Acute Burn Injury

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Almost 2 million people in the United States suffer from burns annually. Children and the elderly have the greatest incidence of burn injuries during cooking or bathing accidents, whereas young adults are most often injured in the work place \[1\]. Approximately 10\% of burn patients require admission to hospitals or major burn centers for care. A small percentage of these injuries are fatal, but all require some degree of medical attention \[1,2\].

Burn injury is associated with anatomic, physiologic, endocrinologic, and immunologic alterations. Cutaneous injury triggers a reaction at a local and systemic level through the release of multiple inflammatory mediators. When the injury is extensive, this becomes a systemic reaction \[2,3\]. On a local level, these reactions will affect the surgical decision making and need to be understood to ensure the best possible short- and long-term outcomes. On a systemic level, the cascade of cellular alterations can lead to generalized inflammation and multiple system organ dysfunction. These problems need to be identified and treated properly to prevent or minimize the extent of the damage \[1,4\].

Although most injuries due to burns are minor, the care of the injuries requires specialized knowledge for improved outcomes. In recent years, advances in burn treatment have reduced morbidity and mortality and improved the quality of life for burn survivors. These advances have been made in the treatment of the acute injury, the quality of the initial resuscitation, the effectiveness of infection control, and the surgical decision making for improved short- and long-term outcomes \[5\].

In major burn centers, a more comprehensive, team-oriented approach has been introduced over the last 10 years. Personnel include a surgeon, critical care specialist, specialized nursing care, nutritionist, pharmacist, and physical therapist. This approach has improved survival rates for victims of increasingly more complex burn injuries and has improved rehabilitation outcomes. Historically, age, depth of injury, and surface area were important predictors of morbidity and mortality; however, current practice has altered these predictors \[1,5\].

Pathophysiology

Local response to injury

The skin is generally divided into epidermis and dermis. The epidermis has the function of barrier against environmental and bacterial elements. The dermis is principally involved in strength and flexibility, while providing specialized functions in moisture, vascularity, and communication \[1,6\].

The local response to burn is multifactorial. Some tissue is destroyed directly by heat, which denatures the proteins and ruptures the cells. Further necrosis
occurs after heat injury and thrombosis of blood vessels in the dermal layer. Stasis can occur in the surrounding tissue, particularly when the patient becomes hypovolemic [1].

On a more cellular level, multiple inflammatory mediators are released that create the potential for tissue ischemia in the zone of stasis. These vasoactive mediators (prostaglandins, histamine, and bradykinin) alter endothelial cell and basement membrane function to increase permeability, producing edema in the surrounding tissue. These mediators and derivatives are involved in the activation of complement, which amplifies the coagulation system, stimulating thrombosis to the microvasculature and causing further damage to the tissue from ischemia. At a cellular level, this ischemia affects free radical formation and plays a major role in increased tissue edema. Although the influx of inflammatory cells is essential to controlling local wound infection, there can be negative effects. Inflammatory cytokines are released, stimulating neutrophil degranulation and releasing toxic byproducts that are harmful to the tissue. Because of the potential for treatment options and its wider application, investigation is ongoing into this area of microcellular effect and destruction. These same cytokines stimulate chemotaxis to the area of injury [1,2].

**Systemic response to injury**

When a burn exceeds 20%, a systemic response ensues, stimulated by cellular mediators at the tissue level and by loss of the physical barrier.

Cellular processes stimulated by an injury of this size contribute to fluid imbalances and metabolic changes that are important to understand for management. Following the release of vasoactive mediators, such as prostaglandins, thromboxane A2, and reactive oxygen radicals, leaky capillaries and hypoprothrombinemia result, leaving the vessel devoid of fluid, with potentially massive amounts of third spacing. Generalized inflammation induces a counterregulatory, anti-inflammatory response that is expressed in immune suppression. Surgical debridement and closure of the wound, as with grafts, can limit this process [4,6].

With an injury of this size, changes in metabolism occur, leading to a near-doubling of cardiac output and high-energy requirements. The loss of physical barrier contributes to a decrease in fluid volume. In addition to the third spacing of fluids, already described, evaporation and leakage of fluid from these open wounds complicates an already difficult fluid management problem [6].

**Management**

**Initial evaluation**

The initial approach to the burn patient should be similar to the ABCs of trauma. Establishing airway and intravenous (IV) access is a priority. IV lines can be placed through burned tissue if necessary. Facial and airway burns present additional challenges. If there is any suggestion of airway injury or large-surface-area burn (>40%), pre-emptive intubations and aggressive resuscitation are recommended. Nasogastric suction and foley catheters are particularly recommended in these patients for aspiration prevention and resuscitation monitoring [2].

A detailed history and physical are necessary and may require the assistance of witnesses or family. Understanding the mechanism of injury can be crucial to the medical investigation. If the patient has suffered polytrauma, for example, a CT scan may be necessary. A thorough physical examination from head to toe should be documented.

Important to the practice of burn care will be the evaluation and understanding of head and neck burn injuries, with special attention to respiratory involvement [4]. Respiratory tract involvement is an important determinant of mortality. Common features include carbon monoxide and cyanide poisoning, potential increased fluid requirements, upper airway obstruction, chest wall restriction, progressive edema formation, and pneumonia. Acute appreciation of the mechanism of injury is the best guide to establishing this diagnosis. The thorough physical examination, which may include a bronchoscopy if necessary, will aid the diagnosis. When the suspicion exists, baseline pulse oximetry and arterial blood gases (ABGs) are helpful [1,4,6].

The airway injury increases the surface area involved and triggers both local and systemic edema. The direct heat injury to the respiratory mucosa, together with chemical irritation, leads to sloughing of the mucosa and cast formation. The combination of this mechanical obstruction with atelectasis can lead to barotrauma and respiratory insufficiency. If the chest wall has been burned, full inspiratory effort may be restricted [1].

Having completed the systemic evaluations of the patient, one must know how to quantify and qualify burns. The authors record location, surface area, and depth of penetration to the best of their ability. Recording the depth of penetration can be difficult, but burn size should be measured accurately, because this will significantly aid the fluid resuscitation effort. The most accurate way to quantitate
burn size is to use Lund-Browder charts that describe the size of each body region. These are available in most burn centers and emergency rooms. A simple alternative method of approximating burn size is the rule of nines. Simply put, the body is divided into regions, and each region is a multiple of nine. The values are as follows: each arm is 9%, the head is 9%, the legs are 18% each, and the torso is 36%.

For smaller burns, it is important to know that a palm-sized defect is recorded as 1%. The specific body area burned has obvious implications for outcome. Full-thickness burns of the face can result in life-altering changes in appearance, whereas the same burn on the back will have little significance [1]. In general, increased age is a poor prognostic factor. An age-specific chart has been included to aid in this calculation.

Resuscitation

After appropriate initial stabilization, fluid resuscitation plays the most important role in patient care and survival. The abundance of formulae that have been derived to help the practitioner with management are a testament to this importance. Because of the large open areas of skin and leaky capillaries, the body requires enormous amounts of fluid to maintain adequate organ perfusion. In a patient with normal kidney function, the authors use urine output as the ultimate objective finding for this end-goal. In an adult, 0.5 to 1 mL per kilogram per hour of urine is a well-known marker for adequate end-organ perfusion. In a child, the concentrating capacities of the kidney are less well developed, and 1 to 2 mL per kilogram per hour is recommended. Inhalation injury dramatically increases these requirements [7]. Most practitioners use lactated Ringer’s solution for resuscitation, using the Parkland formula. Colloids are usually reserved for the second 24 hours. Regular adjustments in the fluid volume are needed to meet these end-goals (Box 1). Ultimately, these patients belong in an ICU setting where additional critical care can be delivered by a well-trained staff. Surgical intensive care, management of failing organ systems, and support to the hypermetabolic response are beyond the scope of this report [1,4,6].

Inhalation injury

The surgeon may be called on to stabilize the burn patient with inhalation injury, which is the most common injury associated with a cutaneous burn. After burn size, respiratory tract involvement is the most important determinant of mortality. Inhalation injury has three components: (1) carbon monoxide poisoning, (2) upper airway swelling, and (3) lung injury. Any of these problems may occur alone or in combination. Diagnosis is suggested by a history of

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Box 1. Case example: diagnosis and resuscitation with Parkland (Baxter) formula

An adult weighing 70 kg has burns to the anterior face and neck, upper half of the back, chest and abdomen, and entire right arm. Patient arrives 2 hours after injury.

Estimate burn size using the rule of nines:

Front half of head = 4.5%.
Right arm = 9%.
Back (half of back) = 9%.
Chest and abdomen = 18%.

Total burn percentage (TBSA) = 40.5% (may use 40% for calculations).

First 24 hours:

- Lactated Ringer’s solution: 4 mL/kg/% burn in 24 h (half of fluid given in first 8 h after injury)
- Colloid: none

Second 24 hours:

- Crystalloid to maintain urine output. Nutritional support should begin by the enteral route.
- Colloid (5% albumin in lactated Ringer’s solution):
  - <50% TBSA burn: none
  - >50% TBSA burn: 0.5 mL/kg/% burn in 24 h

Example for patient above:

Give 5600 mL over 6 h = 933 mL/h for 6 h.
Give 5600 mL over 12 h = 350 mL/h for 16 h.
fire in a closed space, coughing of soot-tinged sputum, or change in voice. Physical findings supporting this diagnosis include large facial burns, singed nasal and facial hair, wheezing, coughing, or accessory muscle use, and intraoral soot and ulceration (Box 2).

Signs and symptoms of carbon monoxide (CO) intoxication are provided in Table 1. Definitive diagnosis is made by direct visualization of the airway with direct or indirect laryngoscopy or bronchoscopy. Normal pulse oximetry does not eliminate the possibility of dangerous levels of carboxyhemoglobinemia [1,2,6].

Specific treatments exist for each of these conditions. CO poisoning is treated by the administration of 100% oxygen. This measure decreases the time during which the CO binds to hemoglobin and prevents oxygen transport. Because of the marked affinity of this gas for hemoglobin, patients may struggle to deliver oxygen to the peripheral tissues. Although isolated CO poisoning can be treated with hyperbaric oxygen, burn experts universally agree that this is not the best treatment for a patient with a significant cutaneous burn. Although low levels of CO are well tolerated, CO levels of more than 40% are associated with significant neurologic dysfunction and death.

The treatment for respiratory tract injury is supportive. Because edema can rapidly progress, early intubation is the key to airway stability. Airway swelling is treated with prophylactic intubation. When airway swelling is an isolated problem, the patient can usually be extubated 3 to 5 days after the injury. It is critical to document the waning of edema by either direct visualization of the airway or a “leak test”—deflating the cuff of the tube, occluding the lumen, and allowing the patient to breathe through the “leak” around the tube. Lung injury results from “toxic products of combustion,” not from heat, and is a chemical pneumonitis. The chemical irritation leads to sloughing of the mucosa and epithelial cast formation. Specialty care with a low-volume, high-frequency ventilator provides protection against worsening barotrauma and aids in the clearance of thick acquired secretions. If the chest wall has been burned, full inspiratory effort may be restricted. Relieving this restriction with medial and lateral axillary escharotomies, using coagulating electrocautery under light sedation, is recommended. All of these factors contribute to an increase in the incidence of pneumonia among inhalation burn injury victims [1,3,4].

### Wound assessment and management

Although the burn surface area is a key ingredient in the formula for resuscitation, the burn depth is a more important determinant for wound healing. Multiple modalities have been proposed to define the ischemic wound, but the physical examination remains the most reliable method of diagnosis. First-degree burns involve only the superficial epidermis. The skin is red, dry, and hypersensitive. With the epidermal barrier intact, the metabolic responses and infection risk are minimal. The treatment includes pain control and topical antimicrobials. In second-degree burns, there is complete destruction of the epidermis and partial dermal loss [3]. The depth of the dermal injury is an important factor. The skin is edematous, red, wet, and painful, with possible blistering. If the epidermal appendages are intact, the wound can heal by proliferation and migration of these cells. The deeper the dermal injury, the greater the challenge to the healing process. A superficial dermal injury can heal in 7 to 10 days with mild support, whereas a deeper dermal injury can take up to 3 weeks and have various degrees of scar contracture and hypertrophy. Third-degree burns are, by definition, full-thickness and have a pale, contracted,

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### Box 2. Signs of inhalational injury

- Burn occurred in closed space, including motor vehicle accident.
- Facial burns
- Singed nasal hairs
- Carbonaceous sputum
- Carbonaceous particles in larynx
- Change in voice
- New-onset hoarseness

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### Table 1

<table>
<thead>
<tr>
<th>Percentage of CO in hemoglobin</th>
<th>Symptoms</th>
</tr>
</thead>
<tbody>
<tr>
<td>0–10</td>
<td>None</td>
</tr>
<tr>
<td>10–20</td>
<td>Headache, confusion</td>
</tr>
<tr>
<td>20–40</td>
<td>Disorientation, fatigue, nausea, and visual changes</td>
</tr>
<tr>
<td>40–60</td>
<td>Hallucination, combativeness, coma, and shock</td>
</tr>
<tr>
<td>&gt;60</td>
<td>Mortality &gt;50%</td>
</tr>
</tbody>
</table>
insensate, and leathery appearance. These injuries require debridement and biologic wound closure. Fourth-degree burns can reach the depth of bone and provide a more challenging ultimate reconstruction. These wounds also require initial excision and immediate closure to maximize tissue survival (Table 2) [1,4,8].

The deeper the cutaneous injury, the more important is the care taken to achieve wound closure without surgery. With the loss of the epidermal barrier, the wounds need support from topical antimicrobials and appropriate dressings to maintain tissue moisture. Burn wound care is designed to create a favorable environment for endogenous wound healing while preventing complications, such as infection. In general, when wounds are large (greater than 10% TBSA) or have significant necrotic tissue or when patients are not reliable, it is best to treat the wound with a topical antimicrobial agent. The most widely used agent is 1% silver sulfadiazine (Silvadene, The Kendall Co., Mansfield, MA), because it has a broad spectrum of activity against gram-positive and -negative bacteria and has few complications. Patients are instructed to clean the wound daily with soap and water, using gauze to remove exudates, debris, and old antibiotic. The wound is then covered with a thin layer of Silvadene and a gauze dressing. Surgi-Net (Medi-Tech International Corp., Brooklyn, NY) will hold dressings in place effectively. These daily dressings are usually painful, and patients should be given adequate analgesics [1]. Ears have the particular problem of chondritis, because of the limited direct blood supply to the cartilage. The capacity of 10% sulfamylon cream to penetrate these tissues effectively treats this problem.

Two alternative agents are useful in special circumstances. Silver-releasing dressings such as Acticoat (Smith & Nephew, Inc., Largo, FL) can provide continuous antimicrobial activity without requiring painful daily dressings. These agents are not useful for wounds with significant necrotic tissue, which necessitates daily wound care. Because Silvadene is a cream, it may run into the eyes, causing conjunctivitis. Face burns are less often infected and need antibiotic treatment only to prevent gram-positive bacteria. Bactroban (Mupirocin) is an effective alternative [1].

### Initial excision and wound closure

**Surgical treatment**

In the past, the accepted method of burn wound management was delayed debridement and grafting. This approach resulted in prolonged periods of wound treatment until burn eschars separated. The result was wound sepsis, frequently leading to fibrosis and contracture. Today, it is widely agreed that immediate debridement and some form of biologic wound closure is the best way to activate healing and shut down the hypermetabolic response. The objective of surgical treatment is to debride all necrotic tissue and provide closure to the wound within the first week. In most cases, these procedures are done at the same time. However, when viability of tissue or control of infection is uncertain, it is reasonable to delay wound closure for a few days [1,4,6].

Debridement should be complete before consideration of wound closure. The authors now routinely inject dilute epinephrine solution (1:1,000,000) sub-eschar to control bleeding, much as in tumescent liposuction. The routine use of lidocaine in the injectate is not indicated, because it frequently will exceed recommended amounts. Tourniquets can be used to limit blood loss when debriding extremity injuries. Several instruments, such as Weck (Pilling Surgical, Horsham, PA) and Watson (Integra Life Sciences, Plainsboro, NJ) knives, are available to allow sequential removal of thin tissue slices (0.008—

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**Table 2**

<table>
<thead>
<tr>
<th>Degree</th>
<th>Common causes</th>
<th>Color</th>
<th>Sensitivity</th>
<th>Appearance</th>
<th>Treatment</th>
<th>Risk of poor scarring</th>
</tr>
</thead>
<tbody>
<tr>
<td>First</td>
<td>Flash, sunburn</td>
<td>Pink</td>
<td>Painful</td>
<td>Intact epithelium</td>
<td>Symptomatic</td>
<td>None</td>
</tr>
<tr>
<td>Second (superficial)</td>
<td>Hot water</td>
<td>Pink</td>
<td>Very painful</td>
<td>Wet</td>
<td>Antibiotic dressing</td>
<td>Low</td>
</tr>
<tr>
<td>Second (deep)</td>
<td>Hot soup</td>
<td>Pink</td>
<td>Painful</td>
<td>Wet</td>
<td>Antibiotic dressing, possible surgery</td>
<td>If delayed healing</td>
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<tr>
<td>Third</td>
<td>Flame, grease</td>
<td>White, brown</td>
<td>Little pain</td>
<td>Dry</td>
<td>Surgery</td>
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**Signs and symptoms of burn injury**

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0.012 in), preserving all viable subcutaneous fat. An electric dermatome can also be used for broad, flat surfaces, such as the chest or back. This type of debridement improves both appearance and mobility [1].

The best choice for wound closure depends on the wound and the patient. Although the most common method of wound closure is skin grafting, other techniques are also used. In general, the simplest solution is the best. Rarely, small, full-thickness burns can be closed by mobilization of the adjacent tissue and primary closure. Although the scar is never perfect and often requires revision, the result is better than a skin graft and donor site scar. Skin grafting usually entails split-thickness skin grafts. For large burns, the grafts can be meshed, but although meshing increases graft take and decreases donor-site area, the resulting scars are always irregular and unsightly. An indication seldom exists for using meshed grafts on the head and neck, in children, or for small burns. The authors use grafts that are 12/1000 of an inch in adults, 10/1000 in older children and the aged, and 8/1000 of an inch in young children. For small burns, a full-thickness graft can be harvested and thinned to partial thickness; the donor site can be closed primarily. Donor sites should be chosen with care, because some will hypertrophy and many become hyperpigmented. The back heals with the best scar but can be inconvenient for the surgeon [9]. Flanks and proximal thighs are good alternatives. A small graft should never be obtained from the middle of the anterior thigh. This procedure creates a scar in a visible location [1].

Advances in tissue engineering have provided the surgeon with several biologic dressings that are available for situations in which the patient is too sick to handle the operative repair or inadequate tissue is available for immediate coverage. Integra (Integra Life Sciences, Plainsboro, NJ) is a dermal analogue that allows the surgeon to close the burn wound when inadequate skin for grafting is available. After the Integra is engrafted, a split graft is placed to reconstruct an epidermis. Compared with expanded mesh grafts, Integra-grafted wounds have better appearance, elasticity, and flexibility [10]. Transcyte is an artificial epidermis that closes the wound. It decreases pain, improves healing, and reduces the need for expensive nursing care [1].

Special needs for grafting the head and neck

In general, burns to the head and neck heal well. However, this does not mean that third-degree burns will heal without surgery. Experienced surgeons have developed the protocol of early debridement and grafting of facial burns, as on the rest of the body. The result is better function, fewer contractures, and improved appearance [11]. The same principles apply: debridement of necrotic tissue and grafting with reasonably thick sheet grafts or Integra. Do not remove normal tissue to complete facial aesthetic-unit grafting for the acute injury. When facial burns are combined with an inhalation injury requiring tracheal intubation, tracheostomy should be considered. A 14 to 16/1000-of-an-inch graft takes well and contracts less than do thinner grafts. The broad, flat areas of the forehead and cheek accept grafts well. The complex three-dimensional pattern of the midface makes grafting more difficult. The use of fibrin glue dramatically decreases the need for suture graft fixation.

Reconstruction

A detailed description of the multiple reconstructive needs after burn injury is beyond the scope of this chapter. Several useful guiding principles can be discussed. First, reconstruction begins during treatment of the acute burn. Early debridement and sheet grafting limit hypertrophic scar and contracture, the most common causes of postburn reconstruction. Second, the general schema for treatment is wound closure, restoration of function, restoration of appearance. The first should occur in days, the second in months, the latter in years. Functional limitations of mouth opening, nasal breathing, eye closure and eyelid motion, and neck motion should be treated aggressively with scar management and therapy. When nonoperative treatment does not restore normal function, surgical release and reconstruction with adjacent normal tissues or grafts is essential. Treatment of the aesthetic deformities requires more judgment and experience. More scars lose their redness and become thinner with time. In general, this improvement continues for 2 to 3 years after injury. Because surgery reinitiates the scar formation process, it is important to delay interventions, or the scars will recur.

References