verting colostomies are not required either to achieve wound closure or to prevent wound infection. Diversion of the fecal stream usually results in significant anal stenosis, which then requires surgical correction.

***Skin* Grafts.** After a burn wound has been excised, optimal closure is accomplished by the use of cutaneous autografts. However, this is possible only when *(a)* the wound bed is unequivocally viable and *(b)* available donor sites exist. In extensively burned patients, the size of the burn wound often exceeds that of the available donor sites.

Meshed autograft skin is the most commonly used permanent covering after burn-wound excision. The **most common expansion ratios are 1.5:1, 2:1, and 3:1,** although up to ninefold expansion is possible (Figures 11-9 and 11-10). Because so long a time is required for the interstices of either six- or ninefold expanded skin grafts to re-epithelize, they are seldom employed. To prevent desiccation of the tissues underlying the open interstices of meshed cutaneous autografts, occlusive dressings soaked in either 0.5% silver nitrate solution or *5%* mafenide acetate solution are applied until epithelial closure is complete. For widely expanded meshed autograft skin or meshed autograft skin over fat, overlays of cutaneous allograft, xenograft, or Biobrane have proven successful in both increasing **graft take and decreasing the time to interstitial clo**sure. In patients with smaller burns involving 30% TBSAB, covering the functional and cosmetically important areas such as the face, feet, neck, and ears may be accomplished with sheet grafts.

The care of donor sites is critical. In extensively burned patients, these areas may need to be reharvested as soon as possible. Donor-site care should maximize re-epithelization and minimize trauma in an environment that promotes epithelial growth. Fine-mesh gauze remains the simplest, most inexpensive donor-site dressing, and when re-epithelization has occurred beneath, the gauze is easily peeled from the wound. Using a synthetic skin substitute such as Duoderm as a donor-site dressing has decreased the healing time of donor sites,[25] but such dressings are expensive, more difficult *to* use, and will in all likelihood be unavailable in a wartime setting.

When donor sites are limited, the burn wound is frequently closed temporarily, using material other **than own skin. The** goals of **temporary** wound coverage are to decrease the physiological impact of the open wound and to prevent bacterial colonization. Various biological dressings and skin substitutes now exist for achieving this goal. Cutaneous allograft, harvested from cadavers, is the most effective biological dressing and is the standard to which all other biological dressings and skin substitutes must be compared. Frozen cutaneous allograft and porcine cutaneous xenograft are the two most readily available skin substitutes, but they are *(a)* less adherent to the wound bed than fresh allograft, *(b)* less able to control the bacterial population of the underlying wound, and *(c)* usually do not become well-vascularized from the underlying wound bed. Synthetic skin substitutes exist but have met with only limited success. An effective synthetic skin substitute should *(a)* be compatible with the patient's own tissue, *(b)* have no antigenicity or toxicity, *(c)* have water va**por permeability similar to that** of **skin, *(d)* be imper**meable to microorganisms, *(e)* adhere to the wound, *(f)* be readily vascularized, and *(g)* have an indefinite shelf life. The available skin substitutes need to be modified to increase their clinical usefulness by enhancing both their resistance to infection and their ability to accelerate the formation of either neodermis or granulation tissue.

The use of culture-derived epidermal sheets has

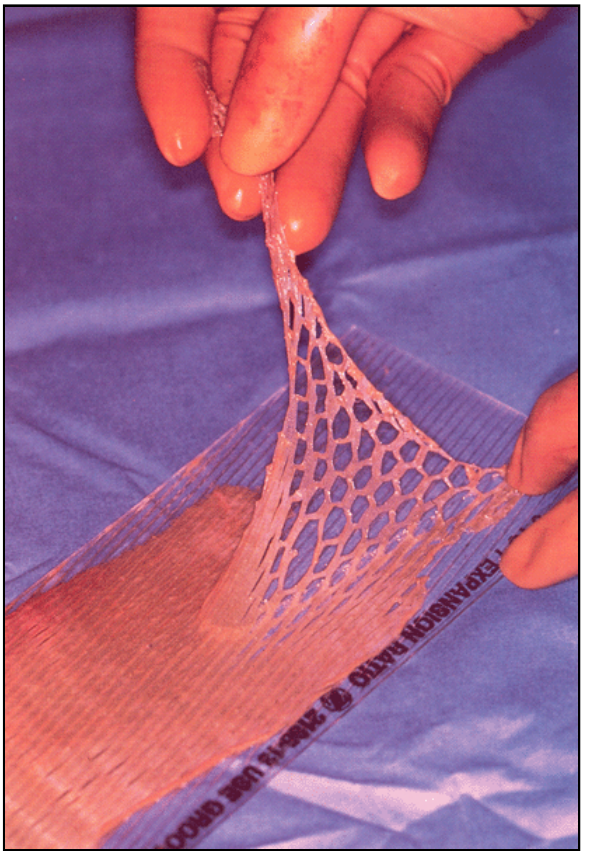

Fig. 11-9. When a casualty's donor **sites are limited, a meshed** autograft, which can expand, allows **a** greater portion of the burned body surface to be covered.

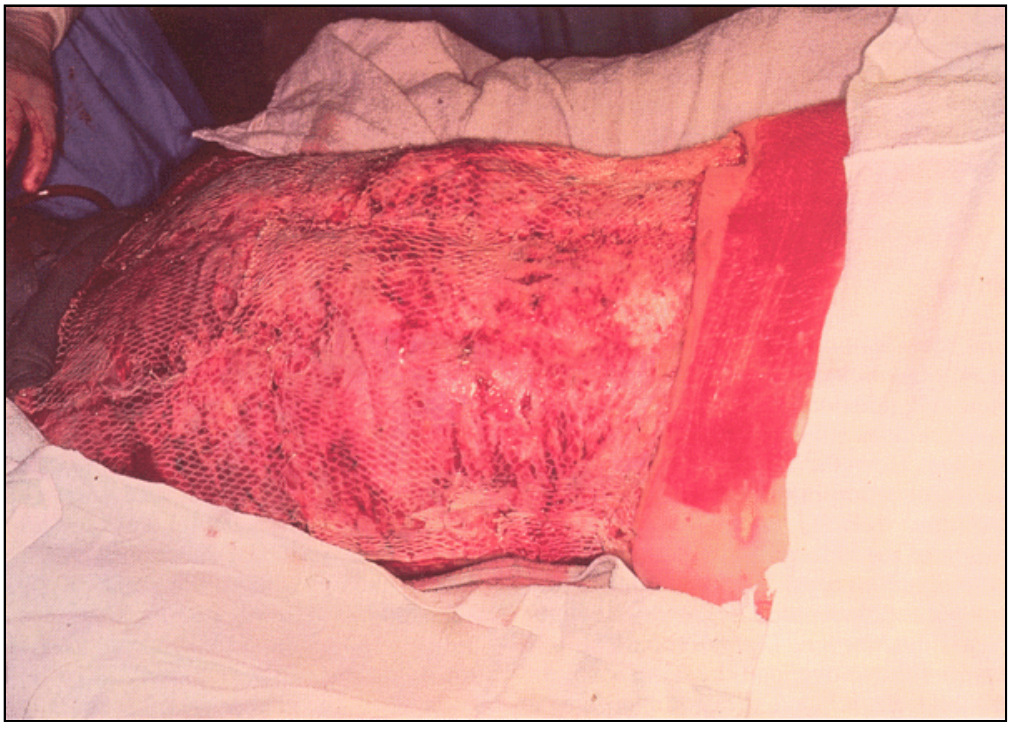

Fig. 11-10. The investing fasciae of this chest and abdomen have been covered with meshed autograft expanded 3:1.

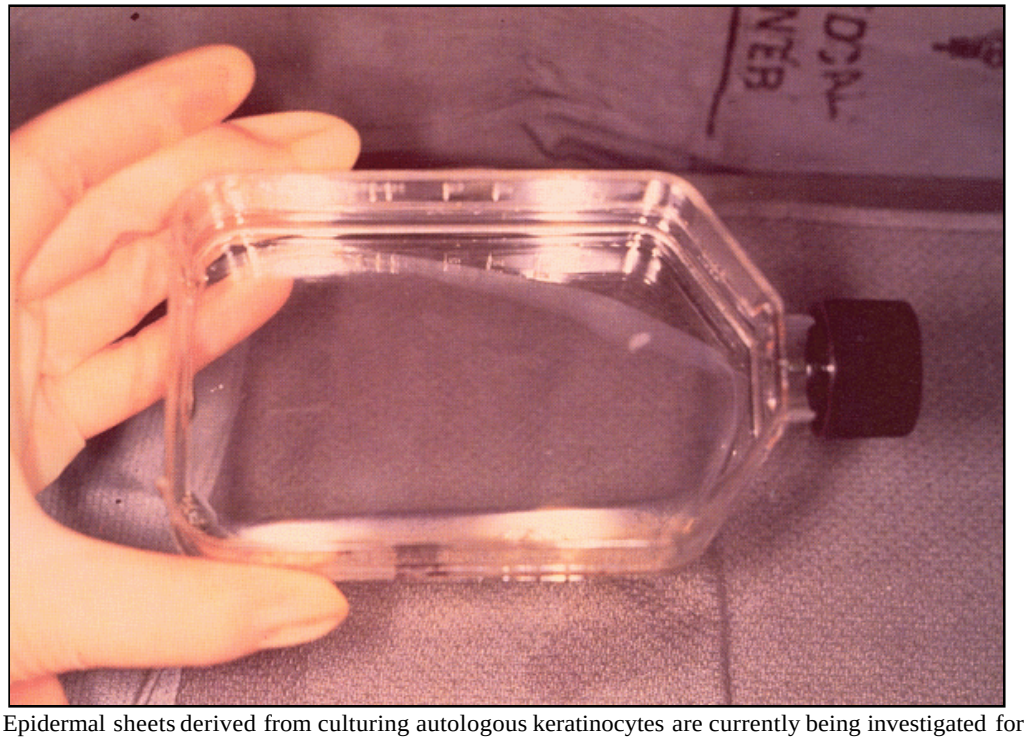

Fig. **11-11.** Epidermal sheets derived from culturing autologous keratinocytes are currently being investigated for use as a substitute for split-thickness skin grafts. The extremely thin sheets average only 5–10 cells thick and are exceptionally vulnerable to mechanical trauma. However, a ***2*** x 2-cm biopsy can be expanded to **1.5–2**m^2 in 25–30 days, allowing for significant wound coverage.

recently gained wide popularity (Figure 11-11).[26] Epidermal sheets are generated from autogenous keratinocytes and are used in place of split-thickness autografts. However, the use of culture-derived keratinocytes now appears to be limited by *(a)* the 3-4 weeks necessary to grow the epidermal sheets, *(b)* the fragility of the tissue, and *(c)* its susceptibility to infection.

Complications

The first complications in burn patients are those associated with resuscitation. Timely institution of resuscitation and infusion of adequate volumes of fluid make acute renal failure infrequent early in the postburn period. However, significant delays in initiating fluid resuscitation may result in a significant plasma-volume deficit, which will lead to hypoperfusion of the kidneys and subsequent renal failure. Later-occurring sepsis, multiple organ failure, and nephrotoxic antibiotics are now the most common causes of renal dysfunction. Dialysis should be initiated to correct volume overload, hyperkalemia, uncontrollable acidosis, a blood urea nitrogen greater than 100, and creatinine greater than 10. Oliguria during the first 24 hours after the burn typically reflects inadequate fluid replacement and should be treated by increasing the rate that the resuscitation fluid is infused.

Complications such as pulmonary edema associated with excess fluid administration may occur if the patient's response to resuscitation is not properly monitored. If resuscitation requirements are grossly overestimated, excessive wound edema may develop and otherwise unnecessary escharotomies may be required.

Early gastrointestinal complications, once common, are now quite rare. Emesis secondary to the ileus that typically occurs after a thermal burn is easily prevented by gastric decompression with a nasogastric tube. Curling's ulcer, the progression of the acute, punctate, shallow, mucosal erosions throughout the proximal stomach and duodenum, may occur in over 80% of patients with greater than 30% TBSAB.[27] Consequently, significant bleeding from acute ulcerations has largely been eliminated by prophylactic measures—including antacids or H_2 blockers or both—to keep the intragastric pH greater than 4.5. Since these preven-

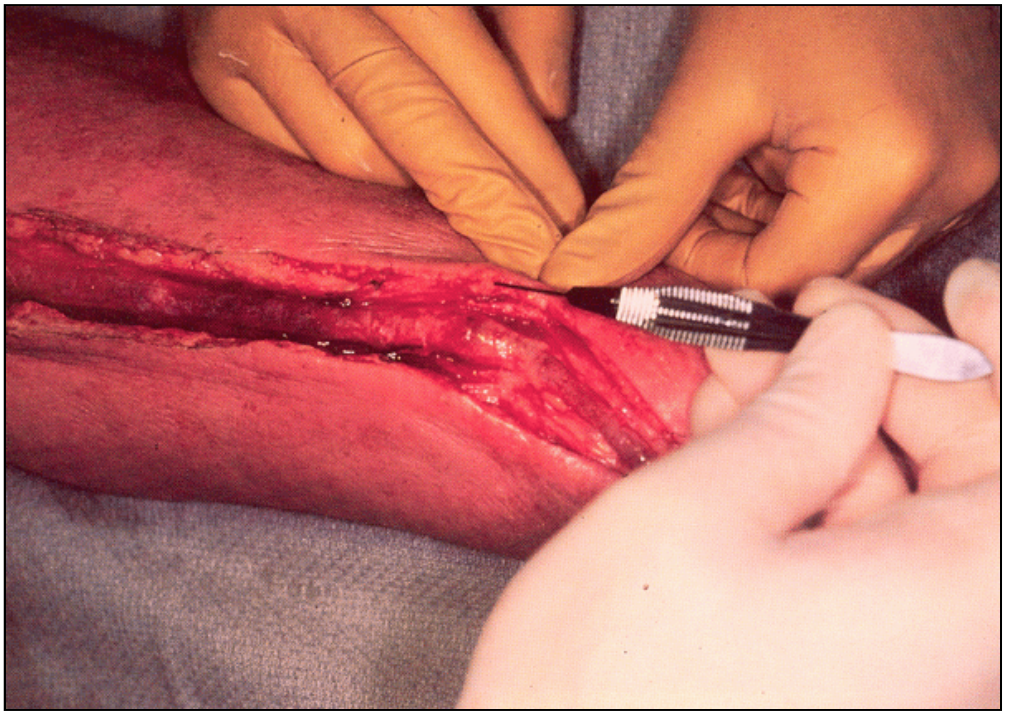

Fig. 11-12. Suppurative thrombophlebitis can occur in any previously cannulated vein. The infected vein should be excised to a point at which it is either grossly normal or has become a tributary of the next-larger order of veins. Note the infected vein's distension, discoloration, and thrombosis.

tive measures have been employed, the need to operate for upper gastrointestinal hemorrhage has markedly decreased.

Sepsis remains the most common complication that occurs after the burn patient's successful resuscitation. Extensive bacterial colonization of the burn wound, as well as the patient's immunosuppressed condition, increases the likelihood that significantly burned patients will develop infectious complications.

Pneumonia has replaced invasive burn-wound infection as the most frequent source of **sepsis** in thermally injured patients. Hematogenous pneumonia is caused by the systemic dissemination of an infecting organism from a Its radiographic hallmark—a solitary, nodular, pulmonary infiltrate that may progress to multiple infiltrates—should prompt a search for the source of infection. Common sources include an infected burn wound, endocarditis, and suppurative thrombophlebitis, all of which require immediate treatment. Airborne vectors of infection have surpassed hematogenous spread as the most common sources of pneumonia. The presence of inhalation injury and the requirement for endotracheal intubation increase the likelihood that pneumonia **will** develop. Fever, sputum leukocytosis, and the presence of infiltrates on chest roentgenograms are sensitive, but nonspecific, indicators of pneumonia.

Suppurative thrombophlebitis can occur in any previously cannulated vein. Microbial seeding at the time of insertion, the composition of the catheter, injury to the vein at the insertion site, and the duration of cannulationall predispose the patient to intraluminal infection. Limiting the time a cannula is in place to a maximum of 72 hours has significantly reduced the incidence of this complication in thermally injured **patients** from 6.9% to **1.4%** in recent years.[28] Any **venous** cannula inserted under less-than-ideal sterile conditions should be removed as soon as possible. If no other obvious source can be identified, the diagnosis of suppurative thrombophlebitis should be considered in any patient with septicemia. If local signs do not indicate an obvious source, then all previously cannulated veins should be surgically explored (Figure 11-12). The treatment of suppurative thrombophlebitis includes *(a)* administration of systemic antibiotics and *(b)* complete excision of the infected vein to the point at which the vein wall is unequivocally normal and blood can be expressed from the lumen, or to the **level** at which the vein becomes a tributary of the next-larger order of veins.

EVACUATION OF CASUALTIES WITH THERMAL INJURIES

Criteria fur Evacuation

The accepted criteria for transferring patients to a burn center include: *(a)* partial-thickness burns of 20% TBSAB or greater in adults, or 10% in children and those over 50 years old; *(b)* full-thickness burns exceeding 5% TBSAB; *(c)* burns involving the face, feet, hands, perineum, or major joints; *(d)* thermally injured patients with significant preexisting medical problems or polytrauma; *(e)* patients with electrical or lightning injuries; and *(f)* the presence of inhalation injury.[29]

During armed conflict, however, the indications for evacuation are modified.[30] For triage purposes, the presence of inhalation injury or associated major trauma may each be counted as an additional 10% burn. Casualties with small burns involving the hand, foot, or perineum should be considered for early evacuation since their activity will be disproportionately limited, but if the burns are superficial, rapid healing may permit the casualty's relatively early return to duty. If resources are severely limited, the upper burn-size limit for those receiving maximum care can be decreased by 10% increments from 70% until patient-care demands match available resources.[30]

Care During Evacuation

If the casualty will reach a combat-support or higher-echelon hospital within 2448 hours after the burn, no topical agent need be applied at the lower echelons, but the wounds should be kept clean, dry, and covered. If several days' delay is anticipated, Silvadene, if available, should be applied before the casualty is evacuated. Before and during evacuation, the wound should be managed with closed dressings *(a)* to protect from exogenous contamination and *(b)* to prevent hypothermia. Once the casualty arrives at the combat-support hospital, Silvadene should be applied to the wound after it has been debrided. Extensive burns with a significant full-thickness component should have Sulfamylon applied when the casualty

reaches a general hospital. At least once a day, all burn wounds, whether treated by the open-exposure or the closed-dressing method, should be cleansed with a surgical disinfectant to permit inspection and evaluation of the wound for evidence of infection.

The successful evacuation of thermally injured soldiers to fixed treatment centers requires the understanding of simple, but commonly misunderstood, principles.[31] If the local environment and the tactical situation permit, hemodynamic and respiratory stabilization should be achieved before the casualty is evacuated, so that resuscitation can be continued relatively simply. Thermally injured patients are best moved during the first 48 hours after being injured, before they have developed the complications of infection, pulmonary insufficiency, and acid-base abnormalities that can follow resuscitation, all of which can make stabilization and safe transport more difficult. Control of the airway and fluid resuscitation should be initiated as soon as conditions permit, preferably before evacuation.

Casualties with significant inhalation injuries, extensive head and neck burns, or decreased levels of consciousness should be intubated before transfer is begun. Fluid resuscitation should be initiated and the patient should demonstrate an adequate response to fluid infusion before evacuation begins.

Although definitive treatment of the wound is not necessary until the casualty arrives at a burn center or other definitive treatment site, a topical antimicrobial chemotherapeutic agent, if available, can be applied to the wound to prevent its desiccation and to limit microbial proliferation during a prolonged, multistop transfer. Escharotomies **should** be performed in extremities with circumferential full-thickness burns, but only if the indications discussed previously are present.

Under ideal circumstances, these guidelines for evacuation should be strictly adhered to in order to insure the casualty's morbidity- and mortality-free transfer. However, evacuating the patient from the battlefield or the unit level of care to a third-echelon MFT is usually accomplished by helicopter under less-than-ideal circumstances. Before the decision is made to move an unstable patient by helicopter, medical personnel must realize that it is impossible either to monitor such a patient adequately or to perform life-saving interventions while in flight.

There are few absolute contraindications to moving patients by air. Active hemorrhage must be controlled before the patient is transported, because bleeding during the flight may be uncontrollable and blood for transfusion is usually unavailable during transport. In **patients** with marginal respiratory reserve and problems with either ventilation or oxygenation, the underlying causes should be corrected before the casualties are evacuated.

Unique Features of Long-Distance Aeromedical Evacuation

Several medical problems are inherent in air transportation. While most military aircraft are pressurized to an altitude of 5,000–8,000 feet above sea level, the ambient pressure of oxygen will fall as the aircraft's altitude increases. Ambient pressure at 8,000 feet, which is the maximum cabin altitude to which one may be exposed, is 516 mm Hg, and would result in an alveolar oxygen tension of approximately 65 mm. Despite the administration of oxygen, lower atmospheric pressures might produce significant hypoxia in patients with impaired oxygenation.

Decreases in ambient pressure present other problems, as explained by Boyle's Law. At 8,000 feet of approximately 30%, compared to sea level. The patient with an untreated pneumothorax that is well tolerated at sea level will require a tube thoracostomy to be placed before being transported by air, because even a small pneumothorax may expand during the flight. Because air in the cuff of an endotracheal tube will also expand, low-pressure, high-volume cuffs should be routinely utilized in order to maintain their pressure no higher than 20 mmHg. Since ileus is the natural consequence of an extensive burn, gastric decompression before the casualty is transported is of utmost importance to avoid emesis and possible aspiration. Air splints that may be used for extremity fractures may become tense enough to obstruct circulation in the limb even at altitudes of 15,000–20,000 feet above sea level unless the excess pressure is relieved.

Patients evacuated in large transport aircraft may require an increase in the rate at which their intravenous fluid is administered to compensate for the low humidity in the cabin. Providing humidified gas through a face mask or an endotracheal tube is necessary to prevent the formation of desiccated and inspissated secretions, which may occlude the airway.

Particular attention to the ambient temperature is necessary in order to maintain normothermia in those casualties who are prone to both hypo- and hyperthermia. Controlling acute problems before evacuation begins, such as maintaining the airway, securing the intravenous lines, and decompressing the gastrointestinal tract, and continually monitoring the adequacy of both hemodynamic and pulmonary functions during the flight ensure the safe transport of the most extensively burned, severely injured patients.

INHALATION INJURY

True inhalation injury is a chemical injury caused by the products of incomplete combustion, and many casualties with thermal burns will also suffer such injury. True thermal damage to the tracheobronchial tree is rare and most often is caused by steam, which can contain 4,000 times more heat than air can. The diagnosis of inhalation injury is based on both history and physical examination, and can be confirmed by bronchoscopic examination and ^{133}Xenon hypoxemia and hypercapnia develop as the casualty's ability to clear secretions decreases because of *(a)* bronchociliary damage, *(b)* alveolar collapse due to decreased production of surfactant, and *(c)* alveolar filling with fluid because of alterations at the endothelial-epithelial interface. Impaired alveolar expansion and clearance of secretions place the patient at significant risk for pneumonia. The treatment includes *(a)* ventilatory support as indicated, *(b)* frequent endotracheal suctioning, and *(c)* bronchodilator therapy and racemic epinephrine if bronchospasm is present. Antibiotics should be reserved to treat diagnosed infections and should not be administered prophylactically to patients with inhalation injury.[33] Patients without respiratory distress but suspected of having a significant inhalation injury (facial burns and singed hairs, or other evidence of injury such as fire in an enclosed space—makes inhalation injury likely) should be prophylactically intubated and placed on mechanical ventilation before being evacuated, if such support is available. These patients may experience tracheobronchial mucosal slough and will require frequent endobronchial toilet, bronchoscopy, or even tracheostomy for adequate pulmonary toilet.

Carbon monoxide is a byproduct of organic material combustion with an affinity for hemoglobin 200-fold greater than oxygen's. Carbon monoxide has little direct effect on the lung, but it reduces the oxygen-carrying capacity of blood by *(a)* binding to hemoglobin, *(b)* shifting the oxygen-hemoglobin dissociation curve to the left, *(c)* binding to myoglobin, and *(d)* binding to the terminal cytochrome oxidase, all of which impair the delivery and utilization of oxygen at the cellular level. Headache, confusion, and irritability are associated with carboxyhemoglobin levels greater than 20%. Levels greater than 40% are usually associated with coma.[22] The diagnosis of carbon monoxide poisoning must be made by direct oximetric measurement of carboxyhemoglobin levels. Routine arterial blood gases, which measure dissolved oxygen, will not exclude this disorder. If measurement is not possible, then all patients suspected of having either carbon monoxide poisoning or of sustaining injury within an enclosed space should be treated with 100% oxygen administered through a tight-fitting face mask or an endotracheal tube. This treatment reduces the half-life of carboxyhemoglobin to approximately 30 minutes, down from 4–6 hours while breathing room air.[34]

COMBINED MECHANICAL AND THERMAL TRAUMA

Although their occurrence is uncommon in civilian trauma, thermal injuries complicated by associated mechanical trauma do occur in a combat environment. In managing patients with thermal and orthopedic injuries, the surgeon's principal objective is to achieve bony union in an optimal position while preserving maximal function. The open burn wound must also be treated to *(a)* prevent infection, *(b)* preserve uninjured soft tissue, and *(c)* maximize functional recovery.

Initial management of these complicated injuries involves cleansing the burn wound (as previously described) and, if the associated fracture is open, copiously irrigating and surgically debriding the devitalized tissue. Topical antimicrobial agents and intravenous antibiotics are also recommended for patients with open fractures and overlying burn wounds.

The actual timing for stabilizing fractures depends on *(a)* the type of fracture encountered, *(b)* the adequacy of the debridement, and *(c)* the patient's overall condition. Early operative intervention, before the adequacy of resuscitation has been established, should be avoided. After a simple transverse fracture has been reduced, a bivalve cast should be applied, which will permit the surgeon to routinely inspect and treat the overlying burn wound. Open or closed fractures of the humerus or femur can be managed with balanced skeletal traction, which permits

local burn-wound care and also reduces the fracture. Unstable fractures involving the shafts of long bones can be managed by placing traction pins through the tissues above and below the fracture, preferably through unburned skin, and applying external fixation devices. This approach permits direct observation and care of the overlying burn. Using internal fixation devices like intermedullary rods or plates should be avoided, because microbial seeding of the prosthesis may occur, secondary to the repetitive bacteremias that occur in patients with extensive thermal injuries. Once the fracture has been stabilized, the burn wound can be closed whenever clinically appropriate.

The combination of significant torso trauma and thermal injury represents a difficult challenge. Although operative intervention cannot be delayed when it is indicated, it should be limited specifically to the one that most quickly and safely addresses the problem. The surgeon must be cognizant of the burn's fluid requirement as well as the associated trauma, and continue burn-related resuscitation during the procedure. Abdominal wounds should be closed with retention sutures in an effort to avoid a potentially catastrophic wound dehiscence.[35] The skin and subcutaneous tissue superficial to the investing fascia of the incision should be left open.

CHEMICAL INJURY

Chemical burns require a sequence of care different from the care that thermal burns receive. Wound care takes initial priority following the American College of Surgeons' Advanced Trauma Life Support ABCs (that is, airway, breathing, and circulatory problems), in an attempt to limit the tissue damage that the chemical causes. Since the severity of the injury is directly related to both *(a)* the amount and concentration of the chemical and *(b)* the duration of the tissue's contact with the chemical, immediate and copious water lavage is essential. All clothing, including gloves and shoes, that have been contaminated with the chemical must be removed during the lavage, and the exposed skin must be well irrigated (Figure 11-13). After the irrigation, however, the topical care for chemical burns is the same as the care for other burns.

Injuries caused by specific agents such as strong alkali powders, hydrofluoric acid, and white phosphorus require that this general treatment plan be modified. Do not attempt to neutralize acid or alkali solutions; the heat that the neutralization reaction generates may cause further tissue damage.

Hydrofluoric Acid

Therapy for hydrofluoric acid burns consists of water lavage followed by specific treatment to detoxify the fluoride ion, which is absorbed through the skin into subcutaneous tissue. Tissue damage can be limited by either *(a)* applying topical 2.5% calcium gluconate gel or *(b)* injecting a 5% solution of calcium gluconate, both of which will combine with the fluoride ions to form an insoluble calcium salt."

White Phosphorus

White phosphorus is an incendiary that oxidizes to phosphorus pentoxide when it is exposed to oxygen. Particles are usually driven into the skin and subcutaneous tissue during explosions and can ignite, causing further tissue damage if allowed to oxidize (Figure 11-14). The casualty's clothing and the visible particles of white phosphorous should be expeditiously removed. The patient's wounds should be kept moist with saline until all particles can be removed. Historically, a 0.5% solution of copper sulfate has been used as a specific agent; it forms a coating of cupric phosphide that both impedes oxidation and facilitates identification of the white phosphorus particles. Since copper sulfate is readily absorbed through the burn, and even through unburned skin, however, significant toxicity—including hemolysis and renal failure—may occur with prolonged exposure. If copper sulfate is used, it should be *(a)* freshly made, *(b)* applied only as a brief wash, and *(c)* promptly lavaged from the wound with a copious volume of water. At present, water lavage and prompt removal of all visible white phosphorus particles is the preferred treatment. Using a Wood's lamp or other sources of ultraviolet light will facilitate the identification of white phosphorus particles in wounds.

While the casualty is being evacuated, the wounds should be kept in moist saline dressings to avoid the further oxidation and possible ignition of the remaining white phosphorus particles. All particles of white phosphorus that are removed should be kept immersed in water to prevent fires in the operating room.

Napalm

The wounds caused by napalm are no different from common flame burns and require no special treatment. The composition of napalm is discussed in Chapter One.

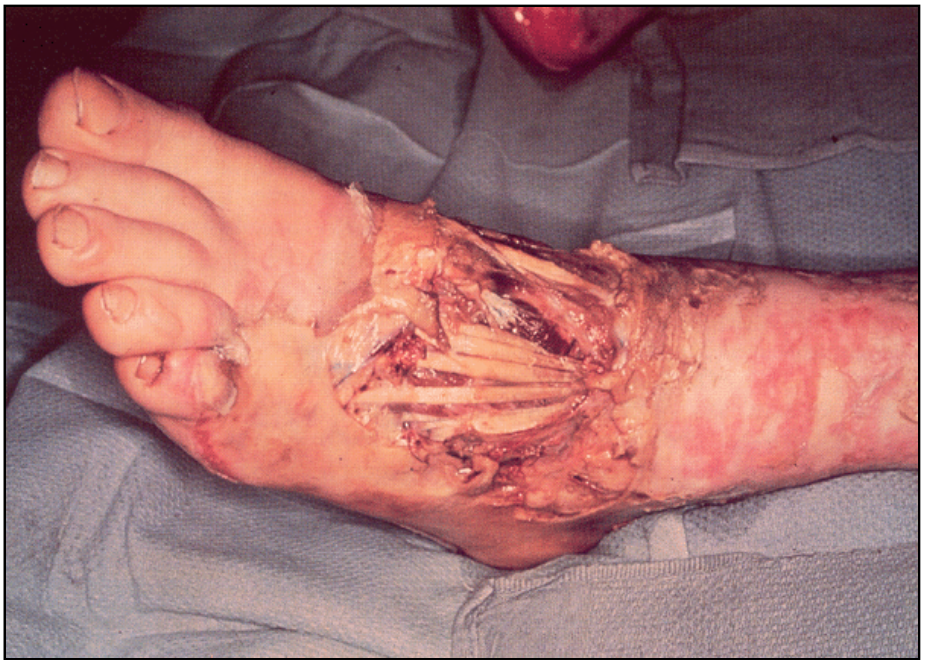

Fig. 11-13. This casualty's footwear was not promptly removed after his foot was exposed to acid. The significant soft-tissue damage should emphasize early wound care to medical officers who may need to treat casualties who have chemical burns.

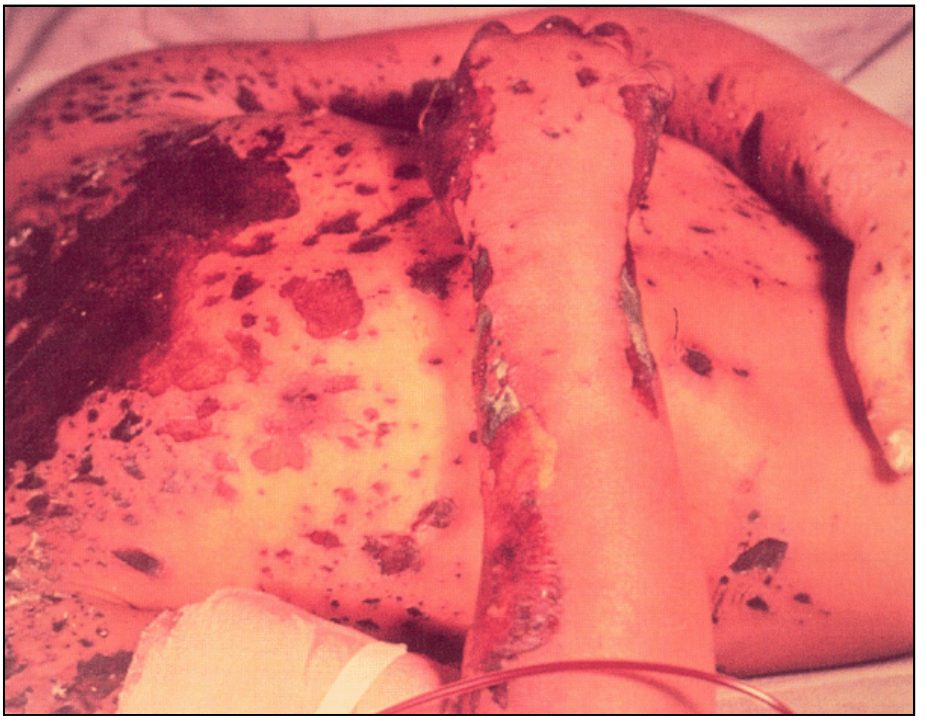

Fig. 11-14. This serpiginous, speckled pattern is typical when particles of white phosphorus are imbedded in the skin and subcutaneous tissues after a white phosphorus-containing munition has exploded. Fragments from the exploding munition can also cause significant soft-tissue injury. During treatment, all wounds must be kept moist to prevent further ignition of the imbedded particles of white phosphorus.

Vesicants

Vesicants, or blistering agents, include mustards, nitrogen mustards, other arsenicals, and mixtures of these compounds. These agents may cause significant damage to the eyes, mucous membranes, pulmonary system, skin, and hematopoietic system. In general, vesicants are odorless and cause little pain upon contact, except for the common arsenical vesicants, such as phenyldichloroarsine and chlorovinyldichloroarsine. As with all chemical injuries, casualties who are contaminated with vesicants place the medical personnel at significant risk of injury; they must take precautions to avoid direct contact with the chemical agent. Unhydrolized vesicants on a casualty's skin may persist for a prolonged period of time, representing a special hazard to medical personnel.

Mustard Gus. Mustard gas is easily aerosolized. Upon contact with a moist surface, the gas hydrolizes to hydrochloric acid, which injures tissue. Mustard gas can be either inhaled or absorbed through the skin or conjunctiva; the eyes are more vulnerable to injury than either the respiratory tract or the skin. Ocular injury may range from mild conjunctivitis and keratitis to significant corneal ulceration and globe perforation, depending upon the duration of exposure. Severe corneal involvement occurs in less than 0.1% of casualties, however. Ophthalmic injuries are treated like any other ocular chemical injury, by rapidly irrigating the eyes with copious amounts of water.

The severity of cutaneous lesions and the rapidity with which they develop depend entirely upon both the concentration of the mustard exposure and its duration. Hot, humid weather increases mustard gas's potential to injure, because high humidity increases the hydrolysis of the parent compound. Cutaneous exposure is followed by a latent period, after which the skin gradually becomes erythematous, resembling a first-degree burn. Except in mild exposures, the erythema is followed by vesication, which is caused by progressive necrosis of the superficial epidermal layers. These lesions are typical superficial, partial-thickness burns. Wound treatment consists of local irrigation and topical antimicrobial burn-wound therapy. Since the wounds are superficial, excision and grafting are rarely needed.

Inhalation injury caused by mustard gas is predominantly a tracheobronchial mucosal injury, with pulmonary parenchymal injury occurring only in the most severe cases. In general, this injury is typical of inhalation injury caused by smoke, and the treatment is the same.

Nitrogen Mustards. Nitrogen mustards are more volatile than mustard gas, but also are significantly less active as vesicants. They are less readily hydrolized than is typical mustard. The eye injuries that nitrogen mustards cause tend to be more severe than those that mustards cause; however, the treatment is the same and requires a rapid decontamination of the eye by irrigation with water. The cutaneous and pulmonary effects of nitrogen mustards are typically less severe than those caused by mustards, and their treatment is also the same.

Arsenical Vesicants. Arsenical vesicants are more volatile than mustards; they react rapidly with water to yield solid arsenoxides, however, which decreases their vesicant activity. Exposure to liquid arsenical vesicants causes severe eye damage. Pain and blepharospasm occur almost instantly. The injury will be severe if the exposure is prolonged.

Liquid arsenical vesicants cause a more severe injury to the skin than liquid mustards. Pain occurs almost immediately upon contact, and there is no latent period following exposure. The burn tends to be a deep, partial thickness injury, but may progress to a full-thickness injury that will require excision and grafting for definitive wound closure.

While vesicant injuries should generally be treated like all other chemical injuries, medical officers must be cognizant of these compounds' propensity to cause significant ocular and pulmonary injuries.

Chemical injuries to the eyes require prompt irrigation, first with water, then continued with a saline solution until the patient has arrived at the site of definitive care and is examined by a physician experienced in the treatment of such eye injuries.

In addition to the direct toxic effects, vesicant exposure can lead to generalized systemic derangements. With extensive exposure to mustard gas, particularly in amounts approaching a lethal dose, hematopoietic tissues such as bone marrow, lymph nodes, and the spleen sustain significant injury. Marked leukopenia and thrombocytopenia may develop with their associated complications. Nitrogen mustards have greater systemic toxicity. Exposure to these agents can produce degenerative changes in the bone marrow, often within 12 hours of exposure, and can actually progress to severe marrow aplasia. The thymus, spleen, and lymph nodes are also vulnerable target organs, and severe granulocytopenia, lymphopenia, thrombocytopenia, and even anemia may develop after exposure.

The arsenical vesicants can also cause systemic poisoning, which manifests as a change in capillary permeability, giving rise to significant fluid shifts, hemoconcentration, and even hypovolemic shock, Those agents that are oxidized in the liver and then excreted in the bile are also markedly toxic to the

hepatobiliary system. Focal necrosis of the liver, biliary mucosal necrosis, and even injury to the gastrointestinal mucosa may occur as a consequence of arsenical vesicant poisoning.

It has also been reported that fishermen exposed to leaking mustard-gas shells that had been discarded after World War II and inadvertently brought aboard their ships have demonstrated chromosomal mutagenicity that presumably increases their risk for developing neoplasia.

ELECTRICAL INJURY

The characteristics of electrical injury require that the treatment plan normally employed for patients with thermal injuries be modified. Tissue damage is caused by a combination of the electrical current's conversion from electrical energy into heat and the ignition of clothing as a result of electrical arcing. The amount of tissue damage depends upon the current's *(a)* voltage, *(b)* type, *(c)* pathway, and *(d)* duration of contact with the casualty.

Resuscitation

Cardiopulmonary arrest is frequently seen in patients who sustain high-voltage injury, and immediate initiation of cardiopulmonary resuscitation is required.

Using the standard resuscitation formulae—based on the extent of cutaneous injury—may result in grossly underestimating the fluid requirements for patients with significant electrical injury, whose extensive deep-tissue injury may be associated with only limited cutaneous injury. Oliguria should be treated with an increased rate of fluid administration until the desired hourly urinary output is achieved. Myoglobinuria secondary to damaged muscle increases the risk of acute renal failure. Unless a brisk urinary output is maintained, myoglobin may precipitate in the renal tubules. The presence of myoglobin in the urine mandates maintaining a urinary output of 75–125 ml/hr. Administering 12.5g of Mannitol per liter of resuscitation fluid and alkalinizing the urine are indicated only if increased fluid infusion fails to clear the urine of hemochromogens.

Hyperkalemia secondary to extensive tissue damage may also occur. This is treated by administering hypertonic glucose, insulin, and calcium gluconate intravenously. Ion-exchange resins such as Kayexalate may be necessary and, rarely, hemodialysis may be indicated.

Compartment Syndrome

Edema beneath the muscle fascia may impair blood flow to the muscle and to distal tissue, resulting in a compartment syndrome (Figure 11-15). The indications for escharotomy and fasciotomy following electrical injury are the same as those following conventional thermal injury, While fasciotomy is seldom required to restore circulation to a circumferentially burned extremity, it may be necessary in patients with a high-voltage electrical injury, associated skeletal trauma, or a severe crush injury. (Ideally, fasciotomy should always be performed in the operating room under general anesthesia, but conditions may permit only local anesthesia and sterile surgical technique.) Limbs with significant electrical injuries should be surgically debrided as soon as the casualty's clinical condition permits. At operation, all obviously necrotic tissue should be debrided, and amputation, if required, should be carried out at a level proximal to the injury. The deep muscles and vital structures should be explored and their viability assessed, because nonviable tissue may underlie superficial viable tissue. Some authors have proposed ^{133}Xenon washout kinetics and tissue-uptake of technetium pyrophosphate as methods of identifying deep-tissue injuries, but the clinical usefulness, accuracy, and reliability of these techniques remain to be proven.[37,38] Surgical exploration remains the standard for delineating tissue viability.

The patient should be returned to the operating room within 48 hours after the initial debridement or amputation, where the wound is inspected and further debridement carried out as necessary. Upon completion of debridement, the wound can be closed by skin grafting or delayed primary closure, or it can be allowed to form granulation tissue and be autografted later.

Neurological Complications

Neurological sequelae may have early or delayed onset after electrical injury, thus mandating a thorough neurological examination on admission and at scheduled intervals later. Inasmuch as spinal fractures can be produced by tetanic contraction of the paraspinal muscles or by a fall at the time the injury occurred, anterior-posterior and lateral radiographs of the cervical, thoracic, and lumbar spine should be performed on all patients with electrical injuries.

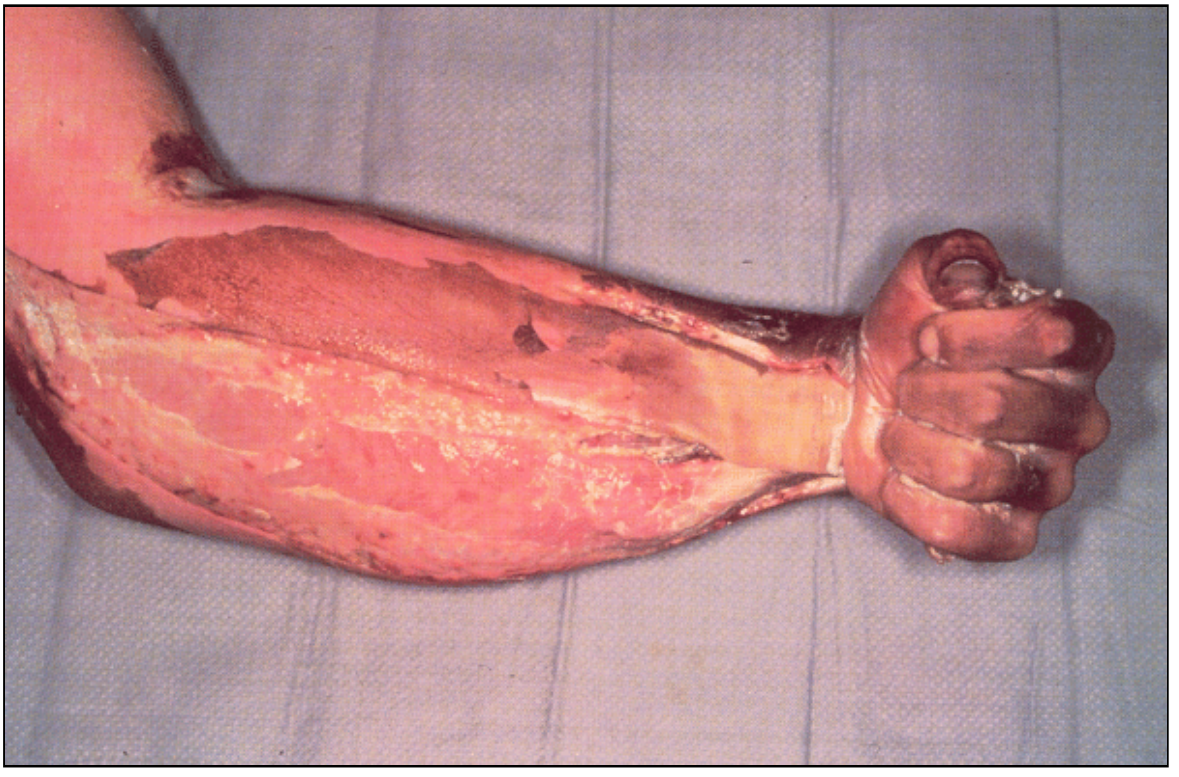

Fig. 11-15. Extensive subfascial edema necessitating fasciotomy may occur after electrical injury. The flexed wrist and hand occurs as a consequence of the nonviable muscle, seen herniating through the fasciotomy incision.

CURRENT RESEARCH AND FUTURE DIRECTIONS OF BURN CARE

The techniques of burn care that have been developed during the past four decades have significantly improved the survival of young-adult burn patients. This progress has both revealed previously unappreciated physiological consequences of burn injury and resulted in the emergence of new complications upon which current research is focused. Research programs sponsored by the U.S. Army Medical Research and Development Command and other agencies address the general systemic effects of severe injury as well as the organ-specific effects of burns.

Diagnostic systems employing spectral reflectance and other physical modalities are being evaluated for use in making early accurate diagnoses of burn depth and in identifying those wounds that require surgical debridement. Hemodynamic and shock studies at the U.S. Army Institute of Surgical Research include using plasmapheresis to produce a model of the acute plasma-volume changes that occur after a burn, and to describe *(a)* the biochemical changes that occur in the burn wound and *(b)* how the composition of the resuscitation fluid affects those changes. The neurohormonal changes that occur soon after a burn and influence salt and water balance are being described in detail to identify pharmacological interventions that might be effective in patients who do not respond to resuscitation as anticipated.

Ongoing correlative laboratory and clinical studies that seek to identify the prevalence and increased mortality of inhalation injury in burn patients have investigated *(a)* the pathogenesis of inhalation injury and *(b)* the interaction of cutaneous and inhalation injuries, and have evaluated synthetic surfactant-replacement therapy and high-frequency ventilation.

Epidemiological studies have documented the ever-changing ecology of infection in burn patients and the emergence of nonbacterial opportunistic agents as the causes of infections in extensively burned patients, whose survival has been prolonged by current treatment techniques. The difficulty of diagnosing infection in hypermetabolic burn patients has focused attention on identifying infection-specific, blood-borne indicators that will permit early, reliable diagnosis of infections.

The effects that burn injury, burn treatment, and infection have on the patient's defenses are also being assessed. Researchers are studying the effects of thermal injury on:

- the number and function of lymphocyte subpopulations
- the immunosuppressive effects of blood transfusions
- the effectiveness of exogenous interleukin-2 therapy
- the relationship between intestinal permeability and bacterial translocation in burn patients
- the effectiveness of hematopoietic growth factors in restoring the immunocompetence of burn patients, and
- the role of prostaglandins, various cytokines, and other products of cell metabolism in the patient's susceptibility to endotoxin and bacterial challenges after a burn

Several investigators are studying the effectiveness of various growth factors as accelerants of burn-wound healing. Studies at the U.S. Army Institute of Surgical Research are documenting that low-amperage direct current, with silver-impregnated nylon used as an anode applied to the burn wound, exerts both prophylactic and therapeutic effects on invasive burn-wound infections and also accelerates healing of deep partial-thickness burns, split-thickness skin grafts, and split-thickness graft-donor sites.

Many investigators continue to define the pathogenesis and characteristics of
lism. Studies of thyroid function have identified altered iodothyronine feedback as an important factor in the disturbed secretion of thyroid-stimulating hormone that occurs after a burn. Other studies have lead to the proposal that the circadian sympathetic unresponsiveness of the pineal gland serves as a model of the sympathetic unresponsiveness observed in critically ill, burned, and injured patients.

Nutritional research at the U.S. Army Institute of Surgical Research has been directed towards defining injury-specific nutritional needs including: *(a)* trace-metal metabolism, *(b)* nutritional efficacy of medium-chain triglycerides, and *(c)* the interrelationship between vitamin metabolism and plasma amino acid levels.

In other laboratories, investigators are studying the effects of *(a)* various fatty acids on immune function, *(b)* glutamine on intestinal integrity, and *(c)* pharmacological manipulations of nutritional balance.

Another area of active research concerns the development of new and improved biological dressings and skin substitutes. Researchers are *(a)* evaluating collagen-based skin substitutes with varying permeability characteristics and *(b)* seeding the dermal analogues of skin substitutes with autologous epidermal cells and fibroblast, to try to effect permanent wound closure with these membranes. Improved techniques for growing sheets of epithelial cells *in vitro* and producing composite tissue cultures for definitive closure of the burn wound are also being evaluated.

Incorporating the results of these research programs into the clinical care of burn patients will further improve their chances for survival and functional recovery. Such improvements will undoubtedly reveal other previously unapparent pathogenetic effects of bum injury and result in the emergence of yet other complications of both injury and treatment. These emerging problems will then form the basis for future research initiatives.

SUMMARY

Not only are burns common in modern warfare, but combat involving armored fighting vehicles and aircraft is also likely to result in combined thermal and mechanical trauma, which increases the complexity of the care that the burned casualty requires. Successful salvage of the severely burned soldier requires a broad base of knowledge and expertise including critical care, infection control, wound management, respiratory care, metabolism, nutrition, and surgical technique.

Immediate early care consists of maintaining vital organ function and treating the potentially life-threat-

ening associated injuries. Fluid resuscitation, the cornerstone of initial burn therapy, will prevent many of the early complications associated with thermal injury, provided it is promptly initiated and carefully monitored. After they have been resuscitated, patients with significant burn injury are best cared for at fourth-echelon or CONUS MTFs.

Early operative intervention is rarely required to treat thermal injuries, and, if performed within the resuscitative phase, it places the patient at increased risk for cardiovascular collapse. Early rare of wound consists of applying a clean, dry dressing (changed as necessary) until the patient can be transported to a third- or fourth-echelon facility. Topical antimicrobials are essential to proper management of the burn wound.

Salvaging a thermally injured soldier is resource-intensive and requires an aggressive team approach. While this is **best** achieved at specialized treatment centers, it may not be possible during mass-casualty wartime situations. All medical officers need to know of caring for

REFERENCES

1. Nebraska Burn Institute. *Advanced Burn Life Support Manual,* 18*Delta.* 1989. Lincoln: Nebraska Burn Institute.

2. Eldad, I., and Torem, M. 1990. Burns in the Lebanon War 1982: "The blow and the cure." *Milit. Med.* 155:130–132.

3. Pruitt, B. A., Jr. 1985. The universal trauma model. *Bulletin of the American College of Surgeons* 70(10):2–13.

4. Goodwin, C. W.; Dorethy, J.; and Lam, V. 1983. Randomized trial of efficacy of crystalloid and colloid resuscitation on hernodynamic response and lung water following thermal injury. *Ann. Surg.* 197:520–531.

5. Graves, T. A.; Cioffi, W. G.; McManus, W. F.; Mason, A. D.; and Pruitt, B. A., Jr. 1988. Fluid resuscitation of infants and children with massive thermal injury. *J. Trauma* 28:1656–1659.

6. Monafo, W. S. 1970. The treatment of burn shock by the intravenous and oral administration of hypertonic lactated saline solution. *J. Trauma* 10:575–586.

7. Caldwell, F. T., and Bowser, B. H. 1979. Critical evaluation of hypertonic and hypotonic solutions to resuscitate severely burned children: A prospective study. *Ann. Surg.* 189:546–552.

8. Shimazaki, S.; Yoshioka, T.; and Tanaka, N. 1977. Body fluid changes during hypertonic lactated saline solution therapy for burn shock. *J. Trauma* 17:38–43.

9. Alexander, J. W., and Wixon, D. 1970. Neutrophil dysfunction in sepsis in burn injury. *Surg. Gynecol. Obstet.* 130: 431–438.

10. Pruitt, B. A., Jr.; Dowling, J. A.; and Moncrief, J. A. 1968. Escharotomy in early burn care. *Arch. Surg.* 96:502–507.

11. Saffle, J. R.; Zeluff, G. R.; and Warden, G. D. 1980. Intramuscular pressure in the burned arm: Measurement and response to escharotomy. *Am. J. Surg.* 140:825–831.

12. Pruitt, B. A., Jr. 1984. The diagnosis and treatment of infection in the burn patient. *Burns* 11:79–91.

13. Pruitt, B. A., Jr., and Foley, F. D. 1373. The use of biopsies in burn patient care. *Surgery* 73:887–897.

14. Kim, S. H.; Hubbard, G. B.; and McManus, W. F. 1985. Frozen technique to evaluate early burn wound biopsy: A comparison with the rapid section technique. *J. Trauma* 25:1134–1137.

15. Kim, S. H.; Hubbard, G. B.; and Worley, B. G. 1985. A rapid section technique for burn wound biopsy. *J. Burn Care Rehab.* 6:433–435.

16. McManus, A. T.; Kim, S. H.; McManus, W. F.; Mason, A. D., Jr.; and Pruitt, B. A., Jr. 1987. Comparison of quantitative microbiology and histopathology in divided burn-wound biopsy specimens. *Arch. Surg.* 122:74–76.

17. McManus, W. F.; Goodwin, C. W.; and Mason, A. D., Jr. 1981. Burn wound infection. *J. Trauma* 21:753–756.

18. McManus, W. F.; Mason, A. D., Jr.; and Pruitt, B. A., Jr. 1980. Subeschar antihintir infusion in the treatment of burn wound infection. *J. Trauma* 20:1021–1023.

19. Pruitt, B. A., Jr.; and Goodwin, C. 1983. Nutritional management of the seriously ill burned patient. In *Nutritional Support of the Seriously Ill Patient*, edited by R. W. Winters, 63–84. New York Academic Press.

20. Wilmore, D. W. 1979. Nutrition and metabolism following thermal injury. *Clin.* 1:603–619.

21. Wolfe, R. R.; O'Donnell, T. F., Jr.; Stone, M. D.; Richmond, D. A.; and Burke, J. F. 1980. Investigation of factors determining the optimal glucose infusion rate in total parenteral nutrition. *Metabolism* 29:892–900.

22. Crocker, P. J. 1984. Carbon monoxide poisoning, the clinical entity and its treatment: A review. *Milit. Med.* 149: 257–259.

23. Curreri, P. W.; Wilmore, D. W.; and Mason, A. D., Jr. 1971. Intracellular cation alterations following major trauma: Effect of supranormal calorie intake. *J. Trauma* 11:390–396.

24. Canizaro, P. C.; Sawyer, R. B.; and Switzer, W. E. 1964. Blood loss during excision of third degree burn. *Arch. Surg* 88:800–802.

25. Roberts, L. W.; McManus, W. F.; Mason, A. D.; and Pruitt, B. A., Jr. 1985. Duoderm in the management of skin graft donor sites. In *Surgical Research: Recent Development. Proceedings of the First Annual Session of the Academy of Surgical Research*, edited by C. W. Hall, 55–58. New York Pergamon Press.

26. Gallico, G. G. III; O'Connor, N. E.; and Compton, C. C. 1984. Permanent coverage of large burn wounds with autologous cultured human epithelium. N. *Engl. J. Med.* 311:448–451.

27. Czaja, A. J.; McAlhany, J. C.; and Pruitt, B. A., Jr. 1974. Acute gastroduodenal disease after thermal injury: An endoscopic evaluation of incidence and natural history. N. *Engl. J. Med.* 291:925–929.

28. Pruitt, B. A., Jr.; Stein, J. M.; and Foley, F. D. 1970. Intravenous therapy in burn patients: Suppurative thrombophlebitis and other life threatening complications. *Arch. Surg.* 100:399–404.

29. Committee on Trauma. 1990. Resources for optimal care of patients with burn injury. In *Resources for Optimal Care of the Injured Patient*. 57–60. Chicago: American College of Surgeons.

30. Bowen, T. E., and Bellamy, R. F., eds. 1989. Burn injury. Chapt. 3 in *Emergency War Surgery*, 2d U.S. revision, 35–56. Washington, DC: U.S. Department of Defense.

31. Cioffi, W. G., Jr., and Pruitt, B. A., Jr. 1989. Aeromedical transport of the thermally injured patient. *Med. Corps Internat.* 4:23–27.

32. Hunt, J. L.; Agee, R. N.; and Pruitt, B. A., Jr. 1975. Fiberoptic bronchoscopy in acute inhalation injury. *J. Trauma* 15:641–649.

33. Levine, B. A.; Petroff, P. A.; and Slade, C. L. 1978. Prospective trials of dexamethasone and aerosolized gentamicin in the treatment of inhalation injury in the burned patient. *J. Trauma* 18:188–193.

34. Halebian, P.; Robinson, N.; and Barie, P. 1986. Whole body oxygen utilization during carbon monoxide poisoning and isocapneic nitrogen hypoxin. *J. Trauma* 26:110–117.

35. Goodwin, C. W.; McManus, W. F.; Mason, A. D., Jr.; and Pruitt, B. A., Jr. 1982. Management of abdominal wounds in thermally injured patients. *J. Trauma* 22:92–97.

36. Caravati, E. M. 1988. Acute hydrofluoric acid exposure. *Am. J. Emerg. Med.* 6:143–150.

37. Clayton, J. M.; Hayes, A. C.; and Hammel, J. 1977. $Xenon^{133}$ determination of muscle blood flow in electrical injury. *J. Trauma* 17:293–298.

38. Hunt, J.; Lewis, S.; and Parkey, R. 1979. The use of Technetium-99m stannous pyrophosphate scintigraphy to identify muscle damage in acute electric burns. *J. Trauma* 19:409–413.