burn wound. Because of the difficulty of accurately distinguishing viable and nonviable tissue at the time of initial debridement, patients with high-voltage electric injury should be returned to the operating room 24 hours or, at the most, 48 hours following initial debridement. At the time of reoperation, further debridement is carried out as is necessary or, if no further necrotic tissue is identified, the wound may be loosely closed over tissue drains.

## CHEMICAL BURNS AND WHITE PHOSPHORUS INJURY

The depth and severity of chemical burns are related to both the concentration of the agent and the duration of contact with the tissues. These are the only burn injuries which require immediate care of the burn wound. The offending agent must be washed from the body surface as soon as possible. Full-thickness, third-degree injury of the skin caused by strong acids may result in tanning or bronzing of the skin which will be waxy, yet pliable to the touch, leading the unwary to underestimate the extent of burn.

Many antipersonnel weapons employed in modern warfare contain white phosphorus. Fragments of this metal, which ignite upon contact with the air, may be driven into the soft tissues; however, most of the cutaneous injury resulting from phosphorus burns is due to the ignition of clothing, and is treated as conventional thermal injury. First aid treatment of casualties with imbedded phosphorus particles consists of copious water lavage and removal of the identifiable particles, following which the involved areas are covered with a saline-soaked dressing and kept moistened until the patient reaches a definitive treatment installation. If transfer will require more than 12 hours, the involved areas should be covered by a liberal application of topical antimicrobial agent to prevent microbial proliferation and the reignition of retained phosphorus particles.

At the site of definitive treatment, the wounds containing imbedded phosphorus particles may be rinsed with a dilute (1%) freshly mixed solution of copper sulfate. This solution combines with the phosphorus on the surface of the particles to form a

blue-black cupric phosphide covering which both impedes further oxidation and facilitates the identification of retained particles. If sufficient copper is absorbed through the wound to cause intravascular hemolysis, acute renal failure may result. To avoid this potential complication, copper sulfate solution should never be applied as a wet dressing, and all wounds must be lavaged thoroughly with saline following a copper sulfate rinse to prevent absorption of excessive amounts of copper. As an alternative to the use of a copper sulfate rinse, a Woods lamp can be used in a darkened operating room, or the lights in the operating room may be turned off to identify retained phosphorescent particles during debridement. The extracted phosphorus particles must be immersed in water to avoid their ignition in the operating room. Inflammable anesthetic agents should not be used with these cases.

Combustion of white phosphorus results in the formation of phosphorous pentoxide, a severe pulmonary irritant. The ignition of phosphorus in a closed space may result in the development of concentrations of phosphorous pentoxide sufficient to cause acute inflammatory changes in the tracheobronchial tree. The effects of this gas can be minimized by placing a moist cloth over the nose and mouth to inactivate the gas and prevent endobronchial irritation. Hypocalcemia and hyperphosphatemia have been described as effects of white phosphorus injury and have been associated with electrocardiographic changes and sudden deaths. Hypocalcemia associated with cardiac arrhythmia should be corrected by the administration of calcium.

## VESICANT GASES

Patients with cutaneous injuries due to vesicant gases are treated as are patients with other chemical injuries by personnel appropriately protected from the gaseous agent. All contaminated clothing must be removed and all skin exposed to the agent immediately lavaged with copious amounts of water. Vesicles should be debrided while being lavaged during the cleansing procedure to prevent injury to contiguous areas by serous vesicle fluid containing the vesicant. Subsequent treatment of the cutaneous injury is as for any burn, with emphasis placed on prevention of infection by the use of topical chemotherapy. Inhalation injury can also be produced by vesicant gases, and the previously described

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endoscopic examination of the airway should be carried out in such patients to determine the need for tracheal intubation and mechanical ventilatory support.

### TOPICAL CHEMOTHERAPY

If the burn patient can be moved to a definitive treatment installation within 48-72 hours, no specific topical antimicrobial therapy need be employed in the field. However, if either the tactical or logistical situation is such that treatment must be continued at a relatively forward area, topical chemotherapy should be begun once the patient has become hemodynamically stable. There are three topical antimicrobial agents which are commonly employed for burn wound care in civilian practice. Each agent has specific advantages and limitations with which the clinician must be familiar to provide optimum wound care. Both mafenide acetate and silver sulfadiazine are available in the form of topical creams which are commonly applied directly to the burn wound twice a day and do not require the twice or thrice daily application of occlusive dressings, as does the 0.5% silver nitrate soak treatment regimen.

Sulfamylon burn cream is an 11.1% suspension of mafenide acetate in a water dispersible base. The active ingredient, mafenide acetate, is water soluble and diffuses freely in the eschar to establish an effective antibacterial concentration throughout the eschar and at the viable/nonviable tissue interface where bacteria characteristically proliferate prior to invasion. Because of this characteristic, Sulfamylon is the best agent for use if the patient to be treated has heavily contaminated burn wounds or is **received** several days **postburn** and a dense bacterial population already exists on and within the eschar. The side effects of Sulfamylon burn cream are: (1) hypersensitivity reactions (usually responsive to antihistamines) in **7%** of patients, (2) pain or discomfort of 20-30 minutes duration when applied to partial-thickness burns (seldom a cause for discontinuing Sulfamylon application), and (3) inhibition of carbonic anhydrase. The inhibition of carbonic anhydrase may produce both an early bicarbonate diuresis and an accentuation of **postburn** hyperventilation. The resulting reduction of serum bicarbonate levels renders such patients liable to a rapid shift from an alkalotic to an acidotic state, if pulmonary

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complications supervene, even **with** a measured  $p\text{CO}_2$  at levels ordinarily considered to be normal. If acidosis should develop during Sulfamylon therapy, the frequency of application of Sulfamylon burn cream should be **reduced** to once a day, or dosage omitted for a 24-48 hour period, with buffering employed as necessary and efforts made to improve pulmonary function.

Silver sulfadiazine burn cream is a 1% suspension of silver sulfadiazine in a water-miscible base. As a consequence of poor water solubility, the active agent shows only limited diffusion into the **eschar**. Silver sulfadiazine burn cream is most effective when applied to burn wounds immediately after thermal injury to prevent bacterial colonization of the burn wound surface as a prelude to intraeschar proliferation. This agent has the advantages of being painless when applied to the wound and being free from **acid-base** and electrolyte disturbances. The limitations of silver sulfadiazine burn cream include neutropenia, which usually relents when application is discontinued, hypersensitivity, which is rare; and ineffectiveness against certain strains of *Pseudomonas* organisms and virtually all strains of *Enterobacter cloacae*.

The characteristics of silver sulfadiazine burn cream recommend it for initial wound treatment at the first echelons of medical care, while the characteristics of Sulfamylon burn cream, especially its more efficient and broader spectrum of antimicrobial action, mandate that it be available for the care of patients with extensive burns at the definitive level of surgical care.

Either Sulfamylon R or silver sulfadiazine burn cream should be applied in a layer one-eighth inch thick to the entire burn wound with a sterile gloved hand immediately following initial debridement and wound care. Twelve hours later, to ensure continuous topical chemotherapy, a one-eighth inch coat of cream should be reapplied to those areas of the burn wound from which it has been abraded by the bed clothes. The topical cream should be gently cleansed once each day from all of the burn wound and the wound inspected by the attending physician. Daily **debridement** should be carried out to a point of bleeding or pain without the use of general anesthesia. Following the debridement, the wound is again covered by the topical cream.

If topical antimicrobial creams are not available, multilayered occlusive gauze dressings, saturated with a 0.5% solution of silver nitrate, can be used. These soaks are changed two or three times

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each day and moistened every two hours to prevent evaporation from raising the silver nitrate concentration to **cytotoxic** levels within the soaks. Transeschar losses of sodium, potassium, chloride, and calcium should be anticipated and appropriately replaced. Silver nitrate soak therapy, as in the case of silver sulfadiazine burn cream therapy, is best used for bacterial control in bum patients who are received immediately after injury before significant microbial proliferation has occurred. Silver nitrate is immediately precipitated upon contact with proteinaceous material, does not penetrate the **eschar**, and consequently is ineffective in the treatment of established bum wound infection.

### **BURN WOUND EXCISION**

Surgical excision of the burned tissue, commonly performed in civilian practice, has no place in the care of burn patients in the theater of operations. The extensive blood loss associated with a bum wound excision, up to 9% of circulating blood volume per each 5% of body surface area excised, would impose a prodigious and unnecessary need on the military blood supply system, and the surgical manpower expended on lengthy excisions would divert such personnel from the care of other casualties in whom surgical treatment could directly influence survival. The time required for skin graft maturation, permitting return to active duty, even precludes excision for patients with third-degree burns of limited extent in the theater of operations.

### **TRIAGE**

Triage is an important aspect of military burn care to ensure that available medical care resources are matched to the severity of bum injury and the number of burn casualties. In civilian practice, with optimum resources available, every bum patient receives emergency care. Thereafter, care at a facility with optimum resources, i.e., a burn unit or bum center, is recommended for adults with second-degree burns of more than 25% of the body surface, for all patients with third-degree bums of 10% or more of the total body surface, and for all patients with significant bums involving the hands, face, feet, and perineum. Similarly, all burn patients with significant inhalation injury, significant high-voltage

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electrical burns, and with associated fractures or other major trauma, should be cared for in a facility with special expertise. Those patients with moderate, uncomplicated burn injury (that is, those with second-degree burns of **15-25%** of the total body surface area and with third-degree burns of less than 10% of the total body surface area without the associated complications or associated injury, as noted above) should be cared for in a general hospital. Patients with less extensive uncomplicated burn injuries can commonly be cared for on an outpatient basis.

In the combat setting, the tactical situation, logistical limitations, or limited availability of health care personnel may necessitate reduction in the upper limits of these categories. In the best of circumstances, optimum treatment results in salvage of approximately 50% of patients whose burns involve **60-70%** of the total body surface. With limited resources, burn care resources should be applied to that group of patients in which greatest benefit will be realized, with less attention given to those with lesser burns or those with more extensive burns. In a situation with resource restrictions or large numbers of casualties, hospital care can be delayed for those patients with burns of 20% or less of the total body surface. Similarly, expectant care should be applied to those patients with burns which exceed 70% of the total body surface and the available care facilities and resources applied to those with burns of from 20-70 % of the total body surface. With even greater restriction of health care availability, the upper limit of the maximum treatment group should be reduced by **stepwise** decrements of 10% until the surgical workload matches available resources. Triage modifiers include significant coexisting inhalation injury and associated mechanical injury, each of which lowers the upper limit of the maximum treatment group by 10%. Conversely, burns of the hands, face, feet, and perineum, occurring in patients with lesser total body surface burns, will increase the medical care necessary for such patients.

## EVACUATION

The burn patient best tolerates movement by either ground or air in the early **postburn** period; that is, after hemodynamic and respiratory stabilization and before the development of septic

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complications which may make movement particularly hazardous. Patency of the airway must be insured throughout the evacuation procedure, and continued appropriate fluid administration via a secure intravenous pathway is essential. Nasogastric intubation with adequate gastric decompression is also necessary during patient movement in the early postburn period if any gastrointestinal dysfunction exists. Bulky dressings may be used effectively during evacuation.

It is essential that adequate documentation of the patient's premovement and in-flight course be maintained and accompany the patient so that continuity of medical care is ensured. Particularly important in this regard is an adequate record of administered fluids, urinary output, medications administered, and any other features of the patient's course that will require serial evaluation, such as neurological deficit. During evacuation, the seriously ill, extensively burned patient should be accompanied by trained surgical personnel familiar with the exigencies of patient movement during the early postburn course.

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## CHAPTER IV

# Cold Injury

### HISTORICAL ASPECTS

Although cold injury is seen only sporadically in the population in peacetime, it can be of paramount importance to an Army. Cold injury has played a major role in the outcome of a number of military operations throughout history. Larrey's description of the loss of over 250,000 soldiers of Napoleon's Army in Russia in 1812 identified cold as the major force in the defeat of this Grand Army. In the Crimean war (1852-1856), 309,000 French troops experienced 5,215 cases of frostbite, of which 1,178 were fatal. In just two nights, in Sevastopol, 2,800 cases of frostbite occurred, 900 of which were fatal. In World War I, the British incurred 115,000 trenchfoot or frostbite injuries. In one six-week period in one hospital in Rouen, there were 1,131 casualties with frostbite. In the Dardenelles, in the winter 1915-1916, there were 14,584 admissions for cold injury. U.S. Army cold injury losses in World War I amounted to 2,061 admissions, which translated to a total of 97,200 man-days lost. In just two months in World War II, December 1941 and January 1942, the German army suffered 100,000 cold injuries requiring 15,000 amputations. That was a major factor in their defeat on the eastern front. US. experience in World War II and Korea reveals that fully 10% of the wounded casualties (90,000 in World War II and 9,000 in Korea) were cold injuries. Recent British experience in the Falklands listed trenchfoot as the major medical problem in that conflict. Argentine amputations in the same conflict exceeded 200. Clearly, the impact that cold injury can have on military operations is a lesson that seems to have to be learned and relearned in each successive conflict.

Any force that is poorly fed, poorly clothed, or in retreat is more likely to sustain serious cold injury. Adding to the problems of

command prevention of cold injuries, the medical personnel who provide care are often unaware of the seriousness of the threat and have little or no experience in dealing with these types of injuries.

### CLASSIFICATION

Cold is the primary etiologic agent in producing these injuries, although wetness, duration of exposure, and other associated injuries may add to the severity or eventual outcome of a particular cold exposure. Long-term exposure in wetlands, even in tropical rice paddies, swamps, and jungles, with its prolonged cooling of the feet and constant wetness can produce an immersion-type injury. These injuries represent a continuum of insult making the definition between one type and another somewhat artificial. The spectrum of cold injury in order of increasing seriousness includes chilblain, trenchfoot, immersion foot, frostbite (including high-altitude frostbite), and systemic hypothermia.

All of these conditions represent progressive degrees of a fundamental pathologic process, which, irrespective of environmental and other modifying factors, are all related to the common factor of cold. Although the distinctions among the various types of cold injury are often artificial, particularly the distinction between trenchfoot and immersion foot, the following definitions are in fairly general use:

1. Chilblain, which frequently affects the hands as well as the feet, may result from exposure to air temperatures **from just** above freezing to as high as **60°F (16°C)**; is more likely to occur in dry, cold, windy air; but can also be associated with high humidity. It is not of major clinical significance in military operations.

2. Immersion foot implies an injury caused by exposure, usually in excess of 12 hours, to water at a temperature of about **50°F (10°C)**. This injury is common in wetjungles and in exposed life rafts.

3. Trenchfoot, which may also occur in the hands, results from prolonged exposure to cold at temperatures ranging from just above freezing to **50°F (10°C)**, often in a damp environment, and usually in connection with immobilization and dependency of the extremities. The blunt trauma of walking on wet feet hastens this injury.

4. Frostbite implies the crystallization of tissue fluids in the skin

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or subcutaneous tissues after exposure to temperatures of **32°F (0°C)** or lower. Depending upon the ambient temperature and wind velocity, the exposure necessary to produce frostbite varies from a few minutes to several hours. Frostbite may occur at various altitudes. Special attention has been given to high-altitude frostbite. The ambient temperature decreases approximately **35°F (2°C)** for every 1,000 feet of increase in altitude. The temperature becomes stable at about **67°F (-55°C)** at an altitude of 35,000 feet or higher, and exposure to these very low temperatures may instantaneously result in severe injuries to exposed parts of the body.

5. Systemic Hypothermia is a condition associated with a drop of the core temperature below **94°F (34.4°C)**. This life-threatening, non-freezing cold injury is usually the result of either long-term exposure to cold air or immersion in cold water. It should be noted that freezing temperatures are not necessary to produce hypothermia, because wind, rain, and cool temperatures increase body heat loss significantly.

## PATHOGENESIS

Trenchfoot, frostbite, immersion foot, and hypothermia are the cold injuries of greatest military significance. It is believed that frostbite will continue to be the cold injury of major importance, but the use of rubberized, insulated footwear and specific training techniques and procedures will limit the trenchfoot injuries in future combat settings. It is noted that the introduction of the insulated vapor-barrier boot to U.S. forces in Korea almost eliminated cold injury as a reason for hospital admission. This insulated vapor-barrier boot revolutionized footwear for combat soldiers and played the key role in preventing of cold injury of the feet. The type of cold injury incurred is dependent upon the exposure temperature, the duration of exposure, and other environmental factors, such as wind and water, which intensify the effect of the temperature. On exposure to cold, there is an initial peripheral vasoconstriction in an attempt to conserve core heat. This vasoconstrictive episode, which is of short duration, is overcome by a physiologic protective mechanism termed cold-induced vasodilation (CIVD). CIVD intervenes to cause arteriovenous shunting to the skin. This allows relatively large volumes of blood to flow through cold extremities. Repeated cold exposures are said to improve this CIVD response, but it may be suppressed or

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absent when the individual is chilled, frightened, exhausted, or malnourished. This mechanism appears to be blunted in blacks and perhaps in other races.

Trenchfoot and immersion foot are essentially the same injury, the major differences being the temperatures involved and the duration of exposure. The colder it is, the shorter the duration necessary to produce trenchfoot, whereas the longer the duration and the warmer the temperature, the more likely one is to develop an immersion foot injury. The average duration of exposure in trenchfoot is three days, but the exposure may range from a few hours to many days, with individual susceptibility apparently playing a considerable role. The average duration of exposure in frostbite is ten hours, but this varies with ambient temperature, moisture, clothing, activity, and other factors which will be discussed below.

### PATHOLOGIC PROCESS

Although a number of physiologic changes induced by cold may explain tissue loss, it is doubtful that they all play a significant part in clinical cold injury. Intracellular molecular changes due to hyperosmolarity, direct metabolic impairment secondary to the cold, and cellular structural damage from the mechanical effect of ice crystals seem far less important than impairment of nutritional blood flow as a final determinant of tissue injury after thawing. Vascular stasis following thaw from freezing injury has been well documented. Clinical and experimental data indicate the importance of capillary blood flow as the determinant of reversibility in tissue freezing.

Alterations in capillary permeability are evident from experimental data and, clinically, from the edema and bleb formation that occur soon after thawing. Endothelial disruption may be responsible for the progressive capillary stasis, plugging, and thrombosis that eventually occur.

### EPIDEMIOLOGY FACTORS

The military community responds to cold trauma according to accepted epidemiologic principles. The specific causative agent is cold. Moisture is closely related because it speeds the loss of body heat, although it alone cannot cause cold injury. Cold **pro-**

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**duces** injury by increasing the rate of body-heat loss. This rate is determined not only by the ambient temperature, but also by other factors such as moisture and wind. Moisture increases the rate of heat loss by conduction and evaporation, wind by convection.

A variety of environmental and host factors combines in the total causation of cold injury and influences the incidence, prevalence, type, and severity of the injury, though these influences vary from situation to situation. The most important **environmental** factors in cold injury are weather, clothing, and type of combat action.

Weather is a predominant influence in the causation of cold injury. Temperature, humidity, precipitation, and wind modify the rate of loss of body heat. Low temperatures and low relative humidity favor the development of trenchfoot. Wind velocity and low temperatures act synergistically, expressed as chill factor, to accelerate the loss of body heat under conditions of both wet and cold.

The type of combat action is apparently the most important environmental factor. Units in reserve or in rest areas have few cases. Units on holding missions or on static defense, in which exposure is greater, show a moderate increase in incidence. Factors which modify the incidence in relation to the rate of combat action include immobility under fire; prolonged exposure; lack of opportunity to warm the body, change clothing, or carry out measures of personal hygiene; fatigue; fear; and state of nutrition. In warfare, in which exposure under conditions of stress may be prolonged, adequate clothing becomes essential to welfare and survival.

### HOST FACTORS

The following are host factors that may or may not influence the development of cold injury:

1. Age There is no convincing evidence that age is a significant epidemiologic factor in cold injury among combat troops.

2. Smoking. There is very clear evidence that the **vasoconstrictor** action of nicotine causes increased cooling of the extremities and an increased likelihood of frostbite. A significant number of severe injuries in military populations occur in heavy tobacco users.

3. Previous Cold Injury. Individuals with previous cold injuries are at a higher-than-normal risk of subsequent cold injury. The

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fact that such repetitive injury does not usually occur at the same site suggests that this relates to the individual's lower resistance to cold rather than as a result of the previous injury.

4. Branch of Service Trenchfoot, immersion foot, and frostbite have a high selectivity for frontline riflemen, especially for riflemen of lower ranks. In World War II, approximately 90% of all casualties from cold occurred in riflemen.

5. Fatigue Both physical and mental weariness contribute to apathy which leads to neglect of all acts except those vital to survival. Fatigue is most evident in troops who are not rotated and must remain exposed and in combat for prolonged periods of time. Three days of being cold and wet appears to be a prudent timeframe within which to consider rotation of troops.

6. Racial Susceptibility. In all studies from World War II, Korea, and recent experiences in Alaska, blacks had four to six times the incidence of cold injury of their white counterparts, matched for geographic origin, training, and education. This increased susceptibility is related to two factors: (a) differences in anatomic configuration, and (b) differences in physiologic response to cold. Because long, thin fingers and toes cool more rapidly than short, fat ones, blacks' hands tend to cool faster than those of whites. However, more importantly, once cold, blacks stay cold longer because of a less potent CIVD response to their extremities. This does not say, however, that blacks themselves must be more vigilant in cold exposure and must take measures sooner to protect themselves from cold injury. Place of origin has a significant role in cold injury susceptibility. Individuals raised in northern-tier states (i.e., cold climates) have a more protective CIVD response. This response also improves in blacks from northern climates. This is not only a physiologic improvement in response to cold but a behavioral response as well. Knowing what clothes to wear, knowing when one's extremities are too cold, not being frightened of the cold, and knowing how to deal with cold extremities all add up to make cold-experienced individuals less likely to have cold injuries. Individuals with labile vasomotor conditions, such as Raynaud's, are also susceptible to cold injury.

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7. Psychological Factors. Cold injury tends to occur in passive, negative individuals. Such persons show less muscular activity in situations in which activity is unrestricted and are careless about precautionary measures when cold injury is a threat. Fear also may increase the incidence of cold injury by reducing the spontaneous rewarming known as CIVD.

8. Other Injuries. Concomitant injuries that result in a reduction of circulating volume or a localized reduction in blood flow predispose the individual to cold injury. In addition, immobilization associated with a concurrent injury increases the risk of frostbite in cold environments if adequate additional insulating protection is not provided. Poor hydration and hypovolemia decrease perfusion of the extremities.

9. Drugs and Medication. Any drug modifying autonomic nervous system responses, altering sensation, or modifying judgment can have disastrous effects on an individual's performance and survival in the cold. These factors must be impressed upon medical officers involved in the care of troops in cold environments and must be impressed upon individual unit commanders and their men. In the civilian community, alcohol *use* is the single most common factor associated with hypothermia.

#### DISCIPLINE, TRAINING, AND EXPERIENCE

Cold injury is preventable. Well-trained, fit, disciplined soldiers can be protected from cold injury even in adverse, pinned-down positions if they are knowledgeable concerning the hazards of cold exposure and informed regarding the importance of personal hygiene, care of the feet, exercise, and the rational use of clothing. Such discipline and training are a command and not a medical responsibility and reinforcement of these principles throughout the field operations is essential to the goal of protection from cold injury. Although cold injury is preventable, commanders may be faced with circumstances that are likely to lead to large numbers of casualties, and a decision may have to be made to accept a certain number of cold injuries to win the battle. The need for a major offensive in a cold, wet environment, or a retreat when faced

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by an overwhelming foe, may prompt a commander to accept cold injuries to change the tide of battle. The highest levels of command must be aware of the medical implications of such decisions. The combination of fit, disciplined soldiers, trained for cold weather operations, plus the provision of dry clothing, adequate food, water, and shelter will minimize the number of cold injuries.

### CLINICAL MANIFESTATIONS

Patients generally describe initial feelings of cold discomfort in their extremities, followed by varying periods of pain and mild discomfort along with a cyclic, dull ache. These symptoms subside into a period of anesthesia. From there, cold injury progresses in a painless fashion. Patients often describe a sensation of walking on a wooden limb. Because of the anesthetic nature of cold injury, patients often say they were unaware that they were developing an injury. The hypothermia victim retreats inward psychologically; has dulled senses, a stumbling gait, muscle incoordination, and slurred speech; and is universally unaware of the insidious decrement in his capability.

In a cold, wet environment, trenchfoot often appears. Anesthesia of the limb in trenchfoot injury comes on rapidly. Pain which does not respond to analgesia limits the deployment of soldiers with normal-appearing extremities. Most patients are unaware of or do not care about the potential severity of their injury. The first physical manifestation of frostbite injury is reddening of the skin, which later becomes pale, waxy white, and hard. Lack of mobility of skin over joints is a common finding. In hypothermia, shivering is a clear indication of loss of body temperature. Shivering varies with age, physical condition, degree of hypothermia, and amount of ingested drugs. Shivering can significantly limit an individual's performance of specific military tasks, including sighting targets, reading maps, and manipulating small dials and radios. It is a form of involuntary exercise that produces heat. When shivering stops, the patient is at the mercy of the environment. CNS involvement appears to be the most common outward manifestation of hypothermia. Decreased dexterity and coordination, speech and memory impairment, and the eventual loss of consciousness indicate progressive loss of **neurologic** function. Dysarthria is a specific early indication of hypothermia and is often one of the first recognizable signs of the

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loss of deep body temperature.

Judgment of the degree of frostbite has historically involved a retrospective grading system involving four categories. It is more useful and realistic, however, to determine two major categories: superficial and deep. Because frostbite is a continuum of events, the differentiation between first, second, third, and fourth degrees is often clouded and may take some days or weeks to become completely obvious.

In first-degree injuries, erythema and edema, along with transient tingling or burning, are early manifestations. The skin becomes mottled blue/grey and red, hot, and dry. Swelling begins within two or three hours and persists for ten days or more, depending upon the seriousness of the injury. Desquamation of the superficial epithelium begins in 5-10 days and may continue for as long as a month, but no deep tissue is lost. Parathesias, aching, and necrosis of the pressure points of the foot are common sequelae. Increased sensitivity to cold and hyperhydrosis may appear, especially with repeated first-degree injuries. It should be noted that it is difficult to differentiate first-degree frostbite from abrasion produced by the insulated vapor barrier boot. Medical personnel must be cognizant of the difference as both injuries occur in the same clinical setting.

Second-degree cold injury starts as does first-degree, but progresses to blister formation, anesthesia, and deep color change. Edema may form, but it disappears within days. Vesicles appear within 12-24 hours. They generally appear on the dorsum of the extremities, and when these vesicles dry they form an eschar. Blisters are a good clinical sign as long as they are filled with clear fluid. If the fluid is hemorrhagic, they are not a good sign. As these vesicles dry, they sluff cleanly with pink granulation tissue beneath or they form black eschars. Throbbing and aching pain occurs 3-10 days after this injury. Hyperhydrosis is apparent at the second or third weeks. Early rupture of the blisters with subsequent infection often occurs in second-degree cold injury. This infection significantly increases the severity of the frostbite injury.

Third-degree injury involves full skin thickness and extends into the subcutaneous tissue. Vesicles are smaller and may be hemorrhagic. Generalized edema of the extremity may occur, but it usually abates within 5-6 days. Subfacial pressure increases and compartment syndromes are common in third- and fourth-degree cold injuries. If pressure rises significantly with loss of distal blood flow,

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faciotomy along with vasodilators is indicated for therapy. The skin forms a black, hard, dry **eschar**, usually thicker and more intense than that of the second-degree injury. When it finally demarcates, sloughing with some ulceration occurs if there is no complicating infection. The average healing time is 68 days. Patients often complain of burning, aching throbbing, or shooting pains beginning on the fifth day and usually lasting through four or five weeks. Hyperhydrosis and cyanosis appear later and extreme cold sensitivity is a common post injury **sequela**.

In fourth-degree injury, there is destruction of the entire thickness of the part, including bone, resulting in extensive loss of tissue. After rewarming, tissue is cyanotic and insensitive, and blister formation, if present, is hemorrhagic. Severe pain on rewarming, along with a deep cyanotic appearance, regularly occurs. In rapidly-frozen extremities or the freeze-thaw-refreeze injury, dry gangrene progresses quickly with mummification. With slower freeze, there is some early swelling and deep pain, and demarcation takes much longer to occur. The line of demarcation becomes obvious at **20-36** days and extends into the bone in 60 or more days.

### MANAGEMENT

A major deterrent to evaluation of therapy has been the inability to predict the outcome in any given cold injury early in the **post-thaw** period. Because of this, nuances of clinical management have been very difficult to evaluate. Since the extent of injury to the tissue is related to temperature and the duration of exposure, rapid rewarming is of primary importance. Other therapeutic programs, including anticoagulant therapy, administration of low molecular weight dextran or similar agents, or surgical or pharmacologic sympathectomy, while theoretically sound and supported in some instances by experimental data, have not had controlled clinical trials sufficient to encourage their general use.

In the light of most clinical experience, it **should** be emphasized that meddlesome manipulations, rubbing, application of **unguents**, or exposure to excessive temperatures should be guarded against carefully. As soon as cold injury is recognized, every effort should be made to avoid compounding the effects of cold with physical injury.

In military operations, the treatment of cold injuries is influenced by (1) the tactical situation, (2) the availability of evacuation

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to a fixed facility, and (3) the fact that most cold injuries are encountered in large numbers, during periods of intense combat, at the same time that many other wounded casualties are generated. Highly individualized treatment under these circumstances may be impossible. Examination and treatment of more life-endangering wounds must take precedence over this injury (lives versus limbs).

As a practical matter, any specific therapy designed to modify the physiologic changes in cold injuries must be instituted very early after thawing. Since, in many cases, the injury is not seen until some time after thawing, contemplation of therapy is purely academic and the major emphasis must be on protection from further injury, avoidance of premature surgery that might sacrifice otherwise viable tissue, early identification and control of infections, attention to maintenance of extremity function through early physiotherapy, and generalized nutritional support.

#### FIRST AID

The emergency treatment of cold injury is as follows:

1. All casualties with involvement of the lower extremities should be treated as litter cases if feasible.

2. Carefully assess concomitant injury or complicating systemic problems.

3. All constricting items of clothing, such as boots, gloves, and socks, should be removed, but only when adequate protection from further cold exposure is available. Boots and clothing frozen on the body should be thawed by immersion in warm water before removal. Vigorous manipulation of frozen parts or attempts at range of motion or massage should be avoided. If the hands are affected, rings should be removed from the fingers early after presentation.

4. If the injured parts are still frozen when first seen, they should be rewarmed rapidly by immersion in water at  $100^{\circ}$  to  $104^{\circ}\text{F}$  ( $37.5^{\circ}$  to  $40^{\circ}\text{C}$ ) with added antiseptic soap, such as **pHisoHex**, and with agitation of the bath water to hasten the warming. A whirlpool apparatus is most satisfactory for this.

5. General body warmth must be maintained. Sleep and rest should be encouraged.

6. A booster dose of tetanus toxoid should be given to those previously immunized. No evidence exists that prophylactic use

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of antibiotics is valuable either in promoting healing or in preventing superficial or deep infection. In fact, the use of prophylactic antibiotics may result in the emergence of a resistant strain of organisms.

7. Large vesicles or bullae should be protected and kept intact if possible. Once ruptured, it is usually desirable to debride the vesicle. Ointment dressings have no place in the usual management of cold injury. Protective dry dressings are desirable during transportation, and sterile cotton should be used between the toes to prevent maceration.

8. Smoking is prohibited.

### LATER MANAGEMENT

When the casualty reaches a definitive care facility, the following treatment should be employed:

1. Continued diligence to avoid injury of already compromised tissue should be maintained. In general, for lower extremity injuries, this is accomplished by keeping the patient at bed rest, with the part elevated on surgically clean sheets under a foot cradle and with sterile pledgets of cotton separating the toes. Bearing-weight on injured feet should not be allowed until mature epithelial tissue has developed over the affected areas. In upper extremity injuries, elevation is also desirable on sterile towels, with special care to avoid injury to bullae.

2. In an effort to reduce superficial bacterial contamination, the affected part is treated by whirlpool bath at 98.6°F (37°C), with povidone iodine or hexachlorophine added, on a twice-daily basis, encouraging active motion on the part of the patient during the whirlpool treatment. Whirlpool baths assist in superficial debridement and make active range of motion exercises more tolerable to the patient and less traumatic to the tissues.

3. Analgesics may be required in the early post-thaw days, but a continued requirement for analgesics in uncomplicated injuries is uncommon.

4. The patient should be encouraged to take a nutritious diet with adequate fluids to maintain hydration.

5. Patients should be placed on surgically clean sheets and all lesions should be exposed to the air at the normal room temperature.

6. Superficial debridement of ruptured blebs should be per-

formed, and suppurative eschairs and partially detached nails should be removed. Close attention should be paid to circumferential eschars or eschars where vascular compromise could be a problem. Such eschars at least should be bivalved, although complete debridement is occasionally necessary. Early amputation has no place in the management of cold injury. Surgical intervention should be deferred until a distinct line of demarcation has developed. There is usually healthy granulation tissue under an **eschar** at the line of demarcation. Delay of surgical procedures, especially in upper extremity injuries, will enhance the potential for a functional result. Rarely, generalized sepsis from large areas of necrotic and infected tissue will necessitate amputation. Skin grafting, while not a function of forward facilities, is occasionally indicated to protect denuded areas over vital structures.

7. Active physiotherapy should be instituted during daily whirlpool as soon as possible.

8. Newly epithelialized areas are susceptible to minor trauma, as in walking, and are especially sensitive to cold. Therefore, continued protection must be offered until normal keratinization has occurred. Subsequently, special skin care may be required to deal with residual hyperhydrotic states.

## PROPHYLAXIS

The successful prevention and control of cold injuries depend, first of all, upon vigorous command interest, the provision of adequate clothing, and a number of individual and group measures. These measures include:

1. A thorough appreciation and comprehension by command, staff, technical personnel, and all combat components regarding the potential losses that may occur from cold injury, both in winter combat and in other circumstances in which cold injury has been known to occur.

2. There should be full command support, by echelon, of a comprehensive and practical cold injury prevention and control program. It should be emphasized again that this is a command, not a medical, responsibility.

3. Indoctrination of all personnel in the prevention of cold injuries individually and by units.

4. The provision of adequate supplies of clothing and **footgear** and their correct utilization to avoid exposure to cold. The **pro-**

gram of supply must provide adequate dry clothing for the daily needs of the soldier who is farthest forward in combat; it must also provide for the correct fitting of clothing and boots. All articles of clothing must be sized and fitted to avoid constriction of the extremities and tightness over the back, buttocks, and thighs.

Clothing for cold weather, based on the layering principle, is now designed as an assembly for protection of the head, torso, and extremities. The clothing is worn in loose layers, with air spaces between the layers, under an outer wind-resistant and water-resistant garment. Body heat is thus conserved. The garment is flexible, and inner layers can be removed for comfort and efficiency in higher ambient temperatures or during strenuous physical exertion. Prevention of loss of body heat by the proper protection of the body is as important as the efficient use of appropriate dry footwear and warm dry gloves. Finally, the most efficient clothing is of no value unless a high level of individual and unit clothing discipline are maintained through training.

5. Special protection for certain groups who may be especially susceptible to cold injury, together with the regular rotation of all troops. It should be remembered that casualties with exposed wounds and injuries are particularly liable to cold injury because blood and transudate from their wounds will freeze from the clothing inward.

6. Effective policies of sorting in forward areas, with provision for early evacuation and treatment of casualties actually suffering from cold trauma.

7. The identification of factors responsible for cold injury in special situations, which is a command responsibility. Significant numbers of cases occur as a result of barehanded contact with cold metal or gasoline; as a result of rapid deployment of troops seated in unheated vehicles, without interruptions for short rewarming marches every few hours; as a result of airdrops of troops into cold areas without adequate protective equipment and training; or as a result of several hours' confinement of artic-equipped airborne troops in heated aircraft, followed by a drop into a subzero environment after their insulating clothing has been saturated with perspiration. Only by the evaluation of these factors can the specific measures necessary in particular units or groups be put into effect.

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## HYPOTHERMIA

Hypothermia victims, depending on their core temperatures and the durations of their exposure, present with different degrees of physiologic depression. Cold suppresses metabolic function and decreases oxygen demand, thereby enhancing survival. Recognition of this survival potential is critical to successful resuscitation. Everyone involved in the treatment and evacuation of these casualties must be cognizant of the phrase "No one is cold and dead, only warm and dead." Failure to respond to rewarming is the only criterion for death in hypothermia.

Two major defenses against hypothermia are peripheral vasoconstriction and shivering. Peripheral vasoconstriction reduces cutaneous blood flow, which conserves core heat by decreasing both radiant and convective heat losses to the environment. Shivering is an involuntary muscle activity that increased heat production. The end result of peripheral vasoconstriction, which decreases circulating volume, is cold diuresis. Shivering produces significant metabolite production, including lactic acid. The longer one is exposed to cold, the greater will be one's metabolic derangement. Dry land hypothermics shiver violently and diurese for long periods of time. This experience diuresis results in more severe metabolic abnormalities. On the other hand, water-immersion hypothermics who cool rapidly do not shiver quite as long and often present with a normal electrolyte and pH profile. As cells drop below 30°C in an acid medium the sodium pump fails and potassium leaks out of cells into the general circulation. As the core temperature drops in the presence of acidosis and hyperkalemia, severe cardiac arrhythmias occur. Hemorrhage from wounds in a cold environment leads to rapid hypothermia.

Hypothermics have decreased cerebral metabolic activity. They show a stumbling gait, incoordination, slurred speech, and a psychologically inward retreat. Their senses are dull; they are apathetic, drowsy, and more exhausted than their activity would warrant. This state progresses to unconsciousness. The disorientation, confusion, irrational judgment, and poor decision-making ability pose a significant threat in leadership roles since the small-unit leader is usually exposed to the same physical and cold stresses as his troops. The leader may, in fact, not be able to recognize the signs and symptoms of hypothermia in those he leads if he is experiencing the same symptoms himself. This scenario can result in disaster.

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FIELD MANAGEMENT OF **HYPOTHERMICS**

Individuals must be stripped of their wet clothing insulated; given warm, sweet drinks; and encouraged to do large-muscle activities that will warm them up. Warming the core with external heat is an extremely difficult physiologic problem. Conscious individuals will shiver and initiate rewarming. If other muscle activity is added, they will warm up quickly. Replacement of fluids is essential to improve peripheral **circulation**, cutaneous perfusion, and cardiac output. Comatose individuals must be handled carefully, as rough handling can produce ventricular fibrillation arrest. The airway should be patent. Wet clothes should be carefully stripped. They should be well covered and insulated. They should then be transported as rapidly as possible to definitive medical care. Positive pressure ventilation is advised but chest compression is not. Such compressions may convert sinus bradycardia to ventricular fibrillation.

Field rewarming procedures for the comatose individual are time consuming. If possible, it is better to move the casualty to a nearby medical facility. A heated, humidified oxygen rewarming device, if available, may be effective, but is certainly not a major method of heat input for the comatose hypothermic victim. Management throughout the evacuation chain involves improving cardiac output, decreasing blood viscosity, adding heat to the core, improving acid-base balance, and the hyperkalemia. Treatment of imbalances in these parameters depends on the level of sophistication at each treatment site. Hospital management should include active core rewarming utilizing peritoneal dialysis, arterio-venous shunts, or peripheral rewarming involving torso water immersion. Rewarming blankets are slow but may be the only rewarming devices available. Volume replacement is essential to decrease viscosity and increase cardiac output. Low central venous pressures are advisable early and are increased slowly as there is an indication of the ability to hold fluid in the vascular space. Lactate-free and potassium-free fluids are advisable, as lactate conversion to pyruvate by the liver does not occur below **32°C** and hyperkalemia probably already exists. Hyperkalemia is improved by fluid replacement and glucose and insulin infusions. Sodium bicarbonate is indicated early to begin correction of acidosis. However, overzealous correction is ill advised. The patient should be kept mildly acidotic throughout the treatment

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process. Improved ventilation during the resuscitation can improve **pH** significantly. Antiarrhythmic drugs are contraindicated. Excessive early manipulation can result in cardiac arrest. This complication is managed by continuing the rewarming process, along with half-rate, closed-chest cardiac massage until the temperature reaches **31° or 32°C**, at which point electrical conversion is more likely to be successful. The patient with severe acidosis and hyperkalemia should not be rewarmed past **30°C**. Post rewarming complications include pneumonia, pancreatitis, intravascular thromboses, gastric erosions, and acute tubular necrosis. Pneumonitis is by far the most common problem. It is managed by pulmonary toilet and appropriate antibiotics.

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