Burns

45

Sara A. Mansfield and Larry M. Jones

Introduction

It has been estimated that over 1.2 million people in the USA suffer burn injuries every year [1]. Most are treated as outpatients, yet nearly 60,000 require hospitalization. Approximately 5000 of these patients die. Advances in resuscitation and monitoring techniques, wound management, nutritional support, and strict use of tissue culture-directed antibiotics have all contributed to decreases in morbidity, mortality, and long-term disability for these patients.

Initial Evaluation and Resuscitation

The initial evaluation of the burn patient should follow the same basic principles as the evaluation of any patient who has suffered traumatic injury. The ABCs of resuscitation, as taught in the American College of Surgeons Advanced Trauma Life Support Course [2], provide the foundation for prompt recognition of life-threatening injuries, stabilization, and resuscitation from shock.

The first step in the resuscitation of a burn patient is the establishment of a patent airway. Patients suffering burns while confined in an enclosed space, such as a burning building or automobile, must be evaluated for the presence of upper airway thermal injury, smoke inhalation injury, and carbon monoxide (CO) poisoning. Signs and symptoms of upper airway thermal injury include hoarseness, soot in the oropharynx and hypopharynx, singeing of the face and facial hair, and stridor. The upper airway should be inspected and, if any airway compromise is encountered, the patient should be quickly intubated. The airway inspection may be aided by the use of fiber-optic laryngoscope with the endotracheal tube positioned over the scope. If soot or vocal cord swelling is observed, the endotracheal tube may be left in proper position as the scope is removed. If there is a chance that the patient may lose airway patency, endotracheal intubation should be performed. This maneuver may be performed easily during the early stages of resuscitation but may become impossible with ongoing vocal cord swelling.

Smoke inhalation injury, which actually constitutes a chemical burn of the lower airways and lungs from noxious products of combustion in inhaled smoke, is heralded by carbonaceous sputum as well as many of the signs and symptoms of upper airway thermal injury. Direct airway injury results in sloughing of the tracheobronchial mucosa, impaired mucociliary clearance, and distal bronchial tree occlusion. This type of damage can induce an inflammatory cascade, which begins with neutrophil chemotaxis and oxygen free radical release and ends in microvascular permeability and pulmonary edema, culminating in pneumonia, acute lung injury, and acute respiratory distress syndrome (ARDS). Treatment should consist of mechanical ventilatory support using well-described lung-protective strategies [3] and positive end-expiratory pressure. An alternative ventilation strategy that has received particular attention in the smoke inhalation injury population is high-frequency percussive ventilation (HFPV). Oxygen is delivered by high-frequency subtidal breaths combined with intrapulmonary percussion and convective washout of carbon dioxide. Proponents point out that HFPV confers a unique mucokinetic effect that facilitates clearance of secretions. Several studies have demonstrated the benefit of this technique in smoke inhalation patients. However in a prospective randomized HFPV was not significantly better than low tidal conventional ventilation strategies when comparing

S.A. Mansfield, MD
Department of Surgery, The Ohio State University,
395 West 12th Avenue, Rm 654, Columbus, OH 43210, USA
e-mail: sara.mansfield@osumc.edu

L.M. Jones, MD (✉)
Department of Surgery, The Ohio State University
Wexner Medical Center, N748 Doan Hall, 410 West 10th Avenue,
Columbus, OH 43210-1228, USA
e-mail: larry.jones@osumc.edu

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J.M. O'Donnell, F.E. Nácul (eds.), *Surgical Intensive Care Medicine*, DOI 10.1007/978-3-319-19668-8_45

ventilator-free days, overall mortality, and ventilator-associated pneumonia. HFPV did provide better early oxygenation especially in inhalation injury patients. HFPV may be more "lung-protective" than low tidal volume (LTV) when considering the 13 % incidence of barotrauma over 28 days compared with 0 % in the HFPV arm [4].

An adjunct to the above ventilation strategies is the use of inhaled anticoagulation regimens (heparin, anti-thrombin, fibrinolytics). During smoke inhalation injury, obstructive casts can occur in the bronchioles consisting of cellular debris, fibrin, leukocytes, mucus, and others which can contribute to pulmonary failure [5]. A systematic review supported the safety of inhaled anticoagulation, without altering systemic markers of clotting. These regimens were also associated with a favorable effect on survival [6]. This strategy has not been widely adopted yet, with larger randomized trials still needed [7].

Bronchoscopic exam of the airways within 24 h of admission can provide useful information regarding degree of airway injury. The grading system of inhalation injury (Table 45.1) has been found to correlate with early physiologic changes and progression to ARDS, especially in Grades 2 and 3 [8]. Overall outcomes are worse with higher grade injuries [9]. Bronchoscopy may also have therapeutic advantages likely related to lavage and pulmonary hygiene. Patients undergoing bronchoscopies had shorter durations of mechanical ventilations and shorter ICU stays [10]. Smoke inhalation injury in association with a cutaneous thermal injury greatly increases the incidence of both nosocomial pneumonia and mortality [11, 12].

CO poisoning is the cause of death in most fires. A normal product of combustion, CO, is transported very rapidly across the alveolar membrane, where it competitively displaces oxygen from hemoglobin. An arterial carboxyhemoglobin level above 15 % indicates significant exposure. Signs and symptoms of toxicity correlate with an increasing level, and range from nausea and headache (15–25 %) to stupor (30–40 %) and finally coma and death (40–60 %). The use of 90–100 % oxygen reduces the half-life of CO from 4 h to 40 min. Studies evaluating outcomes using hyperbaric oxygen treatment have been mixed [13–15]. According to a 2011 Cochrane Review, existing studies do not demonstrate a benefit of hyperbaric oxygen in CO poisoning [16]. Large randomized control trials are still needed to address this issue. Delay in transporting the burn patient to an organized burn unit in favor of hyperbaric oxygen treatment should be avoided.

Another common product of combustion is hydrocyanide (CN). Like CO, CN is rapidly transported across the alveolus, where it binds to the cytochrome system and inhibits oxidative phosphorylation and adenosine triphosphate production. Treatment consists of administering 20 cc of 3 % sodium nitrate followed by 50 cc of 25 % sodium thiosulfate.

A major breakthrough in the management of severe burns was the work performed by Cope and Moore [17] and Baxter and Shires [18, 19]. Their classic articles on fluid distribution in and resuscitation of burn victims have been the foundation upon which modern fluid therapy is based. From those reports comes the Parkland formula recommendation of 4 cc Ringer's lactate solution per kilogram body weight per percent total body surface area burned. One-half of this calculated amount is administered over the first 8 h and the remaining calculated volume is administered over the next 16 h. However, this calculation should be used only as a guide to initial resuscitation fluid administration rates, and the patient's response to these measures must be closely monitored. In recent years, it has become apparent that many patients require fluid volumes well in excess of initial Parkland calculations, a phenomenon christened "fluid creep" by Pruitt [20]. Although it is recognized that burn patients with associated inhalation injury, high voltage electrical trauma, or delayed initiation of fluid resuscitation usually require more fluid than expected, questions continue to arise regarding the accuracy of the Parkland Formula in patients without these associated conditions. At least partly,

Table 45.1 Bronchoscopic gradation of inhalation injury

Grade	Description	% with ARDS within 24 h
0—no injury	Absence of carbonaceous deposits, erythema, edema, bronchorrhea, or obstruction	0 %
1—mild	Patchy erythema, carbonaceous depositis in proximal or distal bronchi	22 %
2—moderate	Moderate erythema, carbonaceous depositis, bronchorrhea, with or without compromise of bronchi	57 %
3—severe	Severe inflammation with friability, copious carbonaceous deposits, bronchorrea, bronchial obstruction	80 %
4—massive	Evidence of mucosal sloughing, necrosis, endoluminal obliteration	Approximately 100 %

Adapted from Mosier et al. [8]

ARDS acute respiratory distress syndrome

these trends seem to be related to inaccurate use of the Parkland Formula [21]. Regardless of the cause, a trend towards higher ventilator days and incidences of abdominal compartment syndrome are seen with higher resuscitation volumes [22]. Strategies to reduce fluid creep include adherence to protocols for fluid resuscitation, the avoidance of early overresuscitation, and use of colloid as a component of resuscitation or for "rescue" [23, 24].

Traditionally, urine output has been taught as the gold standard for measuring resuscitation response. However, urine output alone cannot accurately measure the adequacy of resuscitation [25]. Other parameters that must be measured as baseline indicators of response are blood pressure, pulse pressure, pulse, arterial oxygenation, and pH. In larger burns with major fluid shifts, the use of a Swan-Ganz catheter should be considered [26].

A great deal of investigation has been directed at determining proper endpoints of resuscitation. In a report by Rutherford et al. [27], base deficit was shown to be a sensitive measure of degree and duration of inadequate perfusion and, therefore, adequacy of resuscitation. Before that report, Abramson et al. [28] showed the importance of reversal of anaerobic metabolism, as measured by serum lactate clearance, on survival following trauma. However, burn patients were excluded from both studies. Several reports on burn resuscitation endpoints were published in 1997. In a report by Barton et al. [29], patients with a mean burn size of 45 % were resuscitated using the Parkland formula with additional fluid boluses and dobutamine infusions. Those investigators found that cardiovascular function in burn patients responded to volume loading and inotropic support just as in patients suffering shock due to other reasons. Jeng et al. [30] studied serum lactate and base deficit as measures of burn resuscitation and concluded, "Serum lactate and base deficit might just be the more precise physiologic yardsticks that are required to advance the state of resuscitation from burn shock." An additional report by the same author suggested that serum lactate was predictive of mortality, but neither lactate nor base deficit were reliable indicators as endpoints for resuscitation from burn shock [31]. In a report published by Kaups et al. [32], base deficit was shown to be superior to the Parkland formula for calculating fluid requirements, with a base deficit of −6 or less being a marker of increased mortality. However, this observation has not been borne out in other published studies [33, 34]. Clearly, more work must be performed in this area to define proper resuscitation endpoints.

Wound Management

The initial management of the burn wound involves stopping the burning process. This is accomplished simply by removing all burned clothing and cooling the burned areas in cool (20 °C) water. However, the effect of this cooling is lost after about 30 min. Indeed, cooling large burns with this method for prolonged periods of time results in heat loss and the adverse effects of hypothermia. Patients should never be cooled with ice or dry ice.

The extent and depth of the burn wound must be estimated to determine further care. While superficial (first-degree) burns are painful, they require little treatment other than symptomatic management with nonsteroidal anti-inflammatory drugs. When calculating the surface area, only burns of partial thickness (second degree) or deeper are included. This is best accomplished through the use of a Lund–Browder chart (see Fig. 45.1). However, if unavailable, the physician can estimate the size of the burn because the palmer surface of the patient's hand represents about 1 % of the patient's body surface. When size estimation is complete, the patient is weighed and the initial intravenous (IV) fluid rate is calculated. The elapsed time from infliction of the burn to the initiation of fluid resuscitation, along with the total amount of fluid the patient may have received before the initiation of resuscitation, must both be considered when performing the initial calculation.

Superficial second-degree burns are debrided of blisters and washed with a mild, non-deodorant soap. These burns are then covered by either a topical agent, such as 1 % silver sulfadiazine (see Table 45.2), or a barrier dressing such as one of the many silver-barrier dressings or Biobrane™ [35]. If Biobrane™ is chosen, it can be held in place with wound closure strips. It must be inspected carefully for the first few days following application for underlying fluid collections. If observed, they may be drained by opening the bubble over the collections. If underlying infection develops, the barrier should be removed and topical agents applied.

Systemic antibiotics are not indicated as a prophylactic measure. They should be reserved for use in cases of systemic infection, and then should be culture-directed with as narrow a spectrum as possible. Wound biopsies should be taken on at least a weekly basis and sent for quantitative culture, and sensitivities to common topical agents also should be reported. Research into the use of prophylactic antifungals is currently being conducted, but no final results have yet been reported.

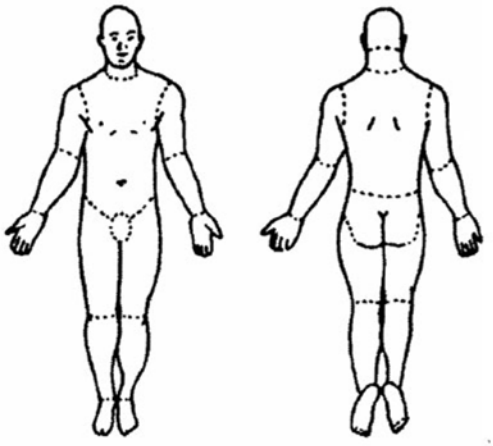
Full-thickness (third-degree) burns require skin grafting in most cases. Even grafting of deep partial-thickness burns should be considered in cases in which hospitalization and return to productivity would be enhanced. The timing of this surgical procedure comes after the full resuscitation of the patient. If the wound cannot undergo a grafting procedure by the third post-burn day, then wound cultures should be taken to assure colony counts of less than 10^5 organisms per gram of tissue. The ambient air temperature in the operating room should be increased to 30 °C (85 °F) for patients with large burns and those who are totally exposed during the procedure.

BURN ESTIMATE AND DIAGRAM
AGE VS AREA

Initial Evaluation

Cause of burn _____
Date of burn _____
Time of burn _____
Age _____
Sex _____
Weight _____
Date of admission _____
Signature _____
Date _____

Burn Diagram



Area	Birth 1 yr	1-4 yrs	5-9 yrs	10-14 yrs	15 yrs	Adult	2°	3°	Total	Donor Areas
Head	19	17	13	11	9	7				
Neck	2	2	2	2	2	2				
Ant. Trunk	13	13	13	13	13	13				
Post. Trunk	13	13	13	13	13	13				
R. Buttock	2 1/2	2 1/2	2 1/2	2 1/2	2 1/2	2 1/2				
L. Buttock	2 1/2	2 1/2	2 1/2	2 1/2	2 1/2	2 1/2				
Genitalia	1	1	1	1	1	1				
R.U. Arm	4	4	4	4	4	4				
L.U. Arm	4	4	4	4	4	4				
R.L. Arm	3	3	3	3	3	3				
L.L. Arm	3	3	3	3	3	3				
R. Hand	2 1/2	2 1/2	2 1/2	2 1/2	2 1/2	2 1/2				
L. Hand	2 1/2	2 1/2	2 1/2	2 1/2	2 1/2	2 1/2				
R. Thigh	5 1/2	6 1/2	8	8 1/2	9	9 1/2				
R. Leg	5	5	5 1/2	6	6 1/2	7				
L. Leg	5	5	5 1/2	6	6 1/2	7				
R. Foot	3 1/2	3 1/2	3 1/2	3 1/2	3 1/2	3 1/2				
L. Foot	3 1/2	3 1/2	3 1/2	3 1/2	3 1/2	3 1/2				
						Total				

Fig. 45.1 Lund Browder diagram with body surface area calculations

The planned procedure should not require more than 2 h to complete. Blood products for transfusion must be readily available before the start of debridement of a large burn and warmed during administration.

Several technologic advances have been made in the areas of wound management. Discussion of the numerous types of dressings and temporary closures is beyond the scope of this chapter.

Pain Management

Arguably, burns are the most painful injury a patient can experience. Control of pain is important, not only for humanitarian reasons, but also to decrease the patient's stress and inefficient use of calories. Full-thickness burns, in which all elements of skin including nerves have been destroyed, are

Table 45.2 Topical agents

Agent	Actions	Indications/considerations	Side-effects
Silver sulfadiazine	Broad spectrum, painless, minimal eschar penetration	Any size or depth of burn for prophylaxis or infection control	May cause leucopenia or skin allergy; creates pseudoeschar on wound
Mafenide acetate	Broad spectrum, effective against Pseudomonas, penetrates eschar, may be painful	Any size or depth of burn, use on burns of the nose or ears	Carbonic anhydrase inhibitor, may cause skin allergy. Available as cream or 5 % solution; fewer side effects with solution
Bacitracin/ polysporin ointment	Limited anti-bacterial coverage, no eschar penetration	Superficial burns resistance; often used on superficial burns of the face	Watch for bacterial resistance
Gentamycin ointment	Broad spectrum, painless, good eschar penetration	Gram-negative organisms resistant o other agents	Watch for bacterial resistance; renal and ototoxicity when used in large amounts
Povidone-iodine ointment	Broad spectrum, patients may complain of pain	Infected granulation tissue, organisms resistant to other topicals	May impair wound healing
Collagenase	Enzymatic debridement of eschar, no anti-bacterial properties; may mix with polysporin powder	Enzymatic debridement when surgical debridement may not be possible	Should not be used with compound containing metal ions such as povidoneiodine or silver
Silver barrier dressings	Antibacterial actions by release of silver ions. Broad spectrum	Good for temporary coverage of any size, infection control	Silver is not absorbed systemically; keep moist; dressing may remain intact for up to 7 days before changing

generally painless before grafting. However, partial-thickness burns are quite painful. Pain control must be initiated before any manipulation of the wound.

Generally, morphine or fentanyl is used for manipulations such as dressing changes. Smaller burns may be managed with less potent analgesics. Larger burns, however, should be treated with IV injections for a more reliable dose titration. Patients who are taking oral medications may find an increase in their pain threshold through the use of methadone. The initial dose of 5 mg is administered every 8 or 12 h. Dosing and frequency may be adjusted depending upon the patient's response. Several authors have reported on the use of ketamine for the management of burn pain [36–38]. In the setting of appropriate Registered Nurse (RN) training, RN administered sedation is also a safe and effective option [39].

Pain management of burns in pediatric patients can be challenging. Brown et al. [40], at the Shriners Burns Institute in Cincinnati, have reported that haloperidol is safe and effective for use with pediatric patients. Humphries et al. [41], who used the Ramsay scale as a measurement, found oral ketamine to be superior to narcotics and sedatives in this clinical setting.

There is little consensus regarding pain management among the various studies. Indeed, it has been reported that health care providers consistently undertreat pain from burn wounds [42]. In response to this, guideline-based approaches to pain management have been proposed by Ulmer [43] and Sheridan et al. [44].

Metabolic Response and Nutrition

Thermal trauma results in marked hypermetabolism and hypercatabolism [45]. Aggressive nutritional support is required to meet these metabolic demands and prevent nutritional collapse. Metabolism increases proportionally with size of burns to as much as 2.5 times normal. Early nutritional support is ideal to prevent autocannibalism of lean body mass [46].

Waxman [47] reported that nitrogen is lost from the wound at a rate of 0.1 × body surface area × percent burn per 24 h. When the burn wound is closed, the patient's metabolic rate returns to normal [48, 49]. Until that time, the nutritional needs of the patient must be intensely supported. Appropriate nutritional support is accomplished by assessing the patient's nutritional status, assessing the caloric and nitrogen requirements of the patient, formulating a plan and approach to each component, determining the modes to accomplish the nutritional goals, and monitoring the nutritional plan and any complications [50]. Early excision and coverage with adequate nutritional support has been shown to abate catabolic effects as well as infectious complications [51, 52].

The use of anthropomorphic measurements, plasma protein levels, and immune competence are of no real value in assessing the nutritional state and requirements of the burn patient. In 1919, Harris and Benedict described their formula

for estimating basal energy expenditures (BEE) in humans. Since then, other formulas including the Curreri and Galveston formulas have been devised. Today, many centers rely on indirect calorimetry to estimate caloric requirements. Despite the numerous formulas and methods, it does not appear necessary or prudent to provide more than two times the patient's BEE.

Calories are provided as 30–40 % fat, and the remainder of the diet is carbohydrates. When providing carbohydrate (especially via parenteral route), it is important not to exceed the maximum rate at which glucose can be assimilated in the body (7 g/kg/day) so as not to provide glucose in excess of the rate at which it can be oxidized. Fat intake is important to prevent free fatty acid deficiency and binding of fat-soluble vitamins. Providing adequate amounts of protein postburn is essential as protein stores are depleted for energy usage and muscle tissue is broken down at rates of up to 150 g/day. The nitrogen losses of severely burned patients can amount to 20–25 g/m^2 total body surface area (TBSA)/day leading to detrimental muscle loss [53]. Exact formulations should be made on a patient-by-patient basis.

Multivitamins, particularly vitamin A for epithelial integrity, vitamin C for collagen synthesis, and vitamins E and B for wound repair, are commonly administered. Zinc is also recommended because of its role in wound healing [54]. Nordlund et al. [55] provide a good overview of micronutrient supplementation and current evidence of each.

In addition to increased nutritional support for the hypermetabolic, catabolic state, the efficacy of modulating these states has been studied extensively. Oxandrolone, a testosterone analog, has been shown to decrease hospital stay in severely burned patients [56], postulated to be because of, in part, its anabolic effects of increasing lean body mass in burn patients [57]. Insulin has been shown to increase skeletal muscle synthesis in severely burned patients, even at submaximal doses [58]. Beta-blockade with propranolol has also been shown to decrease energy expenditure and increase net muscle protein balance [59, 60].

Several aspects of the effects of glutamine supplementation have been recently studied. Severely burned patients experienced decreased hospital stay [61]. In addition, decreases in gram-negative bacteremia, particularly *Pseudomonas* species, and ICU stays have been described [62, 63]. The exact mechanism of improved infectious outcomes has yet to be elucidated, but is believed to involve the enhancement of gut integrity and immune function [62, 63].

For many years, postburn ileus—common with burns over 25 % of the total body surface area—was believed to be a contraindication to early enteral feeding. However, it is now known that postburn ileus does not affect the small bowel. Early administration of 75 % of the estimated caloric needs can be accomplished within the first 6 h after the burn and may lead to a reduction of the hypermetabolic state.

Enteral nutrition is preferred over parenteral support whenever possible. Enteral nutrition has been shown to increase gut blood flow, decrease mucosal atrophy, and preserve gastrointestinal function [64]. Stopping enteral feeding in the immediate preoperative and operative period is unnecessary, although commonly requested by anesthesia colleagues [46, 65]. Intra-operative, post-pyloric feeding may also have a protective effect on intestinal oxygen balance [66].

Complications

The most common complications associated with burns are respiratory failure, infection, and graft failure. However, there are other early and late complications of burn injury that should be kept in mind.

Vascular compromise may result directly from thermal, electrical, or chemical injury. This direct injury affects the outer adventitial layer first and then the media. When vessels are weakened, rupture can occur. However, a more common cause of vessel rupture following burn injury is infection of the wound, which can lead to necrosis of the vessel wall. Burns that cause intimal damage produce a thrombogenic surface [67].

During resuscitation of the patient with full-thickness burns of the trunk or circumferential full-thickness burns of any part of an extremity, consideration must be given to escharotomy (incision into the full-thickness eschar that relieves the underlying pressure resulting from fluid shift into the extravascular space) (see Fig. 45.2). Because escharotomies are performed through full-thickness insensate skin, this procedure can be done without anesthesia, although light sedation may be administered. Escharotomy traditionally is performed using a scalpel, but it also can be performed with cutting electrocautery.

Ulceration of the upper gastrointestinal tract in burn patients was first described by Curling [68] as acute ulceration of the duodenum. This complication now bears his name and is assigned to any erosive disease of the gastroduodenal mucosa. Significant burn injury leads to acute mucosal disease in 8 % of untreated patients [69]. Of these patients, 22 % have hemorrhages and 6 % have perforations [70]. The importance of prevention is clear. Measures include close monitoring of gastric pH and the use of antacids or H_2 blockers, proper nutritional support, and early enteral feeding.

Heterotopic ossification occurs with frequencies reported between 1 and 14 % [71, 72]. The pathogenesis is not fully understood. Factors such as burn size greater than 20 % and immobility have been implicated. Munster et al. [72] reported that even though full-thickness burns are more susceptible, heterotopic ossification has been reported in partial-thickness burns as well [73]. Preventive measures include early ambulation and active and passive joint motion.

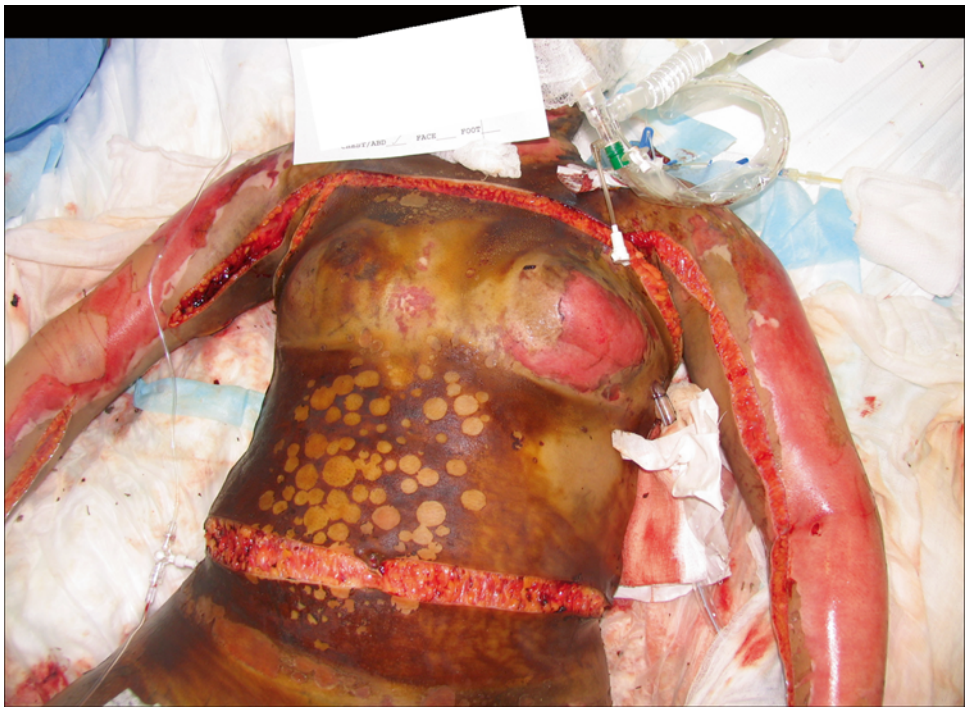


Fig. 45.2 Third degree burns to trunk and arms requiring escharotomies

Contraction is a normal part of wound healing and determines ultimate functional outcome and cosmetic appearance. Return to pre-burn function and the best cosmetic result possible are the two most important goals of the surgeon after patient survival. Therefore, control of wound contraction and scar formation are crucial. McDonald and Deitch [74] reported that poor scar outcome was associated with several factors including dark skin pigmentation, younger patient age, deeper depth of the burn, absence of dermis, and delayed grafting 14 days after the burn. Grafts placed on the head and neck tend to have a worse outcome than grafts placed on the trunk and extremities. As with all complications, prevention is preferred over treatment. The presence of dermal elements decreases heavy scarring and contracture rates. Therefore, thicker grafts containing more dermis tend to be scarless. However, thicker grafts are associated with their own complications and they may not be practical depending on the size of the burn and the area being grafted. Several products now available for the treatment of burns attempt to replace dermal elements. The use of these products has been shown to decrease scarring and maintain function, while at the same time providing acceptable cosmetic results [75]. Compression garments used in the scar maturation period decrease scarring by pressure-induced realignment of collagen fibers and reduction of collagen synthesis [76]. These garments are ideally worn 24 h a day for 12–18 months after the burn occurs; they are removed only for bathing. The mainstay of prevention, however, remains early graft coverage of the wound to avoid septic complications and granulation buildup, as well as early ambulation and movement of joints.

The operative approach to scar revision is beyond the scope of this chapter, and the reader is referred to surgical texts and atlases on the subject.

Squamous or basal-cell malignancies that form with the wound, known as Marjolin's ulcer, are characterized by thickened scars that break down repeatedly over a period of several years. These malignancies generally do not appear until 10 years or more following the burn. Even though they are low grade on histologic examination, by the time the patient presents and a definite diagnosis is made through biopsy, roughly one-third of patients have regional nodal metastasis and the overall prognosis is poor.

The occurrence of abdominal compartment syndrome, first described in the late 1800s, seems to have generated renewed interest among trauma and burn surgeons. The syndrome is characterized by high peak airway pressure, oliguria, and intra-abdominal pressures of greater than 25 mmHg as measured transvesically with a Foley catheter and transducer. Abdominal decompression is the only treatment and may be accomplished either by laparotomy [77] or placement of a temporary peritoneal dialysis catheter [78].

Chemical Burns

Injury from acids or alkalis is caused by desiccation of tissues and denaturation of proteins. As a rule, alkali burns tend to be deeper than those caused by acids, but the alkalis are usually not systemically absorbed. The description of chemical burns is the same as that of thermal burns. However, it is

important to remember that deep chemical burns may appear deceptively superficial on initial presentation.

As with any burn injury, an important first step in management is to stop the burning process. All clothing must be removed. If the chemical is dry, as much of it as possible should be brushed off. This is followed by copious wound irrigation. If the chemical is wet, irrigation of the wound is begun. The earlier wound irrigation is begun, the better the outcome [79, 80]. The irrigation should be continued for a minimum of 30 min [2]. Exceptions to this principle are hydrochloric acid, which should be naturalized with soda lime (avoid saline irrigation) and lithium metal, which may react violently with water. The size estimation and fluid resuscitation guidelines are the same for chemical as for thermal burns.

Electrical Injury

Electrical injuries happen less often than thermal burns, but they are much more difficult to manage and are associated with a higher morbidity and mortality rate. As current passes through the body, it meets resistance from the tissues. The amount of resistance is inversely proportional to the relative amount of water found in the tissue. Those tissues that have relatively little water content (i.e., bone) have more resistance to the current. According to Joules Law, the amount of heat produced by the passage of current is proportional to the resistance. Clinically, the more superficial tissues may appear normal, but the underlying bone and adjacent muscle are destroyed. In these situations, usually encountered with high-voltage injury, amputation of the involved extremities is commonly part of the initial debridement and wound management.

Because the amount of tissue destruction is not obvious with electrical injury, the amount of fluid needed for initial resuscitation may be twice the amount predicted by the application of the Parkland formula. In addition, the destruction of muscle produces myoglobin, which is released into the systemic circulation and excreted by the kidneys. In the normal acid environment of the urine, the myoglobin precipitates in the tubules and causes acute tubular necrosis and renal failure. To avoid this complication, fluid administration is increased over what is calculated with resuscitation formulas, and 25 g of mannitol is administered followed by 12.5 g every 2 h until the urine clears the myoglobin pigment. Additionally, sodium bicarbonate is administered to raise the urine pH to 6.5–7.0, while maintaining blood pH below 7.5 [81].

Electrical injuries can cause cardiac derangements ranging from dysrhythmias to myocardial infarction. While most of these injuries occur at the time of electrocution, some studies suggest that they can occur in the post-electrocution period [82], leading most institutions to admit patients for 24 h of cardiac monitoring. However, recent studies suggest that low-voltage injuries with normal presenting electrocardiograms, without loss of consciousness, do not need prolonged monitoring [83].

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