

Chapter 22

BURN INJURIES

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SUMMARY

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INTRODUCTION

Burn injury is both a predictable consequence of modern combat and a challenge for military medical personnel. For several reasons, thermal injuries will comprise a not-insignificant fraction of the casualties of modern warfare, not only in the army but even more so in the navy and air force¹:

- The very nature of existing antipersonnel and antitank explosive munitions such as shaped-charge warheads makes thermal injury likely.
- Secondary explosions and fires from the fuels required for air and ground mobility due to battle damage make thermal injury a real possibility.
- Other weapons used in conventional warfare, such as fuel-air explosive bombs, which are designed to injure by blast overpressure, can cause thermal injuries.
- Nuclear weapons produce extremely high temperatures capable of producing severe thermal burns at a great distance.

In fact, thermal burns will constitute a major portion of the injuries in nuclear warfare.²

Throughout this chapter, the term *burn injury* refers to thermal injury caused by fire except when chemical or electrical burns are specified. Combat casualties with burns present challenges to those caring for them: some unique to thermal injury, and some common to severe trauma of other types. One of the major distinctions between burned soldiers and civilians is the propensity for soldiers to have additional injuries. Data from a World War II study of tank casualties show that of the 50% of casualties who were injured while in the tank and who survived to reach medical care, one half had penetrating injuries in addition to having been burned.³

Combat action is neither the only nor necessarily the most important source of thermal trauma for soldiers. Historically, nonbattle injuries resulting from fires from vehicular accidents or aircraft crashes have generated more burns than have combat action. Data from the Korean War illustrate this point: 1% of total admissions for combat injuries (766 soldiers) were for treatment of burns, while 7.3% of admissions for with nonbattle-related injuries (5,510 soldiers) were for treatment of burns.⁴

Besides the immediate concerns of narrowing of the upper airway, respiratory compromise, and hemodynamic instability, the burn casualty can also

present with a host of problems including altered fluid requirements, limited intravenous access and monitoring sites, altered thermoregulatory response, hypermetabolism, and immunosuppression. Other physiological perturbations affecting intensive care and anesthetic management include altered drug response, deranged pulmonary dynamics, altered hematological profile, malnutrition, sepsis, and multiple organ failure.

Large numbers of burn casualties will place heavy demands on both the medical personnel and the logistics of the military medical system. A clear understanding of the unique challenges presented by such casualties is necessary for us most efficiently and effectively to use existing medical resources and capabilities through each echelon of combat medical care.

The first step in battlefield burn care is to stop the burning process by removing the source of the burn injury: extricate the casualty from the burning armored fighting vehicle, ship, or aircraft; extinguish the flame; remove the smoldering clothing; break contact with the electrical current source; flush off the offending caustic chemical. Immediate assessment of airway adequacy, respiratory exchange, and hemodynamic stability follows. Then basic life-support measures are begun, including cardiopulmonary resuscitation if indicated. Supplemental oxygen should be administered if available and if the burn occurred in a closed space, as carbon monoxide poisoning is likely.⁵ Extensive upper-airway burns with resultant edema, obtundation, inadequate ventilation, or associated severe facial or chest trauma necessitate endotracheal intubation and ventilatory support.

After cardiopulmonary stabilization, gross estimation of all injuries including extent and depth of burn injury is made, and a clean dressing is placed over the burns. If associated injuries are extensive, blood loss is brisk, or time until definitive treatment will exceed 30 minutes, intravenous access should be secured, preferably via a large-bore peripheral percutaneous cannulation, and a balanced salt solution administered.⁶ The casualty is then transported to a location for more definitive examination and treatment.

At the level of the medical company or battalion, decisions will be made as to the further disposition of the burn casualty. Because subsequent treatment is based on the extent and severity of the burn and associated injuries, a careful physical exam and

assessment of burn depth and percentage of body surface involved should be performed at this echelon of care. Burn severity is described as *partial* thickness or *full* thickness, based on the depth of injury. Initially, burn depth may be difficult to estimate correctly and the depth may even progress over time from partial to full thickness.⁷ Superficial, partial-thickness burns range from erythematous sunburn to a devitalization of the superficial layers of the epidermis with blister formation overlying a wet, red, hypersensitive, edematous skin bed; this injury will heal spontaneously. Deeper partial-thickness burns appear dry, waxy, and white beneath the devitalized epidermis. These injuries are sensitive only to deep pressure and will heal with hypertrophic scarring if protected well. Full-thickness burns involve complete destruction of the skin and appendages, and appear dry, leathery, and somewhat translucent.⁸ Eventual excision and grafting are required for wound closure.

The extent of body surface area burned is an important variable in delivering adequate fluid resuscitation, and is estimated by the "Rule of Nines" (Figure 22-1). This involves assigning percentages of total body surface area (TBSA) to various body parts.¹ Major burn injuries are considered to be those consisting of partial-thickness burns of greater than 25% of TBSA, full-thickness burns of greater than 10% TBSA, or smaller burns (of hands, face, feet, perineum) with complicating features (eg, inhalation injury, extremes of age, significant preexisting disease, or associated trauma).⁹

Current conventional burn care dictates that major thermal injuries be managed in a specialized burn treatment facility. Moderate, uncomplicated burn injuries (ie, 15%–25% TBSA partial-thickness burn or 2%–10% TBSA full-thickness burn) will usually require hospitalization in a general hospital that has some experience in burn care.⁷ Minor burn injuries (ie, < 15% TBSA partial-thickness burn or < 2% TBSA full-thickness burn) may be treated on an outpatient basis. The *Emergency War Surgery NATO Handbook* modifies these recommendations and suggests that, to conserve medical resources in

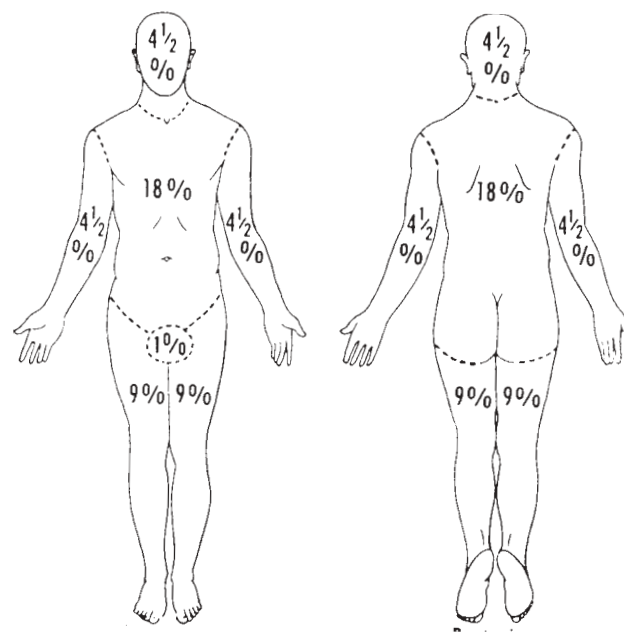


Fig. 22-1. The "Rule Of Nines" for calculating total body surface area burned: head and neck, 9%; anterior trunk, 18%; posterior trunk, 18%; upper extremity, 9%; lower extremity, 18%; and genitalia and perineum, 1%. Reprinted with permission from Moylan JA. First aid and transportation of burned patients. In: Artz CP, Moncrief JA, Pruitt BA, eds. *Burns—A Team Approach*. Philadelphia, Pa: WB Saunders; 1979: 153.

times of mass casualties, casualties with burns of less than 20% TBSA be treated either as outpatients or via self-care in a minimal care facility. In addition, these casualties are removed from active combat. Those with burns of the head and neck, hands, or feet, however, require hospitalization. Casualties with burns of 20% to 50% TBSA; or smaller burns with associated blunt, blast, or penetrating trauma, require hospitalization for definitive care and ultimate surgical closure. Casualties with very severe burns (ie, > 50% TBSA) are given minimal care, based on their increasingly poor chances for survival as the TBSA involved increases. They are treated as time and resources allow.²

EARLY CARE

At the second or third echelon of care, and before evacuation to a definitive burn care facility or general hospital with burn care experience, several steps should be initiated: some important data collected, fluid resuscitation established and monitored, peripheral perfusion maintained, tetanus prophylaxis provided, gastrointestinal precautions

initiated, analgesics administered, and burn wound care initiated.

Data Collection

When the capability to do so exists, several data need to be procured. These include the casualty's

weight (for calculating resuscitation fluid requirements), followed by daily weights; baseline chemistry and arterial blood-gas studies; and a chest radiograph to assess pulmonary status, and as a baseline for comparison with subsequent radiographs during the resuscitation and postresuscitation phases. When thermal injury is complicated by other nonthermal trauma (eg, blunt, blast, or penetrating trauma), other data should be accumulated. These may include cervical spine or inhalation/exhalation radiographs, hematocrit, gross urinalysis, or diagnostic peritoneal lavage cell count, when indicated.

Fluid Resuscitation

Fluid management of the burn casualty presents a particular challenge to the medical officer. Thermal injury necessitates aggressive, early fluid resuscitation. Burn patients exhibit a dramatic, early intravascular/extravascular disequilibrium resulting from the loss of functional capillary integrity, extravasation of intravascular fluid, resultant edema formation, and loss of circulating blood volume. Fluids are administered to restore blood and plasma volume, and organ and tissue blood flow; increase cardiac output; and decrease peripheral vascular resistance.¹⁰ A number of resuscitation formulas exist (Table 22-1), each consisting of crystalloid and colloid volume guidelines for the first 48 hours after the burn.

The widely used Modified Brooke Formula calls for 2 to 4 mL of lactated Ringer’s solution to be infused per kilogram of body weight per percentage of the total body surface area burned (2–4 mL/kg/% TBSA) during the first 24 hours after the burn. The rate of infusion of resuscitation fluid is increased or decreased within this range based on clinical response. Occasionally with massive burns exceeding 50% TBSA, the rate of 4 mL/kg/% TBSA burned is not sufficient, and higher infusion rates (to 6 mL/kg/% TBSA burned) may be required. A colloid-containing fluid equivalent to plasma may be initiated at a rate of 0.3 to 0.5 mL/kg/% TBSA burned during the second 24-hour period, along with 5% dextrose in water at a rate sufficient to maintain desired urinary output (which is discussed later in this chapter).

Including colloids during the second 24-hour resuscitation period may provide some oncotic benefit and allow more-effective restoration and maintenance of intravascular volume, since most pulmonary capillary integrity is reestablished by this time. Five percent dextrose in water makes up the balance of the second 24-hour resuscitation fluids because a high sodium intake typically occurs during initial resuscitation.

Monitoring of Resuscitation

Assessing the adequacy of resuscitation is accomplished by monitoring urinary output, blood

TABLE 22-1
RESUSCITATION FORMULAS FOR ADULT BURN CASUALTIES

Component	Modified Brooke	Parkland
First 24 Hours		
Ringer’s lactate	2–4 mL/kg/% TBSA	4 mL/kg/% TBSA
Colloid	—	—
D5H ₂ O	—	—
Second 24 Hours		
Colloid	0.3–0.5 mL/kg/% TBSA	20%–60% of plasma volume
D5H ₂ O	Volume sufficient to maintain urinary output	Volume sufficient to maintain urinary output

D5H₂O: 5% dextrose in water; TBSA: total body surface area.

Data sources: (1) Pruitt BA Jr. Advances in fluid therapy and the early care of the burn patient. *World J Surg.* 1978;2:139–150. (2) Baxter CR, Shires T. Physiologic response to crystalloid resuscitation of severe burns. *Ann NY Acad Sci.* 1968;150:874–894. (3) Baxter CR. Fluid and electrolyte changes of the early postburn period. *Clin Plast Surg.* 1974;1(4):693.

pressure, mental status, and, occasionally, cardiac filling pressures. A Foley catheter has been described as the single best monitor of the adequacy of resuscitation in patients with normal renal function and an absence of congestive heart failure, pulmonary dysfunction, or history of shock. These criteria probably describe the majority of burn combat casualties if the burn occurs without associated blast, blunt, or penetrating injury. The target urinary output is 0.5 mL/kg/h in adults and 1 mL/kg/h in children who weigh less than 30 kg.¹¹ Hypotension, cardiovascular collapse, renal failure, progression of burn depth, or mental status changes may result from inadequate resuscitation. Overzealous fluid administration can result in increased morbidity and mortality from pulmonary edema and increased peripheral edema, which can impair wound healing and convert a partial-thickness burn into a full-thickness injury.¹²

Several groups of patients have been shown to require more than the standard amounts of resuscitation fluid. These include patients with inhalation injuries, intoxicated patients, those with electrical injuries, and those undergoing delayed resuscitation.^{10,13} The Parkland Formula (see Table 22-1) was originally developed for patients with inhalation injuries and may also be more appropriate for these other subsets of patients. Also, because pediatric patients may have fluid needs not accurately predicted by most of the weight-based formulas, their fluid resuscitation should be individualized.⁹ In every case, the adequacy of resuscitation should be closely monitored, and fluids added or withheld based on the desired response of adequate peripheral perfusion rather than strict adherence to a standard formula.

Maintenance of Peripheral Perfusion

Because full-thickness, circumferential burns of the thorax, upper abdomen, or extremities can lead to loss of life or limb, early, aggressive care is mandatory. Any item of clothing or jewelry must be removed if it could potentially constrict and contribute to subsequent edema formation. Similarly, escharotomy is necessary to prevent life-threatening ventilatory compromise, limb loss, or permanent damage to underlying neurovascular structures.¹⁴ Escharotomy is performed by incising linearly at the midmedial and midlateral lines, down to and just through the subdermal fascial attachments. Anesthesia is not usually required since these deep burns are insensate.⁹

In other large burn injuries, peripheral pulses should be checked at least hourly to assess perfusion as edema worsens. A Doppler flow detector is useful to detect blood flow.

Antibiotics

As a general rule, prophylactic, systemic antibiotics are of no value in the management of burns. However, the chapter on burn injuries in *Emergency War Surgery* recommends the administration of penicillin to all thermally injured combat casualties (unless contraindicated) to prevent β -hemolytic streptococcal burn wound infections.¹ This recommendation, differing as it does from civilian standards of practice, reflects both the different milieu in which combat injuries occur vis à vis civilian trauma and the likely delay that will be encountered in evacuating the casualty from the battlefield to a fourth- or fifth-echelon burn center.

Penicillin is to be given for up to 5 days, after which antibiotics are administered only if there are signs of clinical infection or if burn sepsis is thought to be present. When burn sepsis is thought to be present, incisional biopsy is necessary to identify the origin of the infection. Appropriate antibiotics (eg, penicillin against β -hemolytic streptococcal bacteria) are instituted when the causative organism has been verified by culture or there is a high degree of clinical suspicion. In contrast to systemic antibiotics,

[t]opical antimicrobial agents decrease the incidence of invasive infections in burns. Three effective chemotherapeutic agents that are frequently used to treat burn infections are [mafenide acetate], [silver sulfadiazine], and silver nitrate.^{15(p357)}

Tetanus Precautions

Since all burn injuries are considered contaminated, tetanus prophylaxis is indicated except in casualties who have been actively immunized within the preceding 12 months. If a booster dose was administered within the preceding 10 years, an injection of 0.5 mL of adsorbed tetanus toxoid will provide prophylaxis. If active immunization was not done within 10 years before the burn, 250 to 500 units of tetanus immunoglobulin (human) should simultaneously be administered at another site.¹⁶

Gastrointestinal Precautions

Since most patients with burns of more than 20% TBSA develop a paralytic ileus during the first 24

hours after the injury, all oral intake is withheld and a nasogastric tube is inserted for gastric decompression if not contraindicated by associated injury. In addition, gastroduodenal ulceration is a frequent complication of burn injury. First described by Curling in 1842,¹⁷ these stress ulcers appear within 72 hours of the burn,¹⁸ have the potential to hemorrhage or perforate, and are associated with high mortality rates.¹⁹ Curling's ulcers occur with increasing frequency as burn size increases and, if not prevented, appear in 40% of burns of 70% TBSA.²⁰ Presumptive diagnosis is made on evidence of gastrointestinal bleeding, while definitive diagnosis is by gastroduodenoscopy. Proper preventive treatment, which greatly reduces the incidence, involves early control of gastric pH (to > pH 5) with antacids or histamine 2 receptor antagonists (ie, H₂ blocking agents). Accordingly, antacids or H₂ blocking agents should be instituted via the nasogastric tube, or H₂ blocking agents can be given intravenously.

Analgesia

Although full-thickness burns are insensate, partial-thickness burns or associated injuries can cause marked pain, which further intensifies the neurohumoral stress response and may worsen morbidity and mortality. Administering small amounts of

intravenous narcotics such as morphine sulfate, or partial narcotic agonists such as nalbuphine or butorphanol, will lessen patient discomfort and apprehension, as well as facilitate intravenous line placement and early wound care.

Wound Care

Several objectives are important in early care of combat burn wounds. Perfusion to the wound must be maintained to supply needed tissue-oxygen levels.²¹ This is accomplished by providing hemodynamic stability, restoring circulating blood volume, and performing escharotomy as needed. Remaining viable dermis must be protected in partial-thickness burns by initially leaving large blisters intact.^{22,23} Debris and devitalized tissue should be removed from full-thickness burns²¹ and the wound should be cleaned with soap and water. Since all burn wounds are invariably contaminated, topical antibiotics should be applied to the surface to temporarily retard bacterial growth and to help prevent burn wound infection.²²⁻²⁴ Topical chemotherapy may consist of sulfonamide cream, 0.5% silver nitrate solution, or mafenide acetate cream. Due to its solubility in water, mafenide acetate burn cream diffuses well into the burn eschar and may be the best choice for limiting bacterial proliferation.⁶

PATHOPHYSIOLOGY OF BURN INJURY

Pulmonary Pathophysiology

Derangements in pulmonary physiology accompany all major burns, even in the absence of inhalation injury. These pulmonary manifestations are a major cause of morbidity and mortality, and also clearly worsen the manifestations of inhalation injury. Early in the course of injury, pulmonary derangement is manifested by changes in lung capillary permeability, vasoreactivity, and ventilation. Surface burns cause an increase in pulmonary vascular resistance, paralleling the increase in systemic vascular resistance that occurs with hypovolemia. Subsequently, vasoactive mediators such as histamine, serotonin, and thromboxane A₂ are liberated from the burn wounds and further increase pulmonary artery pressures.²¹ Concomitant hypoproteinemia and complement activation, with subsequent white blood cell aggregation and deposition in the lung vasculature and release of proteolytic enzymes and oxygen free radicals, produce dramatic increases in lung vascular permeability. This

may produce the classic radiological and hypoxemic condition known as adult respiratory distress syndrome.²⁵ Hypoxemia is worsened by elevation of the closing volume (ie, the alveoli become unstable and collapse) and subsequent atelectasis.²¹

Ventilation is initially unchanged or decreased as a result of hypovolemia, concomitant increase in small-airway resistance, pain-induced splinting, or narcotic-induced hypoventilation.²¹ Ventilation may be severely depressed by electrical injury as a consequence of central nervous system depression or from large increases in total lung compliance from chest wall edema. If necessary, endotracheal intubation and mechanical ventilation should be instituted.

When adequate hemodynamics are restored, hyperventilation begins, with an increase in both tidal volume and respiratory rate. This increase in ventilation, which is secondary to the demands of hypermetabolism, peaks at the second week after the burn, subsequently declines to a level 2- to 3-fold greater than normal, and then persists until wound

closure has been effected. Hyperventilation is accentuated by several factors including the presence of topical mafenide acetate cream (which inhibits carbonic anhydrase activity), sepsis, fever, anemia, pulmonary parenchymal disease, pain, and anxiety.⁵

Hemodynamic Derangements

Early hemodynamic changes are due to deficits in blood volume that are related to transvascular loss of fluid, electrolytes, and protein. This hypovolemia causes tachycardia and, when pronounced, reduction of cardiac output. Both systemic and pulmonary vascular resistance rise in response to neurohumoral mediators, including catecholamines. Cardiac filling pressures decrease, which may further decrease the cardiac output. Perfusion to the periphery is ultimately decreased, but output to the viscera is maintained. A circulating myocardial depressant factor was previously thought to be liberated in burn patients but has not been definitively characterized to date. Positive pressure ventilation can superimpose a further reduction in cardiac output. With adequate fluid resuscitation, the cardiac output returns to normal by the end of the first 24 hours after the burn and then to supranormal levels thereafter. Hyperdynamic changes persist until the wound is closed.

Hematological Derangements

Hematological changes are manifested by early destruction, injury, and loss of red blood cells. As much as 8% to 19% of the red blood cell pool may be destroyed by heat. Subsequent loss secondary to wound debridement (as much as one unit every 3–4 d), phlebotomy for frequent laboratory studies, clearance of damaged red blood cells by the reticuloendothelial system, and decrease in hematopoiesis cause anemia that persists until the wound is closed.¹¹ In addition, free plasma hemoglobin and hemoglobinuria can be seen shortly after the burn.²¹ Early anemia is often masked by plasma-volume deficits that are proportionally greater than red blood cell losses. Maintenance of blood volume is important to minimize catecholamine release and subsequent catabolic stress,¹¹ since the hypermetabolic cardiovascular system is already stressed in attempts to provide oxygen delivery.

Other hematological changes include early leukocytosis and development of a hypercoagu-

lable state, probably secondary to activation of clotting factors in the burn wound.²¹ Platelet count and platelet adhesiveness increase for about 3 weeks.²⁶ Factors V and VIII increase to levels 4- to 8-fold greater than normal and remain elevated for 2 to 3 months. Fibrinogen levels are initially reduced, then return to normal within 36 hours, and finally remain elevated for as long as there is an open wound (eg, 3 mo²⁶). Fibrin split products may be elevated for the first 3 to 5 days. A massive burn may also be complicated by consumption of clotting factors, leading to a hypocoagulable state.²¹

Metabolism and Nutrition

After hemodynamic stability has been afforded by adequate resuscitation, a period of hypermetabolism ensues, with increased utilization of substrate, accelerated tissue breakdown, and depletion of lean body mass.²⁷ The metabolic rate rises in proportion to the extent of the injury and may be twice normal. In addition, heat production and skin and core temperatures rise secondary to central thermoregulatory changes. Protein stores are utilized via hepatic gluconeogenesis pathways to provide glucose for burn wound metabolism. Thus, increased energy substrate and nitrogen requirements must be met if large reductions in lean body mass are to be avoided. Energy requirements may exceed 2,000 kcal/m² body surface area, while nitrogen requirements may exceed 15 g/m².¹⁰ The goal should be to meet these nutritional demands as soon as possible, preferably as soon as the gastrointestinal tract is functioning normally. Providing carbohydrate needs will minimize the nitrogen losses. Fat, vitamins, minerals, and nitrogen will be required. The enteral route is preferred for feeding, as it is less expensive, physiologically closer to normal, and may be associated with fewer complications than parenteral nutrition.²⁸

Immunological Changes

The burn casualty's immune function is globally impaired. The mechanical barrier of the skin is destroyed and humoral factors are decreased, as are cellular immune responses including lymphocyte and neutrophil activity, and the reticuloendothelial system is impaired.⁵ In addition, casualties with thermoneutral burns exhibit bone marrow depression with variable leukopenia, particularly of lymphocytes.² Burn casualties, then, are particularly susceptible to infectious complications.

INHALATION INJURY

Deranged respiratory physiology is both the result of inhalation injury and the end-organ response to the generalized systemic insult. Inhalation injury is a major source of morbidity and mortality: 50% to 60% of all burn-related deaths are related to inhalation injury, with most being due to carbon monoxide intoxication.²⁹ Two important facts are associated with inhalation injury:

1. it is the single most important associated injury that contributes to thermal burn mortality,¹¹ and
2. the casualty may or may not have an associated thermal burn.

A high suspicion of inhalation injury should exist if the burn occurred in a closed space such as a vehicle or bunker, or if there are burns on the casualty's face or within the mouth or throat. Difficulty breathing, singed facial or nasal hair, changes in the voice, brassy cough, sooty or bloody sputum, or circumferential burns of the chest should alert the medical officer that inhalation injury is likely.³⁰ A radiograph of the chest is an insensitive, and sometimes a misleading, indicator of inhalation injury. Confirmatory diagnostic studies including fiberoptic bronchoscopy and xenon 131 lung scans are performed when available.

Inhalation injury has three components, each of which may occur independently or concurrently: (1) carbon monoxide intoxication, which is a consequence of incomplete combustion; (2) upper-airway injuries, which are caused by heat; and (3) lower-airway injuries, which are caused by chemical vapors or fumes.

Carbon Monoxide Intoxication

Carbon monoxide intoxication produces severe hypoxemia because carbon monoxide avidly binds to the hemoglobin molecule, forming carboxyhemoglobin, which displaces oxygen, resulting in decreased blood oxygen saturation. This desaturation is worse at the scene of injury as fire consumes much of the available oxygen. Carboxyhemoglobin also shifts the oxyhemoglobin dissociation curve to the left. The result is decreased oxygen delivery to the tissues, causing tissue hypoxia.^{31,32} Initial symptoms of carbon monoxide intoxication include palpitations, headache, dizziness, and confusion. Higher carboxyhemoglobin levels result in restlessness, excitement, and even unconsciousness

when the carboxyhemoglobin level rises above 40%.

Early administration of a high concentration of inspired oxygen is important in treating carbon monoxide intoxication. The half-life of carboxyhemoglobin is reduced from about 4 hours when breathing room air to 30 minutes when 100% oxygen is administered.¹¹ Treatment in a hyperbaric chamber has been reported³³ to be efficacious for severe carbon monoxide poisoning.

Upper-Airway Injury

Because the heat-carrying capacity of air is quite low, and hot vapors are rapidly cooled to body temperature as they pass through the nasopharyngeal area and upper airway, direct heat injury usually only affects the upper airway.³⁴ Heat injury below the level of the upper trachea is rare except with steam injury. Moreover, reflex closure of the glottis often protects the trachea and lung parenchyma.³⁵ Marked edema occurs in the mucosa within minutes to hours after the burn; ulceration and hemorrhage are also found in the mucosa. The edema may be worsened by large amounts of fluid given during resuscitation.³⁶ The airways of patients with these conditions should be protected immediately by intubation, even if only prophylactically, since the edema lasts several days and may make late intubation difficult or impossible. Orotracheal or nasotracheal intubation is preferable to tracheostomy because surgical airway manipulation is associated with high mortality.³⁷

Lower-Airway Injury

Damage to the lower airway is usually in the form of chemical tracheobronchitis resulting from the liberation of water-soluble gases from burning plastics and rubber. These gases—ammonia, phosgene, sulfur dioxide, and chlorine—cause epithelial damage when converted to acids and alkali on contact with airway mucosa. Lipid-soluble toxins—nitrogen oxides and aldehydes—are produced from burning wood and destroy lipid cell membranes and denature proteins. These toxins damage both alveolar and capillary endothelium, which causes increased permeability and pulmonary edema even at normal capillary filling pressures. Ciliary paralysis and severe bronchospasm also occur. The end result is plugged airways, obstructive and secondary atelectasis, relative hypoxemia, postobstruc-

tive emphysema, and occasionally complete lobar collapse.³⁵

Depending on the severity of the initial insult, patients with lower-airway injury progress through three clinical stages of injury. During the first 36 hours after the burn, acute pulmonary insufficiency occurs, characterized by hypoxemia, tracheobronchitis, bronchospasm, cough, atelectasis, and pronounced upper airway swelling. The second stage is pulmonary edema, which develops 6 to 72 hours after injury. Pulmonary edema is seen in 5% to 30% of patients and is associated with high mortality. The third stage is bronchopneumonia, seen in 15% to 60% of casualties, beginning 3 to 10 days after the burn. Bronchopneumonia is associated with 50% to 80% mortality.³⁸

Treatment

Treatment of severe inhalation injury involves endotracheal intubation, mechanical ventilatory support, supplemental oxygen, and aggressive tracheobronchial toilet including humidification, frequent suctioning, postural drainage, and repeated bronchoscopy. Antibiotics are instituted for culture-proven pneumonia. Systemic steroids have

been found to be of no value except to treat bronchospasm unresponsive to parenteral and inhaled bronchodilators.³⁵ These patients' high minute ventilation requirements and reduced pulmonary compliance increase their susceptibility to the complications of mechanical ventilators and place added stress on conventional ventilators. Ventilators that are capable of generating high minute volumes are invaluable for intensive care and operating room use for these patients. In addition, it appears that high-frequency percussive ventilation is effective in the treatment of patients with severe inhalation injury.³⁹

Increasing evidence indicates that oxygen-derived free radicals contribute to the pathophysiology of inhalation injury, especially in the period beginning 24 hours after exposure. Studies⁴⁰ carried out in a laboratory model of inhalation injury show that airway injury was markedly reduced by instillation of the iron-chelating agent deferoxamine complexed with pentastarch. Free iron, which is removed by deferoxamine, is known to catalyze the formation of such reactive species as the hydroxyl radical. Interventions based on these observations may have important therapeutic implications in human victims of smoke inhalation.⁴¹

CHEMICAL AND ELECTRICAL BURNS

Chemical burns to the skin and lungs, and the serious respiratory problems that arise when chemical warfare agents such as mustard and phosgene are inhaled, are described in Chapter 30, Anesthesia for Casualties of Chemical Warfare Agents. Chemical burns caused by acid or alkali have a component of injury caused by heat that is released when the chemical contacts the tissue. The severity of the burn is related to both the duration of exposure and the concentration of the chemical.¹⁴ In general, burns from strong alkalis are more severe than those from strong acids, since the alkalis bind more avidly to the tissues and are therefore more difficult to remove. Initial treatment lies in expedient removal of the chemical by copious water lavage of all exposed surfaces, after all the casualty's clothing has been removed. The prolonged use of cold water and subsequent hypothermia should be avoided, as this increases morbidity. Neutralizing agents should be avoided since their use may liberate more-intense heat. Severe chemical burns may appear deceptively superficial in the initial stages.⁵

Although electrical burns are uncommon among combat casualties, military anesthesiologists need to know about this entity, with its distinctive ap-

pearance and unique associated injuries, fluid requirements, wound care, and complications. Electrical injury is arbitrarily categorized into burns caused by low-voltage sources (< 1,000 volts) or high-voltage sources (> 1,000 volts). High-voltage burns are associated with substantial morbidity and mortality: of patients who survive to reach the hospital, mortality may approach 3% to 15%.⁴²

The tissue's resistance converts electromagnetic energy to thermal energy, which damages tissue.⁶ In addition, electrical injury may cause cardiac irritability or even asystole. Associated injuries are common, including bone fractures (from falls or severe, sudden muscular contraction); neurological damage, which may be progressive (since electrical current may flow through low-resistance neurovascular bundles); ruptured organs; or unexpected and widespread destruction of any soft-tissue structure.^{11,26} In fact, an unimpressive wound may overlie large areas of devitalized tissue; this may lead to underestimating the required resuscitation fluid. Muscle burned by electricity may progress to myoglobinuria and acute renal failure if additional fluids and diuretics are not administered to induce a brisk diuresis. This type of injury may cause

edema to occur beneath the investing fascia, which requires fasciotomy rather than escharotomy.⁶

Initial wound care involves removal of the patient from the electrical source. Unconsciousness, which necessitates intubation and mechanical ventilation, may result from high-voltage injury, as can seizure activity. Along with the thermal damage, electrical injury may cause cardiac arrest, requiring cardiopulmonary resuscitation at the scene. Further treatment involves hemodynamic stabilization; close monitoring; and early tissue exploration for

debridement, assessment of tissue viability, and determining the need for amputation.⁶

Complications, which may be unpredictable, include cataracts; recurrent gastrointestinal dysfunction with a high incidence of cholelithiasis, hemorrhage, or prolonged ileus; immediate or delayed spinal cord damage; peripheral nerve deficits; delayed hemorrhage from large vessels; and causalgia.^{6,26} The most common complication in the hospital is the need for major amputation, which occurs in 25% to 68% of patients.⁴²

FLUID ADMINISTRATION AFTER RESUSCITATION

Vigorous fluid resuscitation causes abnormal expansion of total body water and salt. Thus, the postresuscitation burn casualty's weight may substantially exceed his or her normal weight. A gradual loss of the perhaps 15% to 20% gain in body weight should be accomplished by a 2% to 3% weight loss per day, so as to reach preburn weight by the 10th day after the burn. This is accomplished by reducing the fluid rate by 25% per hour for patients who maintain adequate urinary output.¹⁰ Initial salt loading and subsequent high evaporative losses may predispose the burn patient to hypernatremia, so 5% dextrose in water is used to maintain serum sodium at 140 mEq/L.

Hypertonic Saline

The use of hypertonic saline as a component of the resuscitation fluid may be efficacious for patients in whom it is desirable to limit the total volume of resuscitation fluid. Hypertonic saline may cause less wound edema and may be particularly useful in patients with inhalation injury, circumferential full-thickness burns of the extremities, or intracranial injuries where massive fluid accumulation could be catastrophic.⁹ In addition, hypertonic saline may be desirable for patients with limited cardiopulmonary reserves (ie, the very old or the very young) and those with cardiopulmonary disease.¹⁰ However, no study has demonstrated that any particular sodium concentration is supe-

rior for improving survival in burn patients.⁹ Moreover, the critical factor in administering hypertonic saline is the development of hypernatremia; serious complications may result if serum sodium concentration rises above 160 mEq/L. Only very experienced clinicians should use hypertonic saline.¹¹

Colloids

During the first 12 to 24 hours after the burn, the diffuse capillary leakage allows colloid fluid to extravasate through the endothelial junction, preventing any demonstrable oncotic benefit over crystalloid fluid.¹¹ Thereafter, capillary integrity is re-established, and most resuscitation formulae include colloidal solutions as integral components of fluid therapy.

Diuretics

Diuretics are not included in burn resuscitation, except for a few special classes of patients. These include patients who have large, high-voltage electrical injuries; burns involving muscle; burns associated with large, soft-tissue injuries; and massive burns, who remain oliguric despite adequate fluids. Patients in this last category may be predisposed to acute renal failure secondary to large amounts of hemochromogens in the urine. A brisk diuresis is desirable in these patients.

EVACUATION OF THE BURN CASUALTY

Despite the implementation of Medical Force 2000, which focuses on enhancing far-forward surgical care, increasing intensive-care capability in the combat zone, and returning soldiers to duty as soon as possible, a large proportion of burn patients will be evacuated and transported. Forward surgi-

cal teams, field hospitals, combat support hospitals, and mobile army surgical hospitals will not be equipped to handle definitive surgical burn care. The extensive blood loss, larger surgical manpower requirements, and lengthy period required for skin-graft maturation would place too great a burden on

EXHIBIT 22-1**BURN INJURY EVACUATION CHECKLIST**

Patient Data	<input type="checkbox"/> Preburn weight <input type="checkbox"/> Preevacuation weight <input type="checkbox"/> Allergies <input type="checkbox"/> Medical history <input type="checkbox"/> Medications <input type="checkbox"/> Time of last oral intake <input type="checkbox"/> Vital signs chart
Laboratory Values	<input type="checkbox"/> ABG <input type="checkbox"/> Blood type <input type="checkbox"/> UOP <input type="checkbox"/> Electrolytes <input type="checkbox"/> CXR <input type="checkbox"/> Other
Burn Data	<input type="checkbox"/> % TBSA burn <input type="checkbox"/> Facial burn? <input type="checkbox"/> Airway: intubated/nonintubated? <input type="checkbox"/> Inhalation injury suspected? <input type="checkbox"/> Summary of early care <input type="checkbox"/> Locations, types of catheters and IV lines
Equipment	<input type="checkbox"/> Fluids: Ringer's lactate, D5H ₂ O, albumin <input type="checkbox"/> Airway management: ET tubes, masks, laryngoscopes, O ₂ , positive-pressure breathing circuit or mask-valve-bag device <input type="checkbox"/> Analgesics <input type="checkbox"/> IV administration sets

ABG: arterial blood gases; CXR: chest X ray; D5H₂O: 5% dextrose in water; IV: intravenous; ET: endotracheal; UOP: urinary output; TBSA: total body surface area

these military healthcare resources. Therefore, casualties will be evacuated to a general hospital with burn care capability and experienced personnel, or to a specialized burn care center. However, there are several associated risks and challenges.

The evacuation process may be accomplished very early in the course of injury, be lengthy, and occur when only minimal medical care can be provided. All these factors have particular impact on the burn casualty, since evacuation may occur while critical fluid requirements must be met, when tissue edema may compromise the airway or associated complicating factors (eg, inhalation injury; preexisting cardiorespiratory disease; blunt, penetrating, or blast trauma) may impact heavily on the burn casualty's medical management and survivability.

The anesthesiologist, who may be the last healthcare provider to see the casualty before the lengthy evacuation, can play a key role in reducing the burn casualty's morbidity and mortality.

The anesthesiologist must ensure adequate preparation and communication before evacuation. Preparation involves ensuring that all patient data and records are clearly documented and that adequate airway equipment, resuscitation drugs, and intravenous fluids are assembled. Communication must be clear to both the evacuation team and the receiving burn care team. These objectives are most efficiently accomplished by using a burn injury–evacuation checklist (Exhibit 22-1), which must be completed and included with the casualty.

PERIOPERATIVE CONSIDERATIONS

Early excision and grafting of small burn wounds is considered the norm. Since early excision and

grafting of large burns (> 30% TBSA) have been shown to decrease mortality in patients aged 17 to

30 years,⁴³ these surgical procedures are being accomplished earlier and earlier in the postinjury course. This may pose several challenges for the anesthesiologist, particularly if surgery is planned during the resuscitative phase or if associated injuries are present.

Preoperative Assessment

Preoperatively, the military trauma anesthesiologist should review the history of the current injury, including the amount of associated TBSA burn, elapsed time since injury, and associated traumatic injuries. Concomitant medical problems, chronic and recently instituted medications, family history, postsurgical and postanesthetic history, drug allergies, transfusion history, time since last oral intake, and presence of intravenous lines and invasive monitors should also be assessed. Current vital signs, including hemodynamic data from invasive monitors, should be noted along with present and preburn weight and laboratory data, including hematology, acid–base status, coagulation profile, liver function tests, and electrolytes. The casualty's electrocardiogram and a recent chest radiograph should be reviewed. A physical exam with emphasis on airway, pulmonary, and cardiovascular assessment is important. Evaluation of any concomitant trauma should proceed as indicated.

Preoperative Medications

While an individualized approach to each casualty should be used for preoperative medication, several goals should be met: anxiolysis, sedation, analgesia, amnesia, antisialagogue effect, elevation of gastric-fluid pH, and reduction of gastric volume.⁴⁴ Based on the patient's hemodynamic stability, degree of apprehension, discomfort, personal preference, and planned procedure, rational choices for premedications can be made.

With few exceptions, medications that are being administered before surgery are continued. In particular, medications that support hemodynamics (eg, inotropics, vasodilators, and antidysrhythmics) are continued, as are antibiotics and hyperalimentation fluid. In terms of sedation, critically ill patients may not tolerate depressant medications. An amnestic agent such as scopolamine may be given to these patients. Other seriously ill and intubated patients may be receiving narcotics, sedatives, and nondepolarizing muscle relaxants. If so, intravenous narcotics or benzodiazepines can be adminis-

tered to facilitate patient comfort during transfer and transport.

Patients who are more stable may be given small amounts of intravenous narcotics or benzodiazepines, or, for patients who prefer an oral premedicant, diazepam or lorazepam orally 1 to 2 hours before induction. For patients being given a ketamine-based anesthetic, a deeper level of sedation is provided in the form of oral diazepam 1 to 2 hours before induction, followed by intravenous diazepam or midazolam on induction. This reduces the incidence of undesirable reactions when the patient emerges from anesthesia. Glycopyrrolate is given for its potent antisialagogue effects if a ketamine anesthetic is planned, or if airway manipulation dictates a secretion-free field. The use of other antisialagogues is discouraged; owing to the crossing of the blood–brain barrier, they interact poorly with ketamine.

Owing to their tendency to form Curling's ulcers, most burn patients will be given an H₂ blocking agent or a sucralfate regimen. If so, this is continued perioperatively. If not, oral or intravenous ranitidine, famotidine, or cimetidine is administered. If the patient has been on particulate antacids, these are discontinued 6 to 8 hours before induction. For emergent surgery, an oral, nonparticulate antacid such as Bicitra (sodium citrate and citric acid, manufactured by Baker Norton Pharmaceuticals, Miami, Fla.) administered 15 to 30 minutes before induction is nearly 100% effective in elevating the gastric fluid pH above 2.5.⁴⁴ Metoclopramide speeds gastric emptying and is effective if given orally 30 to 60 minutes before induction; the effect is seen much quicker if administered intravenously.

Monitoring

Burn casualties can pose major challenges in placing and maintaining appropriate monitors. Adequate intraoperative monitoring is especially important considering the potential for extensive blood loss, the frequent changes of position, and the duration of surgery. The monitors that are considered essential for burn casualties are those that ensure the adequacy of oxygenation and ventilation, including continuous pulse oximetry, end-tidal carbon dioxide monitoring, peak airway pressure, and frequent arterial blood-gas analysis. Other routine intraoperative monitors should include a continuous electrocardiograph (ECG); an automated blood pressure cuff; esophageal, nasopharyngeal or rectal temperature-monitoring devices; inspired oxygen

tension–monitoring devices; a peripheral nerve stimulator; and precordial or esophageal stethoscopes. Lack of unburned tissue may necessitate placement of needle ECG electrodes, use of a “back-pack” ECG pad, or even an esophageal ECG lead. Pulse oximetry probes can be placed on any finger, toe, lip, tongue, ear lobe or helix, or ala nasi. Blood pressure cuffs may be placed on the upper arm, thigh, or calf, and may be sterilized and placed over burned or grafted skin intraoperatively, as needed.

For more critically injured patients and those with preexisting severe systemic disease, more-invasive intravenous lines or monitors are placed. A Foley catheter, arterial line, central venous catheter, or pulmonary artery catheter may be placed as indicated. Frequent arterial blood-gas and hematocrit determinations must be made.

Adequate intravenous access must be established before a major excision. This should include two large-bore peripheral intravenous lines or one peripheral and one central line. If lack of access necessitates placing an intravenous line through burned or grafted tissue, a meticulous sterile field must be maintained. Equipment for rapid infusion of crystalloid resuscitation fluid and blood should be at hand. Anesthesia providers may utilize a pneumatic pressure infuser powered by a pipeline oxygen source that can be regulated to achieve flow rates up to 250 mL/min. If pressure devices are used for intravenous infusions, care must be taken to ensure that air is not infused inadvertently.

Transport

Monitoring and therapy must not be compromised while critically ill, burn casualties are transported to and from the operating room. Appropriate preparation includes notifying the intensive care nurse well in advance of the need for organizing lines and tubes. The casualty is counselled again, reassured, and given additional premedication as the level of apprehension warrants. A mechanical lifting device can be used to move the patient from the bed to a stretcher. A portable oxygen tank and a disposable positive-pressure breathing circuit are used for ventilation, except when precluded by extremely high minute ventilation or elevated peak airway pressures. In these cases, a portable transport ventilator is used, accompanied by a respiratory therapist. A portable suction device should be available for casualties who require frequent suctioning.

The monitors considered routine for transport are a precordial stethoscope and a portable pulse

oximeter. A portable ECG monitor with an arterial line channel should be available for more unstable patients. Care must be taken with all lines and invasive monitors, which, if tenuous, should be sutured into place. A free-flowing intravenous line is imperative, since resuscitative drugs might be given during transport. Continuous-infusion devices are attached to the delivery pole on the stretcher, so that any cardioactive medications or hyperalimentation fluids can be continued.

Anesthetic Technique

The choices for the optimum anesthetic technique to use for the burn casualty are varied; however, rational decisions can be made based on known physiological responses to our interventions. Regional anesthesia, although desirable in certain cases, generally is avoided in burn casualties for several reasons:

- The burn may cover the site of anesthetic block placement, and it is undesirable to expose the patient to the risk of central neuraxis infection from colonized burn wounds via spinal or epidural needle placement.
- Excision or grafting procedures are accompanied by large fluid shifts and blood loss, and the loss of sympathetic tone resulting from a regional block may be undesirable.
- These casualties will likely undergo procedures at multiple excision and donor sites, including both upper and lower body sites at a single operation.
- In addition, an awake patient’s comfort is frequently difficult to maintain during lengthy excision and grafting procedures.

Induction and the Compromised Airway

Induction of general anesthesia is accomplished on the operating room table, except when the initial excision or graft harvest is on the patient’s posterior surface. In that case, induction proceeds on the stretcher, with subsequent positioning of the patient in the prone position. Choices for induction of general anesthesia depend on associated hemodynamic instability, presence or absence of intravenous access, and airway status. For the nonintubated patient, a combination of pre-oxygenation, midazolam, a narcotic, sodium thiopental, and a large dose of nondepolarizing muscle relaxant is frequently used. Although

ketamine has been advocated as a better induction choice in hypovolemic patients, this impression has not been proven. Etomidate is an effective induction agent that offers substantial hemodynamic stability, and is a good choice for patients when cardiac depression would be undesirable. While both ketamine and etomidate both support hemodynamics well, military anesthesiologists must be aware that maximally stressed patients may exhibit hemodynamic deterioration on induction due to some degree of myocardial depression or loss of sympathetic tone. Propofol is a satisfactory induction agent in young, physiologically stable burn patients.

Inhalation induction using halothane, isoflurane, or enflurane, with oxygen and nitrous oxide is another option. Small doses of sodium thiopental (1–2 mg/kg) may provide better tolerance for the irritating vapor. After induction, the ability to ventilate by bag is demonstrated. An intubating dose of a nondepolarizing muscle relaxant may be given at this time to improve conditions for airway instrumentation.

The burn casualty may well have a compromised upper airway, secondary to airway burns or to massive peripheral edema secondary to burns elsewhere. Upper-airway obstruction is a life-threatening complication that appears in the first 36 hours after injury.⁴⁵ Large volumes of resuscitation fluid may accentuate supraglottic edema, and concomitant facial and neck burns, constricting dressings, and edema make mask ventilation and airway instrumentation difficult. Later in the course of hospitalization, airway management may be compromised by limited neck extension or mandibular mobility. Lastly, airway management in the burn casualty, both early on and later in the course of treatment, may be difficult and be dictated by concomitant nonthermal trauma (eg, mandibular or facial fractures) that will require individualized approaches.

Several options exist for patients with a compromised airway. An awake intubation may be performed before anesthetic induction using topical airway anesthesia via a blind nasal approach, direct oral laryngoscopy, fiberoptic laryngoscopy, or a lighted stylet (light wand) technique. If airway difficulty is not anticipated, and a full stomach or predisposition to gastroesophageal reflux exists, a modified rapid-sequence induction using pre-oxygenation, intravenous induction using a large dose of nondepolarizing muscle relaxant, ventilation through cricoid pressure, and rapid intubation via direct laryngoscopy or light wand technique is

used. If the patient's airway is not compromised, and if the patient's stomach is empty, then an inhalation induction can be followed by the airway instrumentation of the anesthesia provider's choice. In situations where anatomical alterations suggest difficulty, alternate measures should be at hand, including the ability to perform emergent cricothyroidotomy, tracheostomy, or retrograde trans-laryngeal guidewire technique.

Maintenance of Anesthesia

No specific anesthetic agent is contraindicated in the burn patient except succinylcholine, which can trigger a potentially fatal hyperkalemic response and is discussed later in this chapter. A wide variety of anesthetic agents can be used satisfactorily. The burn patient's altered pharmacokinetic and pharmacodynamic responses, combined with the nature of the physiological insult and the type of surgery planned, dictate the choice of maintenance drugs.

Intravenous Anesthesia. Ketamine, a popular anesthetic for patients with thermal burns, produces sedation, profound analgesia, and a dissociative anesthetic state resulting from functional and electrophysiological disruption of the association between the thalamocortical and limbic systems.^{46–49} (The use of ketamine is also discussed in Chapter 10, Intravenous Anesthesia.) This drug has been described as the only available intravenous agent that can function as a sole anesthetic, without requiring adjunctive agents, owing to its unique combination of amnestic, analgesic, and anesthetic properties. Ketamine has particular application in the critically ill patient, where a period of hypotension or apnea could be life threatening.⁵⁰ Ketamine produces a dose-related rise in the heart rate–blood pressure product and supports hemodynamics through direct stimulation of central nervous system structures.⁵¹ Although ketamine does have some direct myocardial depressant and vasodilatory effects, it will help maintain blood pressure in hemorrhagic and septic shock.⁵² Ketamine maintains respiratory response to carbon dioxide (ie, the respiratory rate is proportional to the carbon dioxide level), precluding significant respiratory depression except when given in a rapid intravenous bolus of more than 3 to 4 mg/kg. Similarly, ketamine effects an increase in pulmonary compliance with a decrease in airway resistance and bronchospasm. This effect is probably mediated by vagolytic and direct, smooth-muscle, relaxant effects rather than through beta receptors. Oral and bronchial secre-

tions are stimulated by ketamine, necessitating pretreatment with an anticholinergic agent. While pharyngeal and laryngeal reflexes are preserved to some degree,⁵³ pulmonary aspiration of gastric contents can occur,^{54,55} and careful airway management is necessary.

Ketamine has been associated with anesthetic-emergence delirium, which occurs after 5% to 30% of surgeries performed under ketamine anesthesia.⁵⁰ This delirium is characterized by vivid dreams, hallucinations, dysphoria, and a sensation of floating in space. The incidence of this reaction can be decreased by (a) preoperative counselling concerning the expected side effects and (b) pretreatment with benzodiazepines. Oral diazepam, 0.15 mg/kg, administered 1 hour before induction, followed by 0.2 to 0.3 mg/kg intravenous diazepam, or 0.05 to 0.14 mg/kg intravenous midazolam, administered just before induction, is effective. Tolerance to the analgesic effects of ketamine occurs with repeated exposure to the drug.⁵⁶

Balanced Anesthesia. A narcotic-based, balanced anesthetic technique is an option for stable intraoperative hemodynamics, smooth emergence, and easily titratable levels of postoperative analgesia. The technique most commonly used at the U.S. Army Institute of Surgical Research Burn Unit consists of fentanyl, sufentanil, or alfentanil administered either in incremental boluses or by continuous infusion, in combination with oxygen, nitrous oxide, and low concentrations of inhaled vapor. Burn patients may have increased narcotic requirements.^{57,58} This may be due to both pharmacokinetic and pharmacodynamic factors,⁵⁹ but perhaps is due specifically to altered responses to the endogenous opioids that are actively released during the stress of thermal injury.^{60,61} In selected patients, morphine is also an attractive choice for use before emergence, because its slower elimination will effect longer postoperative analgesia. Respiratory depression, skeletal-muscle rigidity, and the development of tolerance with repeated use must all be considered with any narcotic.

Inhalational Anesthesia. The four currently used volatile inhalational anesthetics—halothane, isoflurane, enflurane, and desflurane—produce satisfactory anesthesia for burn casualties, and may have particular efficacy in those with inhalation injuries. The inhalation agents all act at various sites to prevent or reverse the bronchoconstriction that may accompany inhalation injury. The mechanisms of action are probably (a) suppression of airway reflexes and (b) direct, smooth-muscle relaxation (through action on the calcium flux in tracheal

smooth-muscle cells, which interferes with excitation–contraction coupling).⁶² To some extent, inhalational anesthetics reverse the hypermetabolic and hyperdynamic state of burn patients.⁶³ Both halothane and enflurane tend to decrease cardiac output and oxygen demand to approximately an equal degree, thus maintaining the oxygen supply-and-demand balance.⁶⁴ To date, halothane hepatitis has not been described in patients with burns, even after repeated exposure.⁶⁵

Some investigators have voiced their concern over the suppression of the immune response by the combination of major surgery and inhalational agents: bone marrow suppression by nitrous oxide⁶⁶; reduced phagocytic function by halothane; and reduction of lymphocyte reactions to antigens.⁶⁷ However, most researchers believe that depressed immunocompetence is not caused by the anesthetics but is the effect of the stress of the surgery itself.⁶⁸

The potential exists that volatile anesthetics will interact with topical epinephrine that has been applied to the exposed capillary bed following burn wound excision to help achieve hemostasis. Halothane, and to a lesser extent isoflurane and enflurane, do sensitize the myocardium to both endogenous and exogenous catecholamines.⁶⁸⁻⁷⁰ In fact, plasma catecholamine levels up to 10-fold higher than normal have been measured in such circumstances.⁷¹ However, various studies show minimal changes in cardiovascular parameters and an absence of arrhythmias after topical epinephrine during burn surgery.^{72,73} This apparent normalcy may be due to the preexisting, injury-induced, elevated level of circulating catecholamines that burn patients have, which leads to a decrease in the number of receptors and a downregulation of the receptor affinity.^{71,74-76}

The burn patient's response to neuromuscular blocking agents is perhaps the most striking of all altered drug responses, and is the most familiar to anesthesiologists. As was mentioned earlier, these patients can have a potentially fatal hyperkalemic response to depolarizing muscle relaxants (ie, succinylcholine). The release of massive amounts of potassium by muscle cells is related to the dose, the extent of burn injury, and the time elapsed after the burn.⁷⁷ Succinylcholine, then, may be used safely in the first 24 hours after the burn but, in a patient with a burn exceeding 10% TBSA, *should be avoided for the subsequent 18 months*.⁷⁸ The mechanism of action is an increased sensitivity of the muscle membrane secondary to a proliferation of extrajunctional receptors, similar to that seen in patients with degenerative neurological disorders.⁷⁸

This response begins within days of the burn and may persist for as long as 18 months after the burn.⁷⁹ If succinylcholine is inadvertently administered, then calcium chloride, a therapeutic mixture of glucose and insulin, and sodium bicarbonate should be administered, along with cardiopulmonary resuscitation, if cardiac arrest results. Calcium chloride should be subsequently continued until peaked T waves in the electrocardiographic tracing return to normal.⁵⁹

Resistance to nondepolarizing muscle relaxants has been well documented with respect to anesthetics such as metocurine,^{79,80} pancuronium,^{81,82} alcuronium,⁸³ *d*-Tubocurarine,^{84,85} and atracurium,^{86,87} and is likely to be encountered with all competitive agents.⁸⁶ (This topic is also discussed in Chapter 11, Neuromuscular Blocking Agents.) The mechanism of this resistance may be alterations both in the pharmacokinetics of the relaxant and at the neuromuscular junction. Pharmacokinetic factors may involve increased plasma-protein binding, depressed hepatic metabolism, and changes in blood flow that cause lengthened equilibration time between the relaxant in the plasma and in the neuromuscular junction, causing an artifactual rightward shift in the dose-response curve. Possible alterations at the neuromuscular junction include an increase in the number of acetylcholine receptors, decreased cholinesterase levels, or some type of prejunctional pathology. Clinically, this resistance is manifested as an increase of 2- to 5-fold in the requirements for both dosage and serum concentration over that of patients who are not burned. Despite this resistance, there appears to be no prolongation of recovery time or difficulty with reversal of the relaxants.⁸⁸ The altered response to nondepolarizing agents has been shown to persist for as long as 463 days.⁸¹

Intraoperative Fluid Management

Casualties with burns pose particular challenges with respect to fluid management. Thermal injury necessitates aggressive, early fluid resuscitation, and, for reasons unconnected to the burn, the casualty may require surgery and anesthesia at any phase of resuscitation. Continued derangements in fluid and electrolyte balance continue in the postresuscitative phase.

Intraoperative fluid therapy for excision and grafting procedures involves very rapid infusion of crystalloid, colloid, and packed red blood cells. The loss of one or more blood volumes is not unusual in a major tangential excision. Pressure infusion de-

vices are necessary, as are efficient blood- and fluid-warming devices.

Thermally injured patients frequently present for anesthesia and surgery with anemia that is caused by heat-induced lysis of red blood cells, reduced erythropoiesis, or frequent blood drawing. Early in thermal injury, anemia may be masked by a loss of plasma volume that exceeds the loss of red blood cells. Transfusion of red blood cells may therefore begin before excision if extensive blood loss is anticipated. Moreover, the adequacy of tangential excision is demonstrated by a briskly bleeding capillary bed. Large amounts of blood may be lost before hemostasis, and estimating this loss is difficult because surface oozing is difficult to collect in a suction device and the blood ends up in soaked gauze; on the drapes; or on the floor, sometimes under the operating room table. Visual estimation, heart rate and blood pressure, urinary output, and perhaps central venous pressure or cardiac filling pressures, all help guide the need for blood replacement. Hematocrit measurement made while blood loss is ongoing is of questionable value.

Because of associated potential risks, including disease transmission and the risk of transfusion reaction, homologous blood transfusion today should be justified only by the need for increased oxygen delivery. However, the burn patient may require a higher hematocrit than a patient who is not burned, secondary to elevated oxygen delivery requirement in the face of hypermetabolism, inability of an already stressed cardiovascular system to compensate for anemia, and reduced hematopoiesis.⁸⁹ While maintenance of hematocrit values in the low to mid twenties may be adequate for an otherwise physiologically healthy burn patient, the presence of concomitant physiological impairment may effect a need for a somewhat higher target hematocrit (ie, in the range of 0.30–0.35).

Temperature Regulation

Paradoxically, hypermetabolic patients whose central thermoregulatory set point is elevated are especially susceptible to hypothermia during anesthesia and surgery. Thermally injured patients have a propensity for abnormal heat loss secondary to the burn itself, the nature of the surgical procedure, the temperature of the operating room, and general anesthesia. Burn injury accentuates heat loss of all kinds, especially evaporative loss, which accounts for most of the difference in heat loss between burned and unburned patients.⁹⁰ Losses from conduction and convection are also increased⁹⁰

since considerably large areas of body surface are often excised or prepared for donor sites, and large areas of body surface are exposed in the operating room.

General anesthetics cause increased heat loss via several mechanisms. Dry anesthetic gases cause evaporative water and heat loss from airways. Anesthetics cause a loss of ability to preserve core temperature through cutaneous vasoconstriction and shivering. Specifically, inhalational agents cause a 15% decrease in the basal metabolic rate,⁹⁰ promote surface blood flow, and promote hypothermia through effects on central thermoregulatory centers.⁹¹ A narcotic–nitrous oxide anesthetic decreases metabolic oxygen uptake (V_{O_2}) by 50%, while ketamine has been shown not to alter V_{O_2} .⁹² In addition, neuromuscular blocking agents prevent shivering and lower the metabolic rate.

Decreases in core temperature produce several intraoperative and postoperative problems. Hypothermia can depress cardiac output, induce dysrhythmias, abolish hypoxic pulmonary vasoconstriction, shift the oxyhemoglobin dissociation curve to the left, and depress hepatic and renal functions.⁹³ A clinical impression is that increased intraoperative blood loss occurs secondary to poor hemostasis from exposed vascular beds following excision or donor harvesting if core temperature is less than 36°C. Hypothermia also causes postoperative shivering, which increases V_{O_2} to 300% to 400% of normal, may cause graft shearing, and can make the patient very uncomfortable.

To prevent intraoperative hypothermia, the patient should be covered with a space blanket while being transported. The operating room is warmed to 25°C to 30°C and humidified to 50% to 60%

relative humidity. All solutions for preparation of the skin are warmed, as are all crystalloid and colloid solutions for infusion. Anesthetic gases are warmed and humidified. A warming blanket is placed under the patient, all nonoperative sites are draped, and portable warming lights are used.

Postanesthetic Considerations

Major postanesthetic considerations include the need for a smooth emergence from anesthesia, the decision whether to extubate, the provision of comfort in the postoperative period, and the need for adequate physiological monitoring. A smooth emergence from anesthesia is desirable, since patient movement causes shearing of freshly placed grafts. Adequate pain control is necessary for this, and extubation while the patient is deeply anesthetized, followed by gradual emergence in the intensive care unit or recovery room, may be appropriate in selected patients.

Many burn patients will remain intubated postoperatively secondary to the need for continued mechanical ventilation, continued airway protection, or pulmonary toilet. Others may be extubated, provided that they are awake, able to follow commands, have adequate ventilation and oxygenation, have had adequate reversal from neuromuscular blockade, and are warm.

Transport to the recovery room should be undertaken with the precautions discussed previously. Adequate analgesia should be afforded via intravenous narcotics. If the anesthetic was ketamine-based, a quiet recovery area and adequate reassurance may reduce the incidence of undesirable emergence delirium.

SUMMARY

Combat casualties with burns are likely to have, in addition, penetrating missile wounds. This fact is a consequence of modern warfare, in which most soldiers who sustain burn injuries are wounded within armored fighting vehicles and are therefore subject to injury from fragments arising from the antiarmor warhead, in addition to the secondary explosives that may accompany battle damage. Thus, military anesthesia providers are frequently faced with the problem of treating casualties with multiple traumas, one of which is a cutaneous burn. In addition, we must maintain heightened vigilance for the possibility of coexisting inhalation injury in casualties who have sustained burn injuries within the closed confines of tanks and warships. Assum-

ing that there is no evidence of airway compromise, initial care of the burn casualty focuses on fluid resuscitation. During the first 24 hours, the essential component of fluid resuscitation is the Modified Brooke Formula (ie, the intravenous infusion of 2–4 mL Ringer's lactate/kg body weight/% TBSA burned). The adequacy of fluid resuscitation must be monitored frequently by observing such indicators of the normalcy of peripheral perfusion as the urinary output.

Anesthesia may be required during several different phases of burn wound care. Casualties with full-thickness burns of the extremities in which the peripheral circulation is compromised, or in whom thoracic excursion is hindered, will require escha-

rotomy. This procedure can usually be performed without anesthesia. When a substantial cutaneous burn is present (> 30% TBSA), the initial wound care is best carried out with a narcotic-based, balanced anesthesia using a low concentration of inhaled vapor. Initial wound care of lesser burns can be carried out with intravenous ketamine. Ketamine

can also be used for subsequent wound management in some casualties. Those casualties who require general endotracheal anesthesia at 24 hours or more after the burn injury should not receive succinylcholine because of its recognized propensity to cause massive release of potassium and possible sudden death from hyperkalemia.

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